SIXTH EDITION

Joint Structure & Function

A COMPREHENSIVE ANALYSIS





Pamela K. Levangie
Cynthia C. Norkin
Michael D. Lewek

F.A. DAVIS

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Editors

Pamela K. Levangie, PT, DPT, DSc, FAPTA

Professor Emerita

Department of Physical Therapy

School of Health and Rehabilitation Sciences

MGH Institute of Health Professions

Boston, Massachusetts

Cynthia C. Norkin, PT, EdD

Professor Emerita

Division of Physical Therapy

College of Health Sciences and Professions

Ohio University

Athens, Ohio

Michael D. Lewek, PT, PhD

Associate Professor

Division of Physical Therapy

Department of Allied Health Sciences

University of North Carolina at Chapel Hill

Chapel Hill, North Carolina

Dedication

It is my honor, on behalf of Dr. Michael Lewek and myself, to dedicate the sixth edition of *Joint Structure and Function* to our colleague and my friend, Dr. Cynthia Norkin. Dr. Norkin passed away shortly before its publication. She and I both joined the faculty of the Physical Therapy Program at Boston University, Sargent College of Allied Health (as it was called then) in 1973. After co-teaching Functional Anatomy for several years with a packet of mimeographed course materials, it was Dr. Norkin's vision to turn these materials into a textbook. She convinced me (the harder sell) and FA Davis, Publishers (the easier sell) of the need at a time when there were few allied health texts on the market. The first edition came out several years later. In 1984, Dr. Norkin became the founding Director of the Physical Therapy program at Ohio University where she remained until her retirement in 1995. She and I continued our close and productive collaboration through the second through fifth editions. Although health issues limited her participation in the development of the sixth edition at the same time that Dr. Lewek joined the editorial team, she remained an active and enthusiastic supporter. I am forever indebted to Dr. Norkin for pushing me into a project that I would never have undertaken on my own initiative. *Joint Structure and Function* strongly influenced my own professional journey; far more importantly, the text influenced the professional journey of more than 100,000 students through the years. Her legacy and influence live on.

Pamela K. Levangie

Preface

With publication of the sixth edition of *Joint Structure and Function,* we continue on a journey started nearly 40 years ago. The text at that time targeted a substantive void in the evidence-based kinesiologic foundations upon which an understanding of typical and impaired movement should be based. With continuing advances in research and information technologies, individuals now have a variety of choices in how to access and use the explosion of information available to them. Our goal in the sixth edition is to be responsive to the needs and preferences of our contemporary audience while continuing the tradition of being an accessible and preferred resource for those seeking current concepts in kinesiology.

The sixth edition reflects changes in an understanding of adult learning and in learning preferences.

- We have increased and enhanced the images that support the underlying concepts.
- We have retained the boxed features, now named *Foundational Concepts, Expanded Concepts,* and *Patient Applications,* to facilitate the reader's ability to prioritize content.
- Chapter outlines with page numbers at the beginning of each chapter allow more efficient chapter navigation.
- We have added *Anatomy Overview* tables at the beginning of each joint complex chapter to provide a quick review of the musculature.

- New chapter contributors and the new member of our editorial team, Dr. Michael Lewek, reflect a commitment to engaging a new generation of researcher educators.

Although our learners and the technology that supports their learning are evolving, *Joint Structure and Function* remains committed to providing a strong, contemporary, and evidence-based foundation in the principles that underlie an understanding of human structure and function while also being as accessible and concise as possible. We very much appreciate our opportunity to contribute to the health of society by assisting in the professional development of the students and providers who are our readers.

Pamela K. Levangie
Cynthia C. Norkin
Michael D. Lewek

Contributors

Noelle M. Austin, PT, MS

Certified Hand Therapist

CJ Education & Consulting, LLC

www.cj-education.com

Lynchburg, VA 24503

David B. Berry, PhD

Department of Bioengineering, Nanoengineering, Orthopaedic Surgery

UC San Diego

San Diego, California

John Borstad, PT, PhD

Professor and Chair

College of St. Scholastica

Duluth, Minnesota

Gary Chleboun, PT, PhD

Professor of Physical Therapy

Director, School of Rehabilitation and Communication Sciences

Ohio University

Athens, Ohio

Diane Dalton, PT, DPT, OCS

Board Certified Clinical Specialist in Orthopaedic Physical Therapy

Clinical Associate Professor

College of Health & Rehabilitation Sciences: Sargent College

Boston University

Boston, Massachusetts

Janice Eng, PhD, BSR (PT/OT), MSc

Professor and Canada Research Chair

University of British Columbia

Vancouver, British Columbia, Canada

Jeff Hartman, PT, DPT, MPH

Assistant Professor

Northwestern University, Feinberg School of Medicine

Department of Physical Therapy & Human Movement Sciences

Chicago, Illinois

Michael A. Hunt, PhD, BHK, MPT, MSc

Associate Professor

Department of Physical Therapy

University of British Columbia

Vancouver, British Columbia, Canada

Benjamin Kivlan, PT, PhD, SCS, OCS

Board Certified Clinical Specialist in Sports and Orthopaedic Physical Therapy

Assistant Professor

Duquesne University

Rangos School of Health Sciences

Department of Physical Therapy

Pittsburgh, Pennsylvania

David S. Logerstedt, PT, PhD, MPT, MA

Board Certified Clinical Specialist in Sports Physical Therapy

Assistant Professor

University of the Sciences

Philadelphia, Pennsylvania

Paula M. Ludewig, PT, PhD, FAPTA

Professor, Division of Physical Therapy

Department of Rehabilitation Medicine

University of Minnesota

Minneapolis, Minnesota

Lee N. Marinko PT, ScD OCS, OMT, FAAOMPT

Board Certified Clinical Specialist in Orthopaedic Physical Therapy

Fellow American Academy of Orthopaedic Manual Physical Therapists

Clinical Assistant Professor

Boston University

Boston, Massachusetts

RobRoy L. Martin, PT, PhD

Certified Strength and Conditioning Specialist

Professor

Duquesne University

Pittsburgh, Pennsylvania

Sandra J. Olney, BSc (PT&OT), PhD, MD (hon)

Professor Emeritus

School of Rehabilitation Therapy

Queens University

Kingston, Ontario, Canada

Pamela D. Ritzline, PT, EdD

Dean, School of Behavioral and Health Sciences

Professor, Physical Therapy

Walsh University

North Canton, Ohio

Amee Seitz, PT, PhD, DPT, MS, OCS

Board Certified Clinical Specialist in Orthopaedic Physical Therapy

Associate Professor

Northwestern University, Feinberg School of Medicine

Department of Physical Therapy & Human Movement Sciences

Chicago, Illinois

Bahar Shahidi, PT, PhD, DPT

Assistant Professor

Department of Orthopaedic Surgery

UC San Diego

San Diego, California

Susan M. Sigward, PT, PhD

Athletic Trainer, Certified

Associate Professor of Clinical Physical Therapy

Division of Biokinesiology and Physical Therapy

University of Southern California

Los Angeles, California

Lynn Snyder-Mackler, PT, ATC, ScD, SCS, FAPTA

Board-certified Clinical Specialist in Sports Physical Therapy

Athletic Trainer, Certified

Alumni Distinguished Professor

Department of Physical Therapy

University of Delaware

Newark, Delaware

Julie Ann Starr, PT, DPT

Board Certified Clinical Specialist in Cardiovascular and Pulmonary Physical Therapy

Clinical Associate Professor

College of Health and Rehabilitation Sciences, Sargent College

Boston University

Boston, Massachusetts

Samuel R. Ward, PT, PhD, FAPTA

Professor and Vice Chair of Research

Departments of Orthopaedic Surgery, Radiology, and Bioengineering

UC San Diego

San Diego, California

Elizabeth Wellsandt, PT, PhD, DPT, OCS

Board Certified Clinical Specialist in Orthopaedic Physical Therapy

Assistant Professor

University of Nebraska Medical Center

Division of Physical Therapy Education

Omaha, Nebraska

Chapter 1: Biomechanical Applications to Joint Structure and Function

Samuel R. Ward; Bahar Shahidi; David B. Berry

Introduction

Humans have the capacity to produce a nearly infinite variety of postures and movements that require the structures of the human body to both generate and respond to forces that produce and control movement of the body's joints. Although it is impossible to capture all the kinesiologic elements that contribute to human musculoskeletal function at a given point in time, knowledge of at least some of the physical principles that govern the body's response to active and passive stresses is a prerequisite to an understanding of both human function and dysfunction.

We will examine some of the complexities related to human musculoskeletal function by examining the roles of the bony segments, joint-related connective tissue structures, and muscles, as well as the external forces applied to those structures. We will develop a conceptual framework that provides a basis for understanding the stresses on the body's major joint complexes and the responses to those stresses. Case examples and clinical scenarios will be used to ground the reader's understanding in relevant applications of the presented principles. The objective is to cover the key biomechanical principles necessary to understand individual joints and their interdependent functions in posture and locomotion. Although we acknowledge the role of the neurological system in motor control, we leave it to others to develop an understanding of the theories that govern the roles of the controller and feedback

mechanisms.

This chapter will explore the biomechanical principles that must be considered in the examination of the internal and external forces that produce or control movement. The focus will be largely on rigid body analysis; the next two chapters explore how forces affect deformable connective tissues (Chapter 2) and how muscles create and are affected by forces (Chapter 3). Subsequent chapters then examine the interactive nature of force, stress, tissue behaviors, and function through a regional exploration of the joint complexes of the body. The final two chapters integrate the function of the joint complexes into the comprehensive tasks of posture (Chapter 13) and gait (Chapter 14).

To maintain our focus on clinically relevant applications of the biomechanical principles presented in this chapter, the following patient application will provide a framework within which to explore the relevant principles of biomechanics.



Patient Application 1–1

John Alexander is 20 years old, 5 feet 9 inches (1.75 m) in height, and weighs 165 pounds (~75 kg or 734 N). John is a member of the university's lacrosse team. He sustained an injury when another player fell onto the posterior-lateral aspect of his right knee. Physical examination and magnetic resonance imaging (MRI) resulted in a diagnosis of a tear of the medial collateral ligament, a partial tear of the anterior cruciate ligament (ACL), and a partial tear of the medial meniscus. John agreed with the orthopedist's recommendation that a program of knee muscle strengthening was in order before moving on to more aggressive options such as surgery. The initial focus will be on strengthening the quadriceps muscle. The fitness center at the university has a leg-press machine (**Fig. 1–1A**) and a heavy ankle cuff weight (**Fig. 1–1B**) that John can use.

FIGURE 1-1

A. Leg-press exercise apparatus for strengthening hip and knee extensor muscles. **B.** Ankle weight for strengthening knee extensor muscles.



A



B

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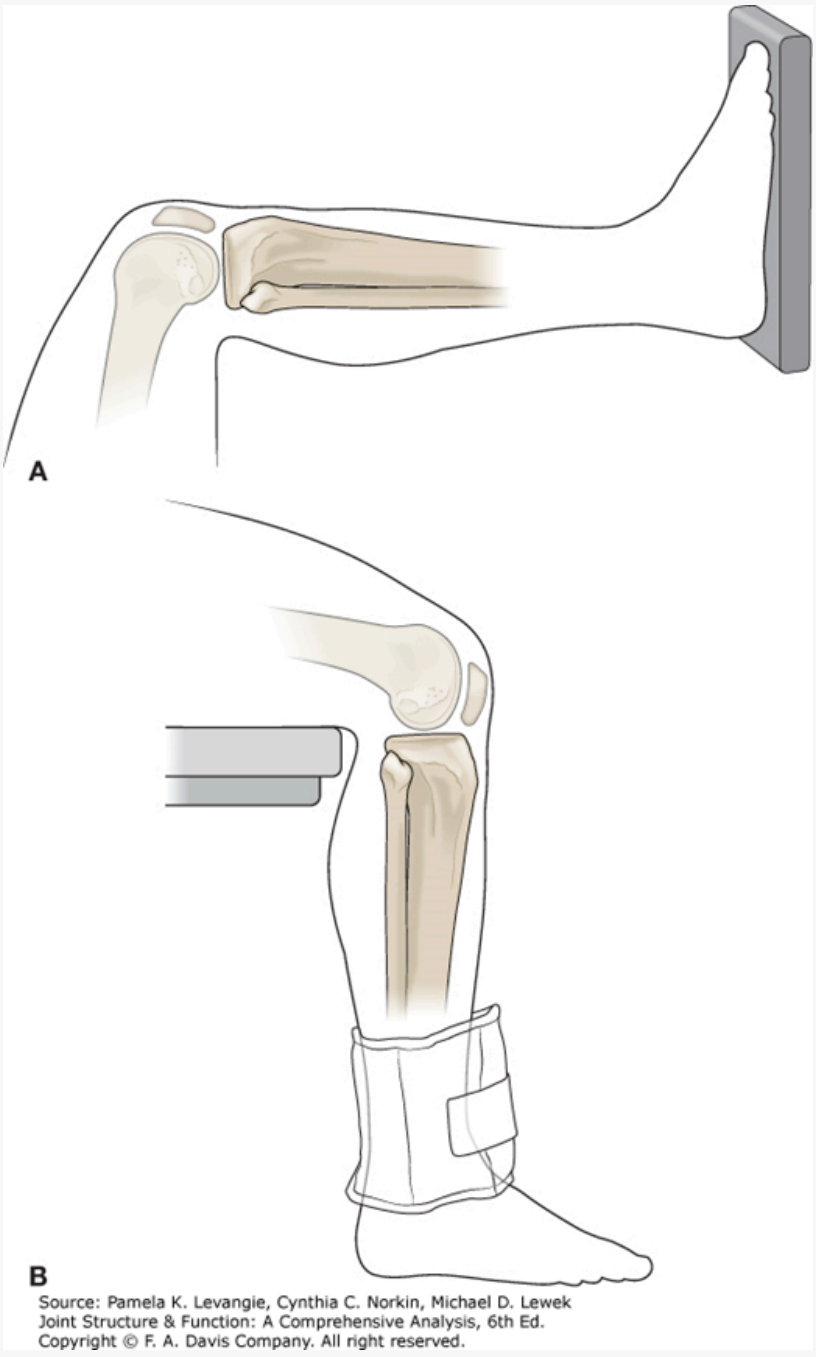
As we move through this chapter, we will consider the biomechanics of each of these rehabilitation exercise options in relation to John's injury and strengthening goals.

Human motion is inherently complex, involving multiple segments (bony levers) and forces that are most often applied to two or more segments simultaneously. To develop a conceptual model that can be understood and applied clinically, the common strategy is to focus on one segment at a time. For the purposes of analyzing John Alexander's issues, the focus will be on the leg-foot segment, treated as if it were one rigid unit acting at the knee joint. Figure 1–2 is a schematic representation of the leg-foot segment in the leg-press and ankle weight scenarios. The leg-foot segment is the focus of the figure, although the contiguous components (distal femur, footplate of the leg-press machine, and the ankle weight) are maintained to give context. In some subsequent figures, the femur, footplate, and ankle weight are omitted for clarity, although the forces produced by these segments and objects will be shown. This limited visualization of a segment (or a selected few segments) can be referred to as a **free body diagram** or a **space diagram.** If proportional representation of all forces is maintained as the forces are added to the segment under consideration, it is known as a "free body diagram." If the forces are shown but a simplified understanding rather than graphic accuracy is the goal, then the figure is referred to as a "space diagram."[1] We will use space diagrams in this chapter and text because the forces are generally not drawn in proportion to their magnitudes for purposes of space efficiency.

FIGURE 1-2

A. The leg-foot segment on the leg-press footrest. **B.** The leg-foot

segment with an ankle weight.



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As we begin to examine the leg-foot segment in either the ankle weight or leg-press exercise scenario, the first step is to describe the motion of the segment that is or will be occurring. This involves the area of biomechanics known as **kinematics.**

PART 1: Kinematics and Introduction to Kinetics

Descriptions of Motion

Kinematics includes the set of concepts that allows us to describe the **displacement** (the change in position over time) or motion of a segment without regard to the forces that cause that movement. The human skeleton is, quite literally, a system of segments or levers. Although bones are not truly rigid, we will assume that bones behave as rigid levers for purposes of simplification. There are five kinematic variables that fully describe the motion, or the displacement, of a segment: (1) the **type** of displacement (motion), (2) the **location** in space of the displacement, (3) the **direction** of the displacement of the segment, (4) the **magnitude** of the displacement, and (5) the **rate** of change in displacement (velocity) or the rate of change of velocity (acceleration).

Types of Displacement

Translatory and rotary motions are the two basic types of movement that can be attributed to any rigid segment. General motions are achieved by combining translatory and rotary motions.

Translatory Motion

Translatory motion (linear displacement) is the movement of a segment in a straight line. In human movement, isolated translatory movements are rare. However, a clinical example of an attempt at isolated translatory motion occurs during joint mobilization, whereby a clinician imposes a near linear motion (sliding) between two articular surfaces such as sliding the tibial plateau on the femur at the knee joint. In fact, translation of a body segment at an intact joint rarely occurs in human motion without some concomitant rotation (rotary motion) of that segment (even if the rotation is

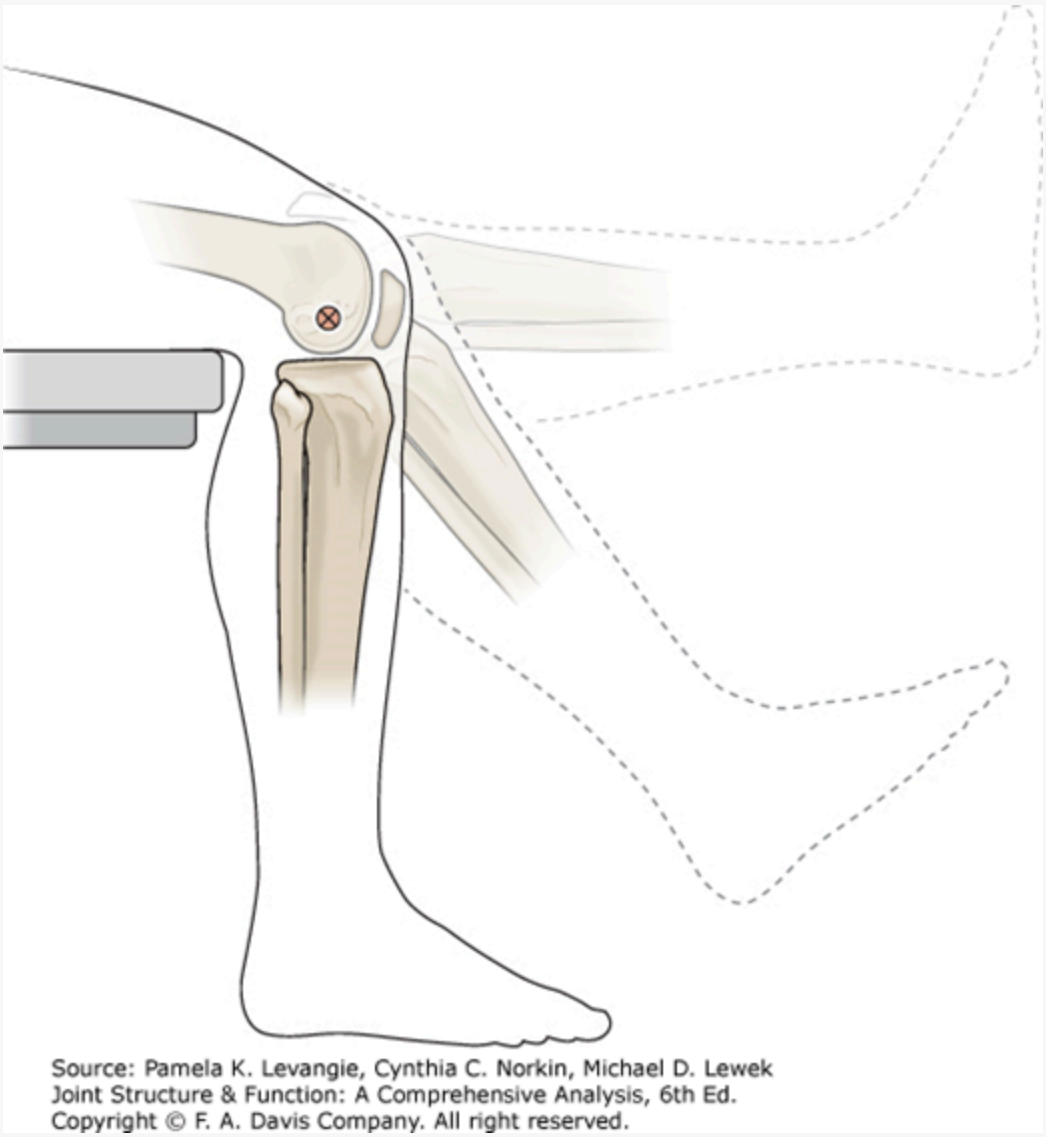
barely visible).

Rotary Motion

Rotary motion (angular displacement) is movement of a segment around a fixed axis **(center of rotation [CoR])** in a curved path. In true rotary motion, each point on the segment moves through the same angle, at the same time, at a constant distance from the center of rotation. True rotary motion can occur only if the segment is prevented from translating and is forced to rotate about a fixed axis. This rarely happens in human movement. In the example in Figure 1–3, all points on the leg-foot segment appear to move through the same angle at the same time around what appears to be a fixed axis. In actuality, none of the body segments move around truly fixed axes; all joint axes shift at least slightly during motion because segments are not sufficiently constrained to produce pure rotation.

FIGURE 1-3

Rotary motion. Each point in the leg-foot segment moves through the same angle, at the same time, at a constant distance from the fixed (or relatively fixed) center of rotation or axis.



General Motion

When nonsegmented objects are moved, combinations of rotation and translation (**general motion**) are common, although a number of terms can be used to describe the result.

Curvilinear (**plane** or **planar**) **motion** designates a combination of translation and rotation of a segment in *two dimensions* (parallel to a plane with a maximum of three

degrees of freedom).[2–4] When this type of motion occurs, the axis about which the segment moves is not fixed but, rather, shifts in space as the object moves. The axis around which the segment appears to move in any part of its path is referred to as the **instantaneous center of rotation (ICoR),** or **instantaneous axis of rotation (IaR).** An object or segment that travels in a curvilinear path may be considered to be undergoing rotary motion around a fixed but quite distant CoR;[3,4] that is, the curvilinear path can be considered a segment of a much larger circle with a distant axis.

Three-dimensional motion is a general motion in which the segment moves across all three dimensions (planes). Just as curvilinear motion can be considered to occur around a single distant center of rotation, three-dimensional motion can be considered to be occurring around a **helical axis of motion (HaM),** or **screw axis of motion.**[3]

As already noted, the motion of a body segment is rarely sufficiently constrained by the ligamentous, muscular, or other bony forces acting on it to produce isolated rotary motion. Instead, there is typically at least a small amount of translation (and often a secondary rotation) that accompanies the primary rotary motion of a segment at a joint. Most joint rotations, therefore, take place around a series of instantaneous centers of rotation. The "axis" that is generally ascribed to a given joint motion (e.g., knee flexion) is typically a midpoint among these instantaneous centers of rotation rather than a truly fixed center of rotation. Because most body segments actually follow a curvilinear path, the true center of rotation is the point around which true rotary motion of the segment would occur and is generally quite distant from the joint.[3,4]

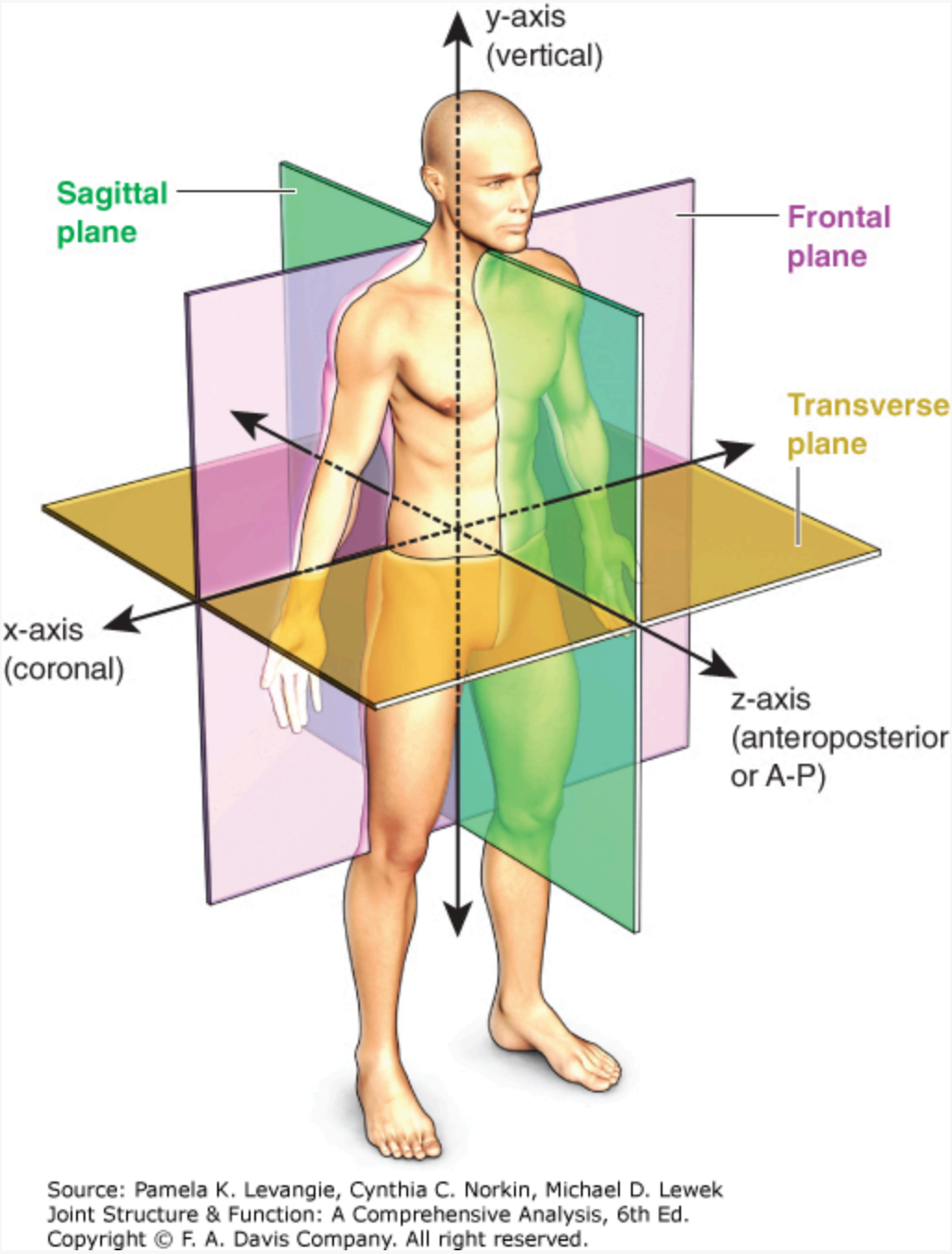
Location of Displacement in Space

The rotary or translatory displacement of a segment is commonly located in space by

using the three-dimensional Cartesian coordinate system as a useful frame of reference. The origin of the x-axis, y-axis, and z-axis of the coordinate system for describing human movement is traditionally located at the **center of mass (CoM)** of the human body, assuming that the body is in **anatomic position** (facing forward in standing with palms forward) (Fig. 1–4). According to the common system described by Panjabi and White, the x-axis runs side-to-side in the body and is labeled as the **coronal axis**; the y-axis runs up and down in the body and is labeled as the **vertical axis**; and the z-axis runs front to back in the body and is labeled as the **anteroposterior (A-P) axis.**[3] Motion of a segment can occur either *around* an axis (rotation) or *along* an axis (translation). An unconstrained segment can either rotate or translate around each of the three axes, resulting in six potential options for motion of that segment. The options for movement of a segment are also referred to as **degrees of freedom.** A completely unconstrained segment always has six degrees of freedom because it can rotate around the three axes (three degrees of freedom) as well as translate along the three axes (three additional degrees of freedom). Segments of the body, of course, are not unconstrained and—consequently—often have less than six degrees of freedom.

FIGURE 1-4

Body in anatomic position showing the x-axis, y-axis, and z-axis of the Cartesian coordinate system (the coronal, sagittal, and transverse planes, respectively).



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Rotation of a body segment is described not only as occurring around one of three possible axes but also as moving in or parallel to one of three possible **cardinal planes** (Fig. 1–4). As a segment rotates *around* a particular axis, the segment also moves in a plane that is perpendicular to its axis of rotation. Rotation of a body segment *around*

the x-axis (coronal axis) occurs in the **sagittal plane.** Sagittal plane motions are most easily visualized as front-to-back motions of a segment (e.g., flexion/extension of the upper extremity at the glenohumeral joint).

Rotation of a body segment *around* the y-axis (vertical axis) occurs in the **transverse plane.** Transverse plane motions are most easily visualized as motions of a segment parallel to the ground (e.g., medial/lateral rotation of the lower extremity at the hip joint). Transverse plane motions at a joint often occur around an axis that passes through the length of the rotating long bone, resulting in an axis that is not truly vertically oriented. Consequently, the term **longitudinal** (or **long**) **axis** is often used instead of "vertical axis." Rotation of a body segment *around* the z-axis (A-P axis) occurs in the **frontal plane.** Frontal plane (also referred to as coronal plane) motions are most easily visualized as side-to-side motions of the segment (e.g., abduction/adduction of the upper extremity at the glenohumeral joint).

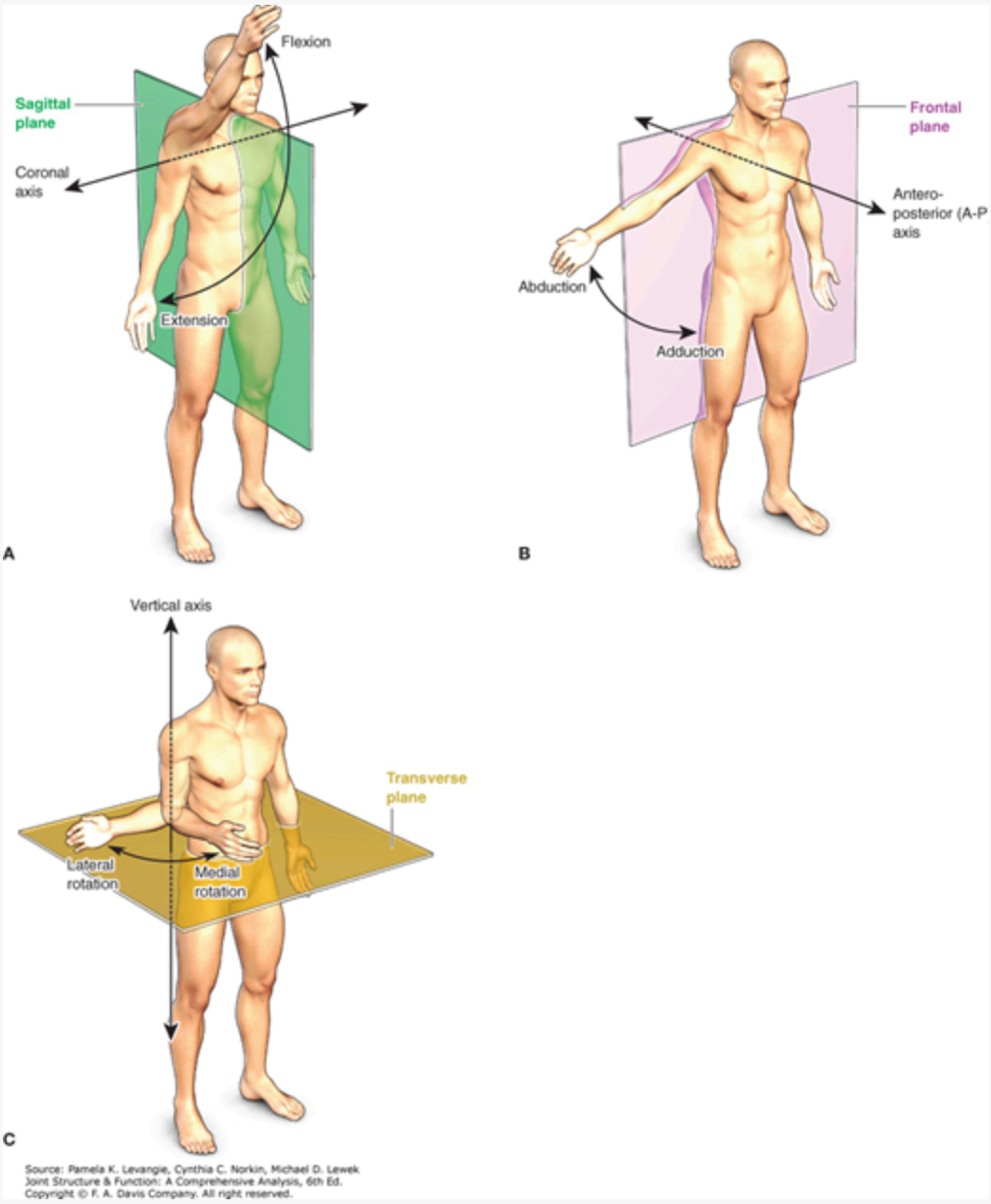
Rotation and translation of body segments are not limited to motion along or around cardinal axes or within cardinal planes. In fact, cardinal plane motions are the exception rather than the rule. Although useful for describing available motions at joints, cardinal planes and axes are an oversimplification of human motion. Much more commonly, a segment moves in three dimensions with two or more degrees of freedom.

Direction of Displacement

Even if displacement of a segment is confined to a single axis, the rotary or translatory motion of a segment around or along that axis can occur in two different directions (Fig. 1–5). For rotary motions, the direction of movement of a segment around an axis can be described as occurring in a clockwise or counterclockwise direction.

FIGURE 1-5

Anatomic definitions of motion. **A.** Flexion and extension are, respectively, forward (anterior) and backward (posterior) motions, typically around a coronal axis in the sagittal plane. **B.** Abduction and adduction are, respectively, movements away from the midline of the body and movement toward the midline of the body, typically around an A-P axis in the frontal plane. **C.** Medial and lateral rotation are, respectively, a “rolling” toward and away from the midline of the body, typically around a vertical (or longitudinal) axis in a transverse plane.



Flexion and **extension** are motions of a segment occurring around the same axis and in the same plane (uniaxial or uniplanar) but in opposite directions (Fig. 1–5A). Flexion and extension generally occur in the sagittal plane around a coronal axis, although exceptions exist (e.g., carpometacarpal flexion and extension of the thumb). **Abduction** and **adduction** of a segment occur around the A-P axis and in the frontal plane but in

opposite directions (although carpometacarpal abduction and adduction of the thumb again serve as exceptions; Fig. 1–5B). Anatomically, abduction brings the segment away from the midline of the body, whereas adduction brings the segment toward the midline of the body. When the moving segment *is* part of the midline of the body (e.g., the trunk or the head), the rotary movement is commonly termed **lateral flexion** (to the right or left).

Medial (or **internal**) **rotation** and **lateral** (or **external**) **rotation** are opposite motions of a segment that generally occur around a vertical (or longitudinal) axis in the transverse plane (Fig. 1–5C). Anatomically, medial rotation occurs as the segment moves parallel to the ground and toward the midline, whereas lateral rotation occurs opposite to that. When the segment is part of the midline (e.g., the head or trunk), rotation in the transverse plane is simply called rotation to the right or rotation to the left. The exceptions to the general rules for naming motions must be learned on a joint-by-joint basis.

Magnitude of Displacement

The magnitude of rotary motion (or angular displacement) that a body segment can move through is known as its **range of motion (ROM).** The most widely used standardized clinical method of measuring available joint ROM is goniometry, with units given in degrees. If an object rotates through a complete circle, it has moved through 360°.

Translatory motion (displacement) of a segment is quantified by the linear distance through which the object or segment is displaced. The units for describing translatory motions are the same as those for length. The unit for the *Système International* system

(SI) is the meter (or millimeter or centimeter); the corresponding unit in the U.S. system is the foot (or inch). This text will use the SI system but include a U.S. conversion when this appears to facilitate understanding (1 inch = 2.54 cm). Linear displacements of the entire body are often measured clinically. For example, the 6-minute Walk Test[5] (a test of functional status in individuals with cardiorespiratory problems) measures the distance in feet or meters that someone walks in 6 minutes.

Rate of Displacement

Although the magnitude of displacement is important, the rate of change in position of the segment (the displacement per unit time) is equally important. Displacement per unit time, regardless of direction, is known as **speed,** whereas displacement per unit time in a given direction is known as **velocity.** If the velocity is changing over time, the change in velocity per unit time is **acceleration.**

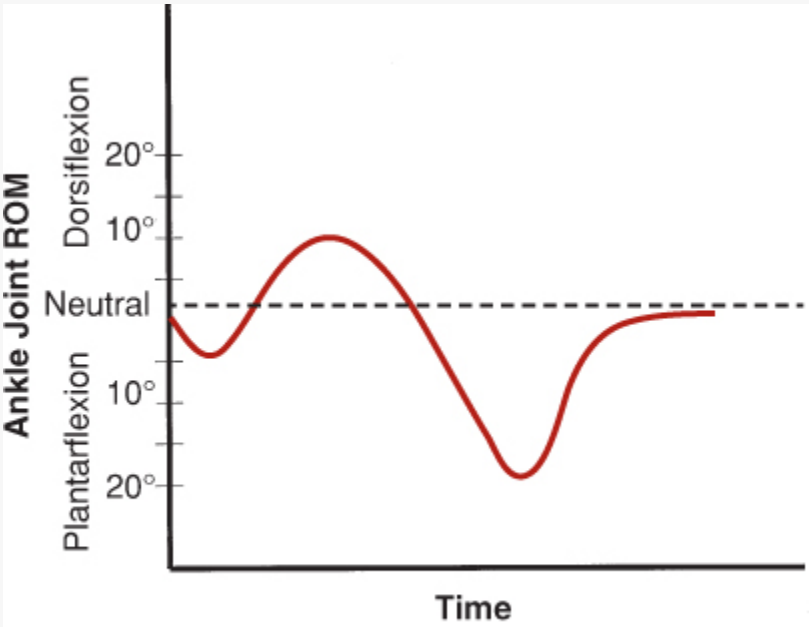
Linear velocity (velocity of a translating segment) is expressed as meters per second (m/sec) in SI units or feet per second (ft/sec) in U.S. units; the corresponding units for acceleration are meters per second squared (m/sec^2) and feet per second squared (ft/sec^2). Angular velocity (velocity of a rotating segment) is expressed as degrees per second (deg/sec), whereas angular acceleration is given as degrees per second squared (deg/sec^2).

An electrogoniometer or a three-dimensional motion analysis system can be used for documentation of the changes in displacement over time. The outputs of such systems are increasingly encountered when summaries of displacement information are presented. A computer-generated time-series plot, such as that in Figure 1–6, graphically portrays not only the angle between two bony segments (or the rotation of

one segment in space) at each point in time but also the direction of motion. The steepness of the slope of the graphed line represents the angular velocity. Figure 1–7 plots the variation in linear acceleration of a body segment (or a point on the body segment) over time without regard to changes in joint angle.

FIGURE 1-6

When a joint's range of motion is plotted on the y-axis (vertical axis) and time is plotted on the x-axis (horizontal axis), the resulting time-series plot portrays the change in joint position over time. The slope of the plotted line reflects the velocity of the joint change.

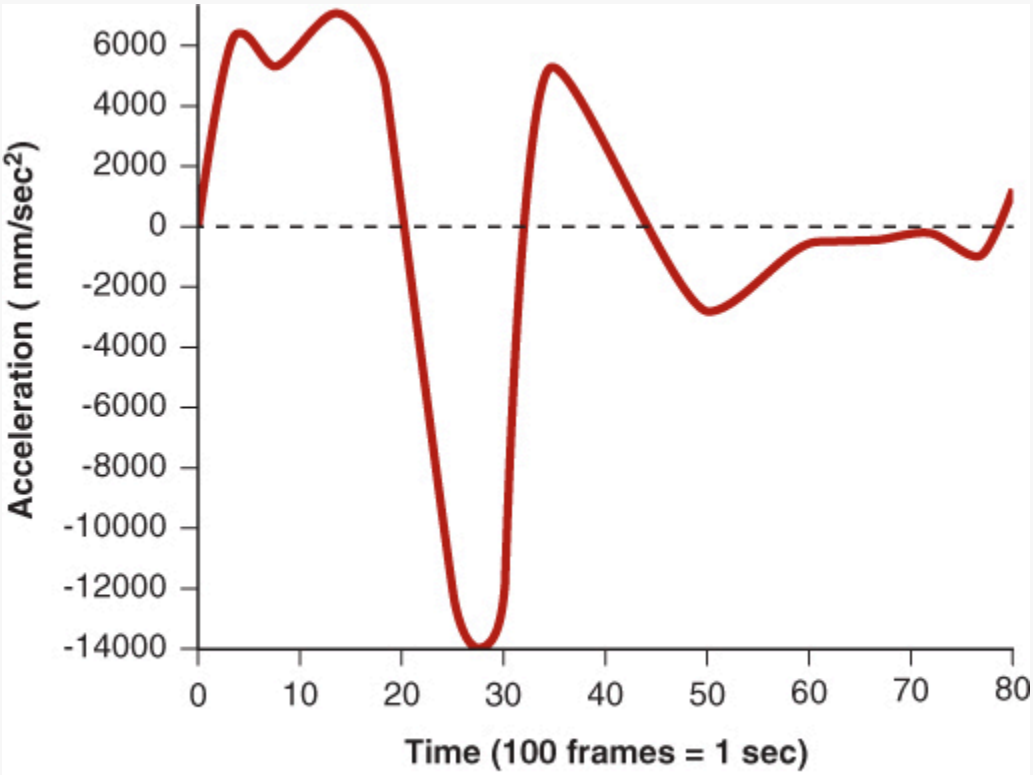


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FIGURE 1-7

Movement of a point on a segment can be displayed by plotting the

acceleration of the segment (y-axis) over time (x-axis). The slope and trend of the line represent increases or decreases in magnitude of acceleration as the movement continues. *(Courtesy of Fetters L. Boston University, 2003.)*



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Introduction to Forces

Definition of Forces

Kinematic descriptions of human movement permit us to visualize motion but do not give us an understanding of why the motion is occurring. This requires a study of forces. Whether a body or body segment is in motion or at rest depends on the forces exerted on that body. A force, simplistically speaking, is a push or a pull exerted by one object or substance on another. Any time two objects make contact with each other, they will

either push or pull on each other with some magnitude of force. The unit for a force (a push or a pull) in the SI system is the **newton (N);** the unit in the U.S. system is the **pound (lb).** The concept of a force as a push or pull can readily be used to describe the forces encountered in evaluating human motion.



Foundational Concepts 1–1

A Force

Although a force is most simply described as a push or a pull, it is also described as a "theoretical concept" because only its effects (e.g., acceleration) can be measured.[4] A force (F) on an object can be described if it is understood that the force is directly proportional both to the acceleration (a) and to the mass (m) of that object; that is,

$$\text{Force} = (\text{mass})(\text{acceleration})$$

$$\text{or } F = (m)(a)$$

Because mass is measured in kilograms (kg) and acceleration in m/sec^2, the unit for force is actually kg-m/sec^2 or, more simply, the newton (N). A newton is the force required to accelerate 1 kg at 1 m/sec^2 (the pound is correspondingly the amount of force required to accelerate a mass of 1 slug at 1 ft/sec^2). A "slug" is a rarely used term for a unit of mass in the U.S. system.

External forces are pushes or pulls on the body that arise from sources outside the body. **Gravity (g),** the attraction of the earth's mass to another mass, is an external force that under normal conditions constantly affects all objects. The **weight (W)** of an object is the pull of gravity on the object's mass with an acceleration of 9.8 m/sec^2 (or 32.2 ft/sec^2) in the absence of any resistance; that is:

$$\text{weight} = (\text{mass})(\text{gravity})$$

$$\text{or } W = (m)(g)$$

Because weight is a force, the appropriate unit is the newton (or pound). However, it is common to see weight given in **kilograms (kg),** although the kilogram is more correctly a unit of mass. In the U.S. system, the pound is commonly used to designate mass when it is appropriately a force unit (1 kg = 2.2 lb).



Expanded Concepts 1–1

Force and Mass Unit Terminology

Force and mass units are often used incorrectly in the vernacular. The average person using the metric system expects a scale to show weight in kilograms rather than in newtons. In the United States, the average person correctly thinks of "weight" in pounds but also—less correctly—considers the pound to be a unit of mass. Because people commonly tend to think of mass in terms of weight (the force of gravity acting on the mass of an object), the pound is often used to represent the mass of an object in the United States.

One attempt to maintain common usage while clearly differentiating force units from mass units for scientific purposes is to designate "lb" and "kg" as mass units and to designate the corresponding force units as "lbf" (pound-force) and "kgf" (kilogram-force).[3,4] When the kilogram is used as a force unit:

$$1 \text{ kgf} = 9.8 \text{ N}$$

When the pound is used as a mass unit:

$$1 \text{ lb} = 0.031 \text{ slugs}$$

These conversions assume an unresisted acceleration of gravity of 9.8 m/sec^2 or 32.2 ft/sec^2, respectively. It might be obvious to the reader who is unfamiliar with the kgf and the slug that these terms have not come into common usage.

The distinction between a measure of mass and a measure of force is important because mass is a scalar quantity (without action line or direction), whereas the newton and pound are measures of force and have vector characteristics. In this text, we will consistently use the terms *newton* and *pound* as force units and the term *kilogram* as

the corresponding mass unit. The term “slug” is not in common usage and will not be used further in the text.

Because gravity is the most consistent of the forces encountered by the body, gravity should be the first force to be considered when attempting to identify the potential forces acting on a body segment. However, gravity is only one of an infinite number of external forces that can affect the body and its segments. Examples of other external forces that may exert a push or pull on the human body or its segments are wind (the push of air on the body), water (the push of water on the body), other people (the push or pull of an examiner on John Alexander’s leg), and other objects (the push of floor on the feet, the pull of an ankle weight on the leg). A critical point is that the forces on the body or any one segment must come from something that is touching the body or segment. The major apparent exception to this rule is the force of gravity. However, we can circumvent this exception by using the conceit that gravity (the pull of the earth) “contacts” all objects on earth, thereby allowing a standing rule that *all forces on a segment must come from something that is contacting that segment* (including gravity). The obverse also holds true: *anything that contacts a segment must create a force on that segment,* although the magnitude may be small enough to disregard.



Foundational Concepts 1–2

Primary Rules of Forces

- All forces on a segment must come from something that is contacting that segment.
- Anything that contacts a segment *must* create a force on that segment (although the magnitude may be small enough to disregard).
- Gravity can be considered to be “touching” all objects.

Internal forces are those forces that act on structures of the body and arise from the body's own structures (i.e., the contact of two structures within the body). A few common examples are the forces produced by the muscles (the pull of the biceps brachii on the radius), the ligaments (the pull of a ligament on the bones to which it attaches), and the bones (the push of one bone on another bone at a joint). External forces can either facilitate or restrict movement. Internal forces are most readily recognized as essential for *initiation* of movement. However, it should be apparent that internal forces also control or counteract movement produced by external forces, as well as counteracting other internal forces. Much of the presentation and discussion in subsequent chapters of this text relate to the interactive role of internal forces in producing or controlling joint rotations as well as in maintaining the integrity (stability) of joint structures against the effects of external forces and other internal forces.

Force Vectors

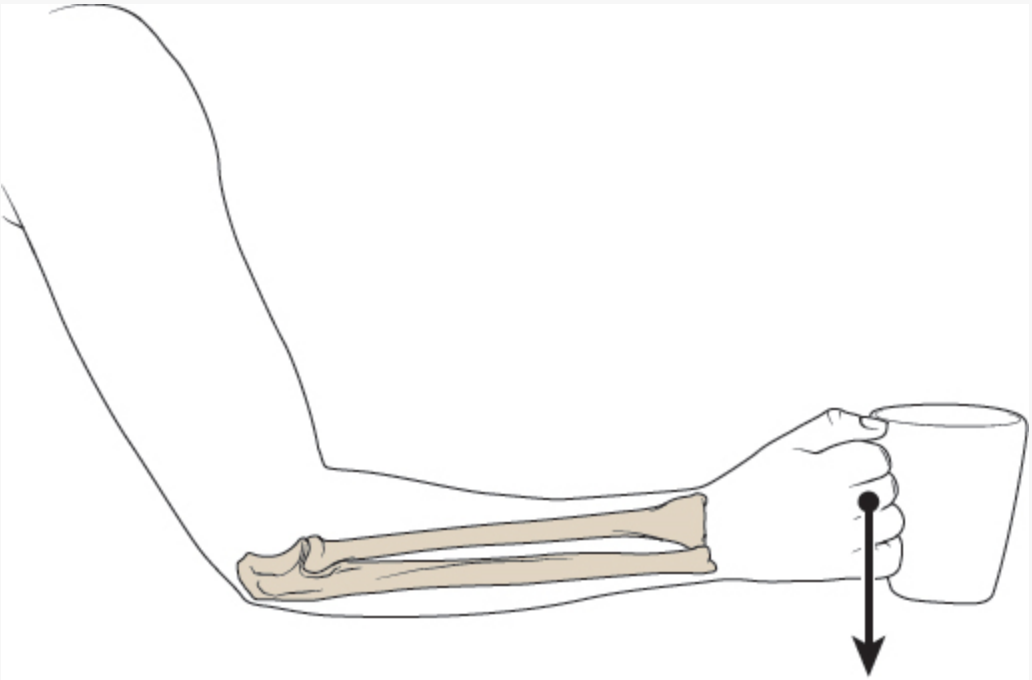
All forces, regardless of the source or the object acted on, are **vector** quantities. A force is represented by an arrow (vector) that (1) has its base on the object being acted on (the point of application), (2) has a shaft and arrowhead in the direction of the force being exerted (direction/orientation), and (3) has a length representing the amount of force being exerted (magnitude). As we begin to examine force vectors (and at least throughout this chapter), the *point of application* (*base*) of each vector in each figure will be placed on the segment or object to which the force is applied—which is generally also the object under discussion.

The contact of a coffee mug on the forearm-hand and the pull of a muscle on the forearm-hand serve as examples of external and internal forces, respectively. Figure 1–8

shows a forearm-hand segment with the hand holding a coffee mug (an external force). Because the mug makes contact with the forearm-hand segment, the mug must exert a force (in this case, a downward pull) on the segment. The force, called mug-on-forearm/hand (MFh), is represented by a vector. The point of application on the forearm-hand segment is at the hand where the mug exerts its pull; the action line and direction indicate the direction of the pull and the angle of pull of the mug in relation to the forearm-hand; and the length is drawn to represent the magnitude of the pull (equivalent to the weight) of the mug. The force MFh is an external force because the weight of the mug is not part of the body, although it contacts the body. Figure 1–9 shows the force of an elbow flexor (an internal force) pulling on the forearm-hand segment. The point of application is at the attachment of the muscle on the forearm, and the orientation and direction are toward the center of the muscle (a "pull" is always toward the source of the force). The force is called muscle-on-forearm/hand (represented by the vector MFh). Although the designation of a force as "external" or "internal" may be useful in some contexts, the rules for drawing (or visualizing) forces are the same for external forces, such as the mug, and internal forces, such as the muscle.

FIGURE 1-8

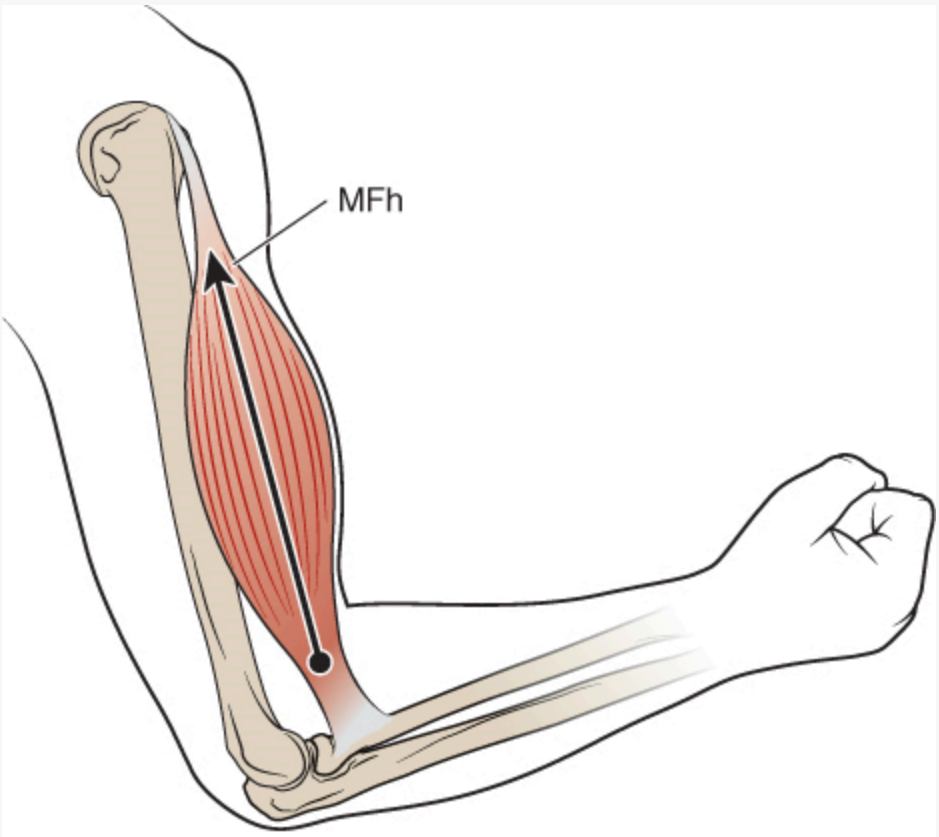
Vector representation of the pull of the weight of the mug on the forearm-hand segment (mug-on-forearm/hand [MFh]), with a magnitude proportional to the mass and equivalent to the weight of the mug.



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FIGURE 1-9

Vector muscle-on-forearm/hand (MFh) represents the pull of a muscle on the forearm-hand segment.



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Vectors can represent either a push or a pull on a segment depending on the point of application and the source of the force. Figure 1–10 shows the force vector of both a *push* and a *pull* of an external resistance on the forearm. In Figure 1–10A, the push vector is directed away from the source of the manual resistance; in Figure 1–10B, the pull vector is directed toward the source of the manual resistance. Both vectors look identical and would have an identical effect on the forearm-hand segment if the magnitudes of the force were the same.

FIGURE 1-10

Push vs. pull forces. **A.** The clinician's manual resistance to the forearm is applied as a push whereas in **B.** the clinician's manual resistance is applied to the forearm as a pull. Both vectors look similar and would be indistinguishable as a push vs. a pull if the clinician's hand was removed from each photo.



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Foundational Concepts 1–3

Force Vectors

Force vectors are characterized by:

- A **point of application** *on the object acted upon.*
- An action line and **direction/orientation** indicating a *pull toward* the source object or a *push away from* the source object, at a given angle to the object acted upon.
- A length that represents, and may be drawn proportional to, its **magnitude** (the quantity of push or pull).
- A length that may be extended to assess the relation between two or more vectors or to assess the relation of the vector to adjacent objects or points.
- A force **name** that uses the *"X-on-Y"* where the first part of the force name ("X") will always identify the *source* of the force and the second part of the force name ("Y") will always identify the object or segment that is acted upon.

Force of Gravity

As already noted, gravity is one of the most consistent and influential forces that the human body encounters in posture and movement. For that reason, it is useful to first consider gravity when examining forces. As a vector quantity, the force of gravity can be fully described by point of application, action line/direction/orientation, and magnitude. The force of gravity acting on an object or segment is considered to have its point of application at the center of mass (CoM) or **center of gravity (CoG)** of that object or segment—the hypothetical point at which all the mass of the object or segment appears to be concentrated. Every object or segment can be considered to have a single center of mass.

In a symmetrical object, the center of mass is located in the geometric center of the object. In an asymmetrical object, the center of mass will be located toward the heavier end because the mass must be evenly distributed around the center of mass. In cases where the center of mass lies outside the object, it is still the point from which the force of gravity *appears* to act. The center of mass of an object can be approximated by thinking of it as the balance point of the object (assuming you could balance the object

on one finger).

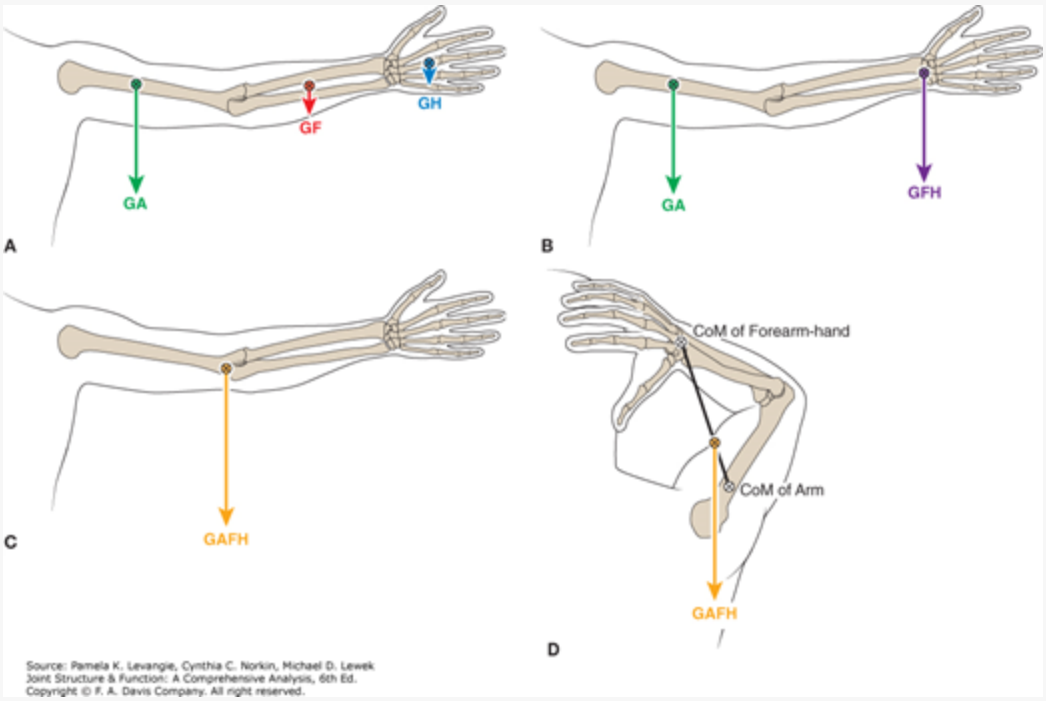
Although the direction and orientation of most forces vary with the source of the force, the force of gravity acting on an object is *always* vertically downward toward its source—the center of the earth. The gravitational vector is commonly referred to as the **line of gravity (LoG).** The length of the line of gravity can be drawn to scale (as in a free body diagram where length is determined by the magnitude of the force) or it may be extended (like any vector) when the relationship of the vector to other forces, points, or objects is being explored. The line of gravity can best be visualized as a string with a weight on the end (a plumb line), with the string attached to the center of mass of the object.

Segmental Centers of Mass and Composition of Gravitational Forces

Each segment in the body can be considered to have its own center of mass and line of gravity. Figure 1–11A shows the gravitational vectors acting at the centers of mass of the arm, the forearm, and the hand segments (vectors GA, GF, and GH, respectively) where the hand is treated as a single solid segment. It is often useful, however, to consider two or more segments as a single rigid segment (such as the leg-foot segment in the patient application). In Figure 1–11B, the gravitational forces on the forearm and hand have been combined into a single vector (GFH) acting at the combined center of mass of these two segments. In Figure 1–11C, the gravitational force for all three segments is represented by a single vector (GAFH) applied at the center of mass of all three segments taken together.

FIGURE 1-11

Gravity acts at the center of mass of each segment—or may be shown acting at a center that combines the mass of two or more segments taken together. **A.** Gravity is shown acting separately at the centers of mass of the arm (GA), forearm (GF), and hand (GH) segments. **B.** Gravity acting on the combined masses of the forearm and hand can be shown as a single resultant vector, GFH, with a point of application at the new combined center of mass of those two segments. **C.** Gravity acting on the combined masses of the arm-forearm-hand segment is shown as a single resultant vector, GAFH, applied at the new center of mass of all three segments. **D.** When the masses of the arm and forearm-hand segments are rearranged, the point of application of the gravitation vector, GAFH, shifts to the new center of mass that still lies in between and along a line connecting the original centers of mass.



When the masses of two segments are combined (e.g., forearm and hand segments as in Figure 1–11B or the arm and forearm-hand segments as in Figure 1–11C), the new point of application (the new center of mass) will be located between and in line with the original two segmental centers of mass. When the linked segments are not equal in mass, the new center of mass will lie closer to the heavier segment. The new vector will have the same effect on the combined segment(s) as on the original vectors and is known as the **resultant force.** The process of combining two or more forces into a single resultant force is known as **composition of forces.** When segments are rearranged as in Figure 1–11D, the rule for identifying the location of the combined center of mass (point of application of GAFH) remains the same but may actually lie outside the body; the magnitude of GAFH remains unchanged.

Center of Mass of the Human Body

When all the segments of the body are combined and considered as a single rigid object in anatomic position, the center of mass of the body lies approximately anterior to the second sacral vertebra (S2). The precise location of the center of mass for a person in the anatomic position depends on the proportions (weight distribution) of that person. In Figure 1–12, the line of gravity is between the person's feet (**base of support [BoS]**) as the person stands in anatomic position; the line of gravity is parallel to the trunk and limbs. If the person is lying down (still in anatomic position), the line of gravity projecting from the center of mass of the body will lie perpendicular to the trunk and limbs, rather than parallel as it does in the standing position. In reality, of course, a person is not rigid and does not remain in anatomic position. Rather, a person is constantly rearranging segments in relation to each other. With each rearrangement of body segments, the location of the individual's center of mass will potentially change.

FIGURE 1-12

The center of mass (CoM) of the human body lies approximately at S2, anterior to the sacrum (inset). The extended line of gravity (LoG) lies within the base of support (BoS) between the feet.



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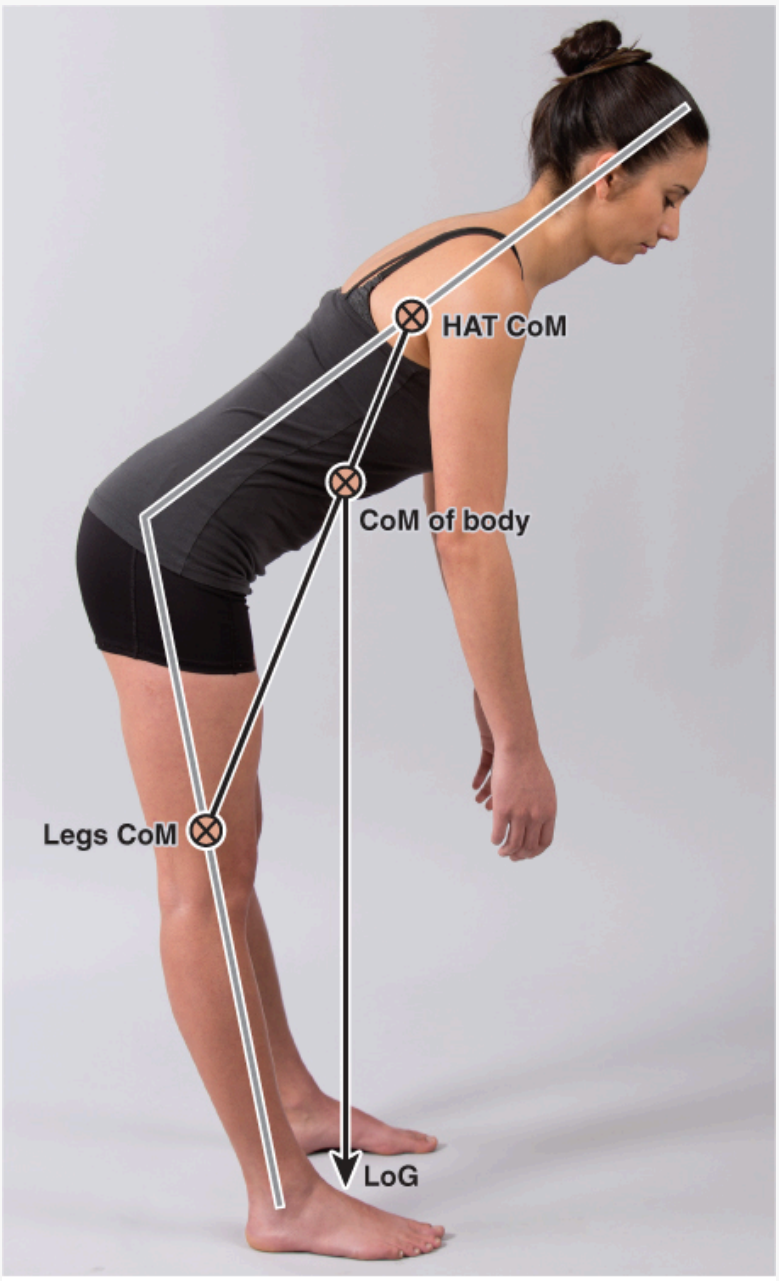
Example 1–1

If a person is considered to be composed of a rigid upper body (head-arms-trunk [HAT]) and a rigid lower limb segment, each segment will have its own center of mass. If the trunk is inclined forward (see Fig. 1–13), the segmental masses remain

unchanged but the composite center of mass of the rearranged body segments shifts from its original location within the pelvis just anterior to S2 to a new location on a line between the original two centers of mass. Because the head, arms, and trunk together typically have twice the mass of the lower extremities, the new center of mass will be closer to HAT. The relocated center of mass is physically located outside the body, with the line of gravity correspondingly shifted forward within the base of support.

FIGURE 1-13

Rearrangement of the head, arms, and trunk (HAT) in relation to the lower extremities (legs) relocates the center of mass (CoM) and shifts the line of gravity (LoG) forward in the base of support.



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Center of Mass, Line of Gravity, and Stability

In Figure 1–13, the line of gravity falls toward the front of the feet that serve as the base of support. For an object to be stable, *the line of gravity must fall within the base of support.* When the line of gravity is outside the base of support, the object will be

unstable and will start falling.

When the base of support of an object is large, the line of gravity is less likely to be displaced outside the base of support and the object, consequently, is more stable. When a person stands with his or her legs spread apart (Fig. 1–14), the base is wide and the trunk can move side-to-side a good deal without displacing the line of gravity from the base of support and without the person falling over. Whereas the center of mass remains in approximately the same place within the body as the trunk shifts to each side, the *line* of gravity moves within the wide base of support. It is useful here to think of the line of gravity as a plumb line. As long as the plumb line does not leave the base of support, the person should not fall over.

FIGURE 1-14

A wide base of support permits a wide excursion of the line of gravity (LoG) without the LoG falling outside the base of support (the two widely spaced feet).



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Just as an object is more stable when the base of support is large, an object is also stable when its center of mass is low (close to the ground). When the center of mass is low, the vertical line of gravity projecting from the center of mass to the base of support will be shorter and, therefore, less likely to “swing” outside the base of support.

 Foundational Concepts 1–4

Stability of an Object or the Human Body

- The larger the base of support of an object, the greater the stability of that object.
- The closer the object's center of mass is to the base of support, the more stable the object.
- An object cannot be stable unless its line of gravity is located within its base of support.

Introduction to Statics and Dynamics

The primary concern when looking at forces that act on the body or a particular segment of the body is the effect that those forces will have on the body or segment. If all the forces acting on a segment are "balanced" (a state known as **equilibrium**), the segment will remain at rest or in uniform motion. If the forces are not "balanced," the segment will accelerate. **Statics** is the study of the conditions under which objects remain at rest. **Dynamics** is the study of the conditions under which objects move. Isaac Newton's first two laws govern whether an object is static or dynamic.

Newton's Law of Inertia

Newton's first law, the **law of inertia,** identifies the conditions under which an object is in equilibrium. **Inertia** is the property of an object that resists both the initiation of linear motion and a change in linear motion and is directly proportional to its mass. Similarly, **moment of inertia** is the property of an object that resists rotary motion and changes in rotary motion. The term *moment* (also known as torque) refers to the magnitude of rotation produced by a force. The law of inertia states that an object will remain at rest or in uniform motion unless acted on by an unbalanced force (producing

translation) or torque (producing rotation). If the forces or torques acting on an object are balanced, the object can be motionless **(static equilibrium).** However, an object acted upon by balanced forces or torques may also be in uniform motion, that is, moving with a constant speed and direction. Velocity (rate of change) is a vector quantity that describes both speed and direction/orientation. An object in equilibrium can have a velocity of any magnitude, but its velocity will remain unchanged. When the velocity of an object is constant but not zero, the object is moving and is in **dynamic equilibrium.** An object in dynamic equilibrium can be moving linearly (translatory motion), around an axis (rotary motion), or in a combination of both translation and rotation (e.g., curvilinear motion). In joints of the body, dynamic equilibrium infrequently occurs. Therefore, within the scope of this text, equilibrium will be simplified to mean an object at rest (in static equilibrium) unless otherwise specified.

Newton's law of inertia (or law of equilibrium) can be restated thus: for an object to be in equilibrium, the sum of all the forces (F) and the sum of all the torques (τ) applied to that object must be zero.

$$\Sigma \mathbf{F} = \mathbf{0} \text{ and } \Sigma \tau = \mathbf{0}$$

The equilibrium of an object is determined *only by forces or torques applied to that object*—that is, with points of application on that object. There is no restriction on the number of forces or torques that can be applied to an object in equilibrium, although there must be more than one force or torque. If one (and only one) force or torque is applied to an object, the sum of the forces or torques cannot be zero. When the sum of the forces or torques acting on an object is not zero ($F \neq 0$ or $\tau \neq 0$), the object *must* be accelerating.

Newton's Law of Acceleration

The magnitude of acceleration of a moving object is defined by Newton's second law, the **law of acceleration.** Newton's second law states that the linear acceleration (a) or angular acceleration (α) of an object is proportional, respectively, to the net unbalanced forces (F_{unbal}) or torques (τ_{unbal}) acting on the object. Further, acceleration is inversely proportional to the mass (m) or moment of inertia (*I*) of that object:

$$a = \frac{F_{unbal}}{m}$$

$$\alpha = \frac{\tau_{unbal}}{I}$$

Note that the concept of "moment of inertia (*I*)" is far less intuitive than the concept of mass. Moment of inertia is defined as $I = mr^2$, where m is the mass of an object and r is the distance that mass lies from the axis of rotation. Therefore, resistance to rotary motion is dependent both on the total mass of an object *and* on the spatial distribution of that mass. In other words, a heavy object has more rotary inertia than a lighter object, and an object with mass further from the axis of rotation will have more rotary inertia than an object with mass focused closer to the axis of rotation.

An object acted upon by a net unbalanced force or torque *must* be accelerating, and the acceleration of the object will be in the direction of the net unbalanced force or torque. A net unbalanced *force* will produce translatory motion; a net unbalanced *torque* will produce rotary motion; and a combination of unbalanced force and torque will produce general (e.g., curvilinear) motion.



Foundational Concepts 1–5

Applying the Law of Acceleration (Inertia)

To put the law of acceleration into simple words: a large unbalanced push or pull (F_{unbal}) applied to an object of a given mass (m) will produce more linear acceleration (a) than a small unbalanced push or pull. Similarly, a given magnitude of unbalanced push or pull on an object of large mass or moment of inertia will produce less acceleration than that same push or pull on an object of smaller mass or moment of inertia. From the law of acceleration, it can be seen that inertia (a body's or object's resistance to change in velocity) is resistance to acceleration and is proportional to the mass of the body or object. The greater the mass or moment of inertia of an object, the greater the magnitude of net unbalanced force or torque needed either to get the object moving or to change its motion. A very large woman in a wheelchair has more inertia than a small woman in a wheelchair; an aide must exert a greater push on a wheelchair with a large woman in it to get the chair in motion than on the wheelchair with a small woman in it.

Translatory Motion in Linear and Concurrent Force Systems

The process of composition of forces is used to determine whether a net (unbalanced) force (or forces) exists on a segment because this will determine whether the segment is at rest or in motion. Furthermore, the direction/orientation and location of the net unbalanced force or forces determine the type and direction of motion of the segment. The process of composition of forces was oversimplified in Figures 1–11 and 1–13. The process depends on the relationship of the forces to each other; that is, whether the forces are in a linear, concurrent, or parallel force system. When vectors applied to the same segment have action lines that lie in the same plane, and act in the same line (collinear and coplanar), these two vectors are part of a **linear force system.** If two forces are collinear but not coplanar (parallel to each other) or coplanar but not collinear

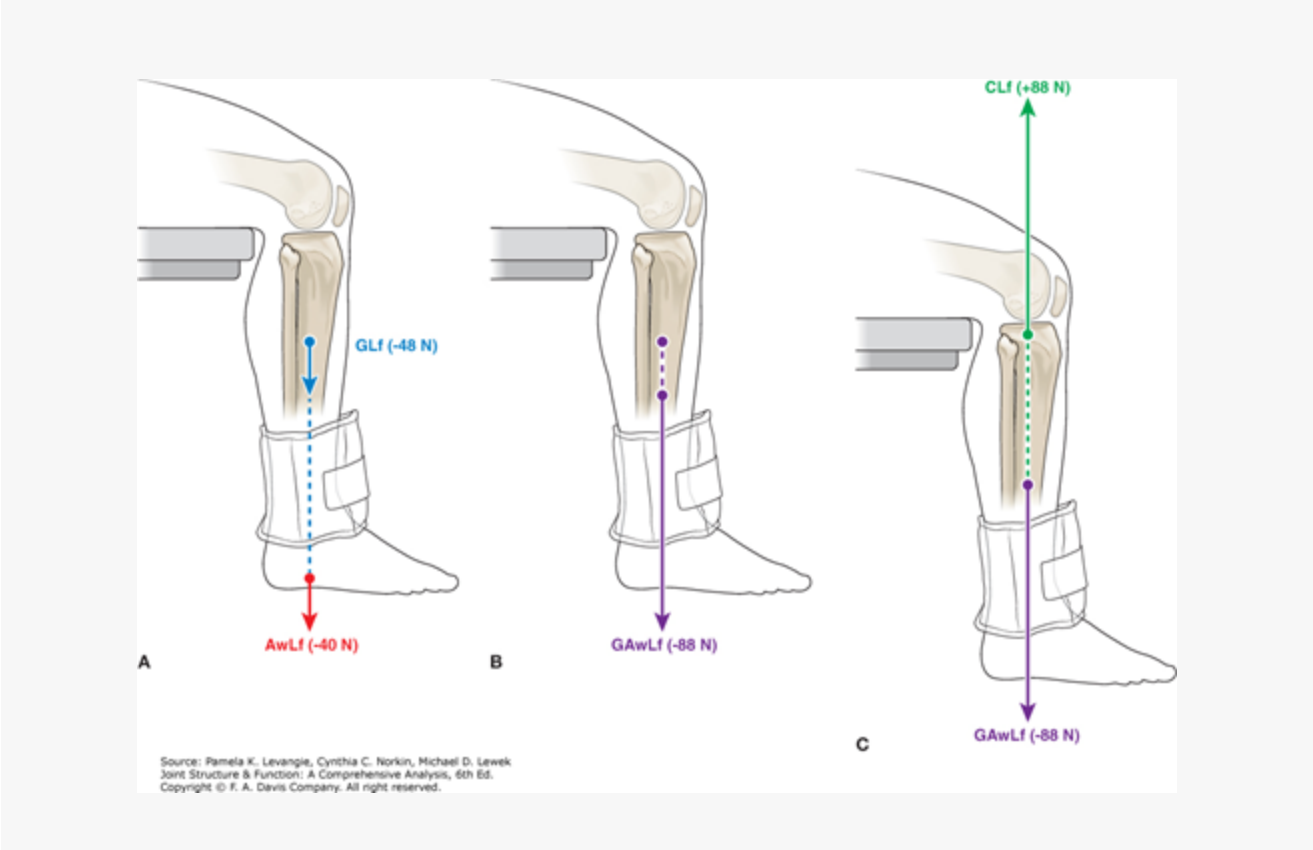
(lying at angles to each other), the forces are not part of the same linear force system.

Linear Force Systems

A linear force system exists whenever two or more forces acting *on the same segment* lie in the same line (collinear) and in the same plane (coplanar); their action lines, if extended, would completely overlap (Fig. 1–15A). Forces in a linear force system are assigned positive or negative signs. Forces applied up (y-axis), forward or anterior (z-axis), or to the right (x-axis) will be assigned positive signs, whereas forces applied down, back or posterior, or to the left will be assigned negative signs.

FIGURE 1-15

Resultant forces in a linear force system. **A.** The forces of gravity-on-leg/foot (GLf) and ankle weight-on-leg/foot (AwLf) are in the same linear force system because they are applied to the same object and are both collinear and coplanar. **B.** GAwLf is the resultant of GLf and AwLf, with a magnitude of –88 N (the sum of the original two forces of –48N and –40N) and with a point of application between those of GLf and AwLf (slightly toward the force of greater magnitude). **C.** A force like capsule-on-femur (CLf) must exist on the leg-foot so that the sum of the linear forces is zero and the leg-foot segment is at rest.



Determining Resultant Forces in a Linear Force System

The resultant, or net effect, of all forces that are part of the same linear force system can be composed into a single resultant (net) vector (Fig. 1–15B). The resultant vector has an action line in the same line and plane as that of the original composing vectors; the magnitude is equivalent to the arithmetic sum of the composing vectors and is in the direction of the net force. Because the vectors in the same linear force system are all collinear and coplanar, the point of application of the resultant vector will lie along the common action line of the composing vectors.

A net force that moves a bony segment away from its adjacent bony segment is known as a **distraction force.** A distraction force tends to cause a separation between the bones that make up a joint. In Figure 1–15B, GAwLf is the only force shown acting on the leg-foot and would appear to indicate that the leg-foot segment is accelerating downward

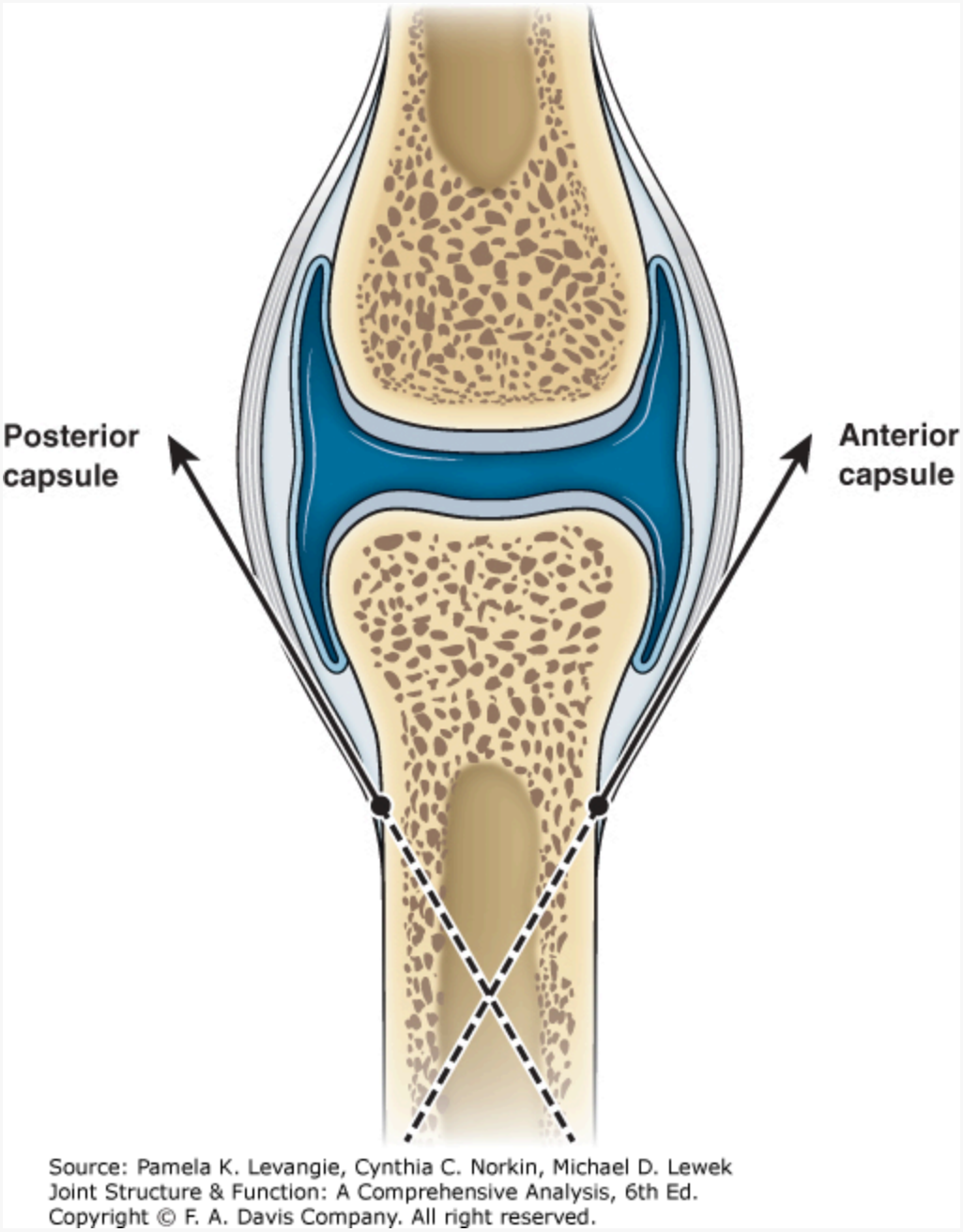
away from the femur. At least one additional force must act on the leg-foot segment for it to be at rest because an object cannot be at rest unless the sum of the forces is zero. That force must come from something that is touching the leg-foot segment.

The two bones in a synovial joint (e.g., the knee joint) are connected (and, therefore, "touched") by a joint capsule made of connective tissue. Although we are over-simplifying for now, the force of the capsule ligaments (capsule-on-leg/foot) can pull up on the leg-foot segment (**Fig. 1–15C**) along the same line as GAwLf and with a force that is equal in magnitude and opposite direction to GAwLf; thus, the net of forces acting on the leg-foot segment is zero and the leg-foot segment will be in equilibrium.

The joint capsule does not exert the simplified pull on the bone shown in Figure 1–15C. In reality the capsule *surrounds* the adjacent bones in three dimensions, acting like a sleeve that surrounds the joint, with the sleeve attaching circumferentially both to the proximal joint segment and the distal joint segment. The force of the capsule, therefore, would be a set of forces applied around the periphery of the adjacent articular surfaces. **Figure 1–16** shows a schematic drawing of the capsule of a generic synovial joint on cross-section. The vector arrows for the pulls of the anterior and posterior capsules must follow the fibers of the capsule at their respective points of application and continue in a straight line. *A vector is always a straight line*; the vector continues in the same straight line even if the fibers of the capsule change direction beyond their bony attachment. Even in this two-dimensional representation, it is apparent that the anterior and posterior fibers of the capsule will pull (capsules cannot "push") on the distal segment at different places with forces that lie at angles (rather than in line) with each other.

FIGURE 1-16

This schematic cross-section of a generic synovial joint shows the pull of the anterior and posterior capsule on the distal segment of the joint. The two forces are applied to the same object but lie at angles to each other and, if extended, intersect. These forces form a concurrent force system.



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The forces shown in Figure 1–16 are part of a **concurrent force system.**

Concurrent Force Systems

It is quite common (and perhaps nearly universal in the human body) for forces to be applied to the same object but with action lines that lie at angles to each other. To

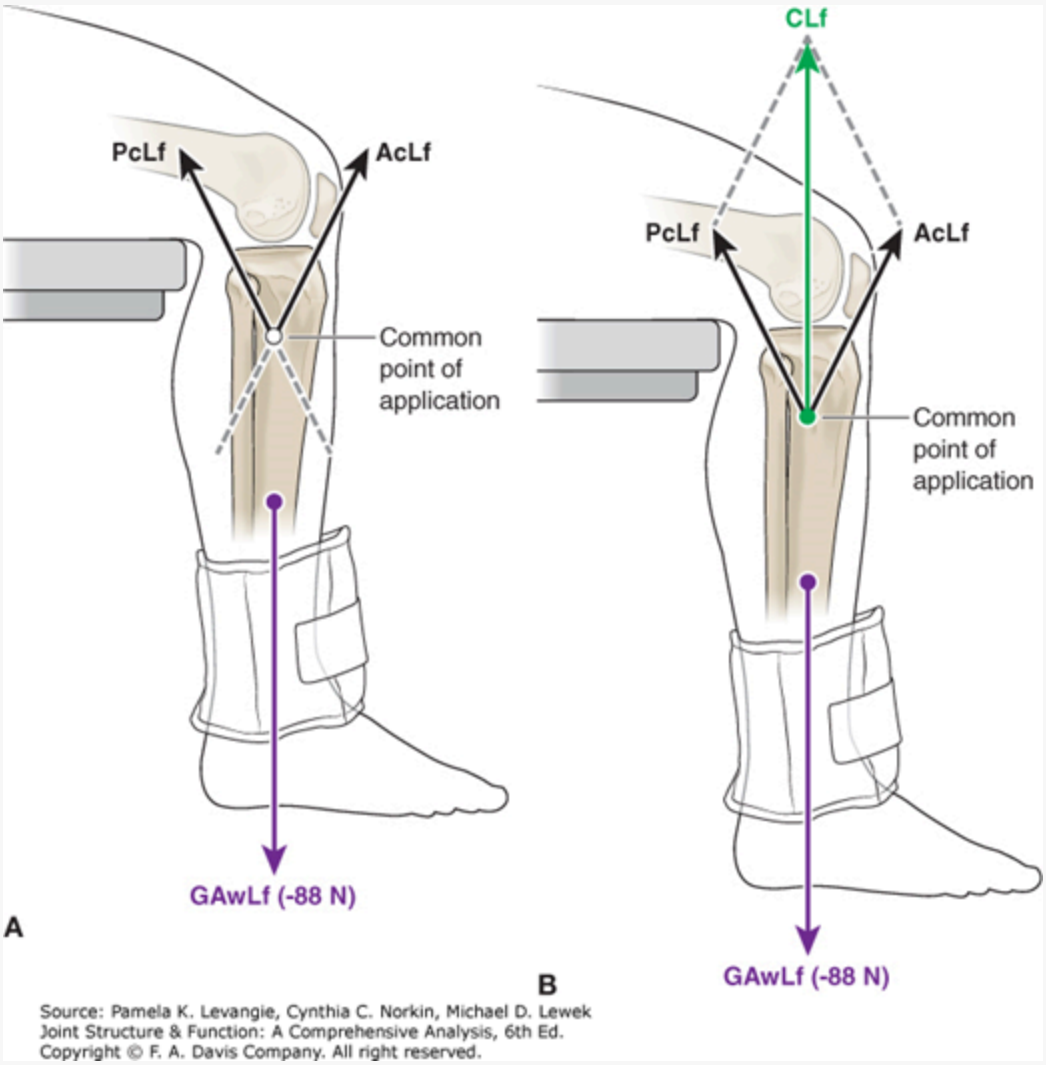
understand the net effect (resultant) of two or more forces acting on a segment in a concurrent force system, a different method of composition of forces is needed than is used for composition of forces in a linear force system. Any two forces in a concurrent force system can be composed into a single resultant force through a graphic process known as **composition by parallelogram.**

Determining Resultant Forces in a Concurrent Force System

In composition by parallelogram, two forces that are part of the *same* concurrent force system (more than one can exist on an object) are taken at a time. **Figure 1–17A** shows John Alexander's leg-foot segment with the resultant GAwLf as shown in Figure 1–15B with the addition of the forces of the anterior capsule on leg-foot (AcLf) and posterior capsule on leg-foot (PcLf). Vectors AcLf and PcLf are extended to identify their common point of application and their relationship to each other. In **Figure 1–17B**, vectors AcLf and PcLf (now starting from their common point of application) form two sides of a parallelogram, with the parallelogram completed by adding dotted lines *parallel* to AcLf and to PcLf as shown in the figure. The resultant (CLf) is the diagonal of the parallelogram with the same point of application as the original vectors and with its length determined by the intersection of the two dotted lines. Note that the resultant CLf in Figure 1–17B is essentially identical to CLf in 1-15C, indicating that the simplification used in that earlier figure was a reasonable representation of the combined pulls of the anterior and posterior fibers of the joint capsule on the leg-foot segment, albeit still an over-simplification of the pull of a circumferential capsule.

FIGURE 1-17

Composition of concurrent forces. **A.** The vectors of the anterior capsule (AcLf) and posterior capsule (PcLf) acting on the leg-foot segment can be extended to identify their common point of application. **B.** With AcLf and PcLf shown now as acting from their common point of application, the remaining two sides of the parallelogram are drawn. The resultant pull of AcLf and PcLf is the diagonal of the parallelogram (CLf). Capsule on the leg-foot (CLf) has the same effect on the leg-foot segment as do the combined forces of AcLf + PcLf.



The magnitude of the resultant force of two concurrent forces has a fixed proportional relationship to the original two forces. The relationship is dependent both on the magnitudes of the composing vectors *and* the angle between (orientation of) the composing vectors. The magnitude of the resultant of two concurrent forces will always be smaller than the sum of the composing forces (e.g., CLf < AcLf + PcLf in Fig. 1–17B) because a single push or pull is more efficient than two divergent pushes or pulls, although the net effect is the same.

The concept of composition by parallelogram can provide an understanding (and, perhaps, a visualization) of the net effect of two concurrent forces. If the magnitudes of the composing forces and the angle at which the composing forces intersect are known, trigonometric functions can be used to determine the magnitude of the resultant. Because the angle values are rarely known outside a research setting, the mathematic (trigonometric) solution is conceptually valuable but not as practical in a clinical situation as the potential "visual" solution of composition by parallelogram.



Expanded Concepts 1–2

Trigonometric Solution to Composition by Parallelogram

Let us assume that AcLf and PcLf (originally shown in Fig. 1–17A) each have a magnitude of 51 N and that the vectors are at a 60° angle (6) to each other (**Fig. 1–18**). A parallelogram still needs to be drawn and is completed by forming two lines parallel and opposite to AcLf and PcLf ($AcLf_1$ and $PcLf_1$, respectively); the resultant (CLf) is drawn as the diagonal of the parallelogram. That diagonal vector will divide the parallelogram into two equal triangles. The magnitude of the resultant vector (CLf) can be determined using the law of cosines, which permits calculation of the length of the side of a triangle that is opposite to a known angle when the triangle is not a right triangle.

The reference triangle (shaded) is formed by PcLf, $AcLf_1$, and CLf. To apply the law of cosines, angle β must be known because vector CLf (whose length we are solving for) is the "side opposite" that angle. We already identified the angle (6) in Figure 1–18 as 60°. If PcLf is extended (as shown by the red dotted line in Fig. 1–18), the angle θ is replicated because AcLf is parallel to AcLf so it has the same angular relationship to PcLf. Angle β will be the complement of angle α, or:

$$\beta = 180° - 60° = 120°$$

By substituting the variables given in the example, the magnitude of the resultant, CLf, can be solved for by using the following equation:

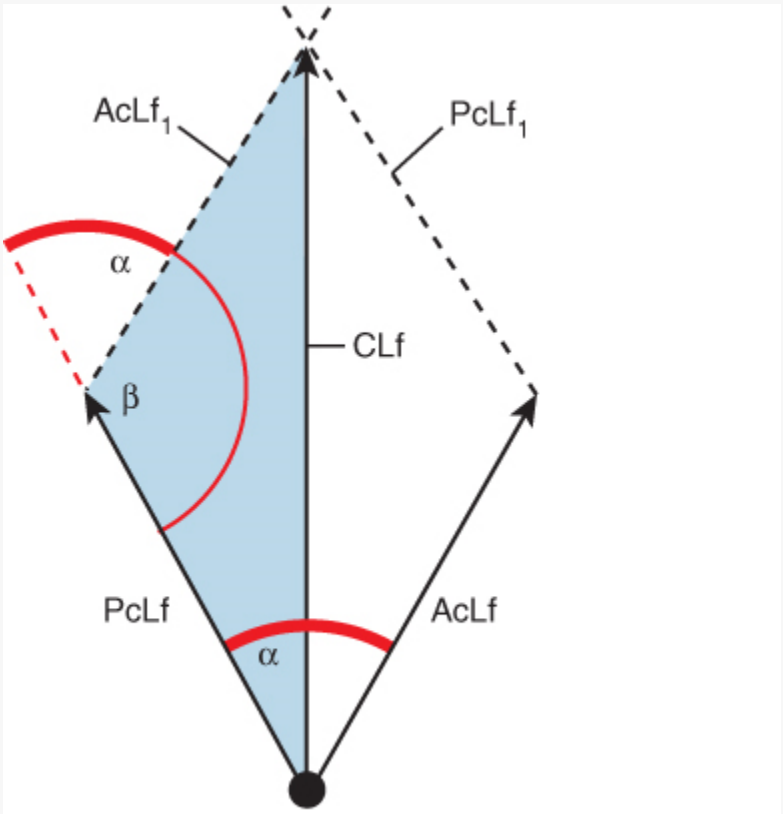
$$CLf = \sqrt{PcLf^2 + AcLf^2 - 2(PcLf)(AcLf)(\cos\beta)}$$

If the value of 51 N is entered into the equation for both PcLf and AcLf and an angle of

120° is used, vector CLf = 88 N.

FIGURE 1-18

The law of cosines for triangles is used to compute the magnitude of CLf, given the magnitudes of AcLf and PcLf, as well as the angle of application (α) between them. The relevant angle (β) is the complement of angle α (180° – α).



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Newton's Law of Reaction

Newton's third law (the law of reaction) is commonly stated as follows: for every action, there is an equal and opposite reaction. In other words, when an object applies a force to a second object, the second object *must* simultaneously apply a force equal in

magnitude and opposite in direction to the first object. These two forces that are applied to the two contacting objects are an **interaction pair** and can also be called **action-reaction** (or simply **reaction**) **forces.**



Expanded Concepts 1–3

Action-Reaction Pairs

In Figure 1–17B we see the force vector of capsule-on leg/foot (CLf) that is due to the pull of the capsule on the leg-foot segment. If the capsule is touching the leg-foot segment and pulling on the leg-foot segment, the leg-foot segment must also be touching and pulling on the capsule, introducing the force of leg/foot-on-capsule (LfC). CLf and LfC are an action-reaction pair, co-existing because two objects must touch *each other.* Because we have been limiting our interest to forces on the leg-foot segment, we could theoretically ignore LfC. However, John has injured his medial collateral ligament, which is closely associated with the knee joint capsule and has a similar line of pull. We will subsequently need to consider the forces pulling on the capsule (and its associated ligaments) to understand the effect of the exercises on John's injured knee structures.

Reaction forces are always in the same line and applied to the *different* but contacting objects. The directions of reaction forces are always opposite to each other because the two touching objects either pull or push on each other. Because the points of application of reaction forces are never on the same object, reaction forces are never part of the same force system and typically are not part of the same space diagram. However, we will see that reaction forces can be an important consideration in human function, because no segment ever exists in isolation (as is done in a space diagram).

Gravitational and Contact Forces

A different scenario can be used to demonstrate the sometimes subtle but potentially important distinction between a force applied to an object and its reaction force. We

generally assume when we get on a scale that the scale shows our weight. However, "weight" is the pull of gravity on a person's mass (gravity-on-person or GP) and is not applied to the scale. If GP is not applied to the scale, it cannot act on the scale. What is actually being recorded on the scale is the contact (push) of the "person-on-scale" (PS) and not "gravity-on-person." The distinction between these forces and the relation between these two forces can be established by using both Newton's first and third laws.

The person standing on the scale (**Fig. 1–19**) must be in equilibrium ($\Sigma F = 0$) because the person is not moving. If the gravity-on-person vector (GP) is acting down with a magnitude of –734 N (the person's weight), there must also be an upward force of equal magnitude acting on the person for the person to remain motionless. The only other object, besides gravity, that appears to be contacting the person in Figure 1–19 is the scale. The scale, therefore, must be exerting an upward push on the person (scale-on-person, or SP) with a magnitude equal to the pull of gravity-on-person (+734 N). Per Newton's third law (and common sense), if the scale is pushing on the person, the person must also be pushing on the scale. As is always true for reaction forces, the reaction force to scale-on-person will be equal in magnitude (734 N) and opposite in direction (down) but applied to the scale (person-on-scale, or PS in Fig. 1–19). In this scenario, the magnitude of the person's weight (GP) and the magnitude of the person's push on the scale (PS) are equivalent in magnitude *although applied to different objects.*

FIGURE 1-19

Although a scale is commonly thought to measure the weight of the person standing on the scale (gravity-on-person, or GP), it actually measures the contact of person-on-scale (PS). Because nothing else is touching the person in this figure but gravity and the scale, the push of scale-on-person (SP) must be equal in magnitude and opposite in direction to gravity-on-person (GP) for the sum of the forces on the static person to equal zero. Per Newton's second law, SP has a reaction force of person-on-scale (PS) that is equal in magnitude, opposite in direction, and applied to the scale. As long as nothing else is touching the person, then the magnitudes of GP, SP, and PS are all equivalent. That is, the "weight" of the person (GP) is equal to the person's contact with the scale (PS).



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The distinction is not always made between gravity-on-person and its reaction force of person-on-scale. However, it can be very important if something else is touching the person or the scale. If the person is holding something while on the scale, the person's weight does not change, but the contact forces between the person and the scale will

increase. Similarly, a gentle pressure down on the bathroom countertop as a person stands on the scale will result in an apparent weight reduction as the person makes contact with a new surface. Situations are frequently encountered in which the contact of an object with a supporting surface and its weight are used interchangeably. A recognition of the difference between gravity's pull on a body or segment (weight) and the force of contact a surface makes with that body or segment permits more flexibility in understanding how to modify these forces if necessary.

The reaction forces of person-on-scale (PS) and scale-on-person (SP) occur as a result of two contacting surfaces *pushing* on each other. When reaction forces arise from the *push* of contacting surfaces, the force pair are often referred to as **contact forces** (F_C). When contact forces are perpendicular to the surfaces that produce them, the term **normal force** (F_N) is also used.[5,6,7,8,9] Contact forces, therefore, are a subset of reaction forces.



Foundational Concepts 1–6

Action-Reaction Forces

- Whenever two objects or segments touch, the two objects or segments exert a force on each other. Consequently, every force has a reaction or is part of an action-reaction pair.
- The term *contact force* is commonly used to label one or both of the forces in an action-reaction pair where the "touch" of contacting objects is a push (rather than a pull).
- Reaction forces are *never* part of the same force system and cannot be composed (cannot either be additive or offset each other) because the two forces are, by definition, applied to different objects.
- The static or dynamic state of an object cannot be affected by another object that is not touching it or by a force that is not applied to it.
- The reaction to a force should be acknowledged but may be ignored graphically

and conceptually *if* the object to which it is applied and the other forces on that object are not of interest.

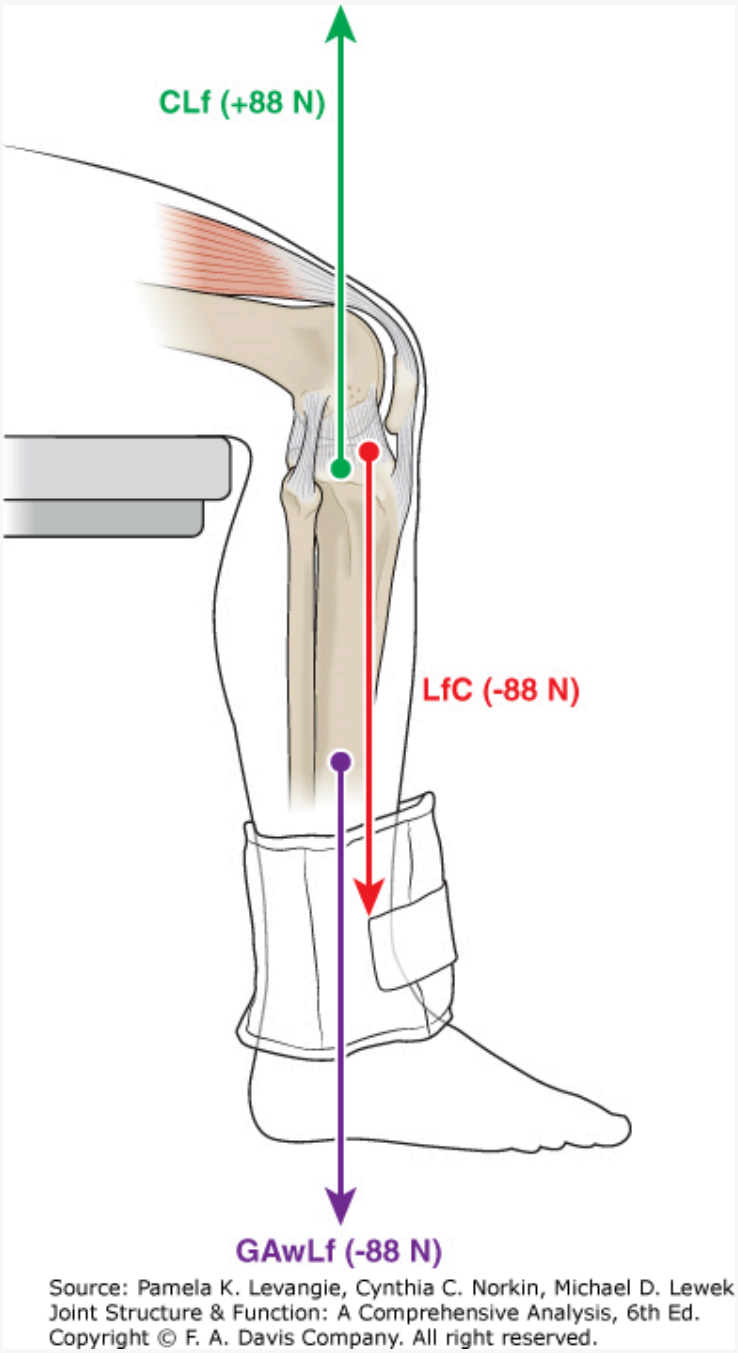
Additional Linear Force Considerations

To return to John Alexander's leg-foot segment as he sits with the dangling ankle weight, equilibrium in the leg-foot segment is dependent on the upward pull of the capsule (and its associated ligaments) on the leg-foot segment (CLf) with a force equal in magnitude to the downward pull of gravity and the ankle weight (GAwLf) (Fig. 1–20). We now know that if the capsule pulls on the leg-foot segment with a magnitude of 88 N, Newton's third law (law of reaction) stipulates that the leg-foot segment must also be pulling on the capsule (leg/foot-on-capsule, or LfC) with an equivalent 88 N force. *[Note: vectors CLf and LfC in Fig. 1–20 are effectively collinear and coplanar although applied to different objects. The vectors are separated in the figure so that each can be seen.]* John sustained an injury to his medial collateral ligament. The medial collateral ligament reinforces the knee joint capsule and has a similar line of pull to that of the capsule. Therefore, a pull on the capsule could be creating a pull on the injured medial collateral ligament. To understand how the pull of the leg-foot on the capsule affects the capsule (and its associated ligaments), we need to explore **tensile forces** and the forces that produce them.

FIGURE 1-20

The pull of the capsule on the leg-foot segment (CLf) is needed to offset the pull of gravity and ankle weight (GAwLf) for the leg-foot

segment to remain at rest. Newton's third law (law of reaction) says that if the capsule is pulling on the leg-foot segment (CLf), the leg-foot segment must also be pulling on the capsule (LfC) with a force equal in magnitude and opposite in direction. *[Note: Vectors CLf and LfC are effectively collinear and coplanar although applied to different objects. The vectors are separated in the figure so that each can be seen.]*



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Tensile Forces

Tension in a joint capsule, just like tension in any passive structure (including relatively solid materials such as bone), is created by opposite pulls on the same object. If there are not two opposite pulls on the object (each of which is a tensile force), there cannot

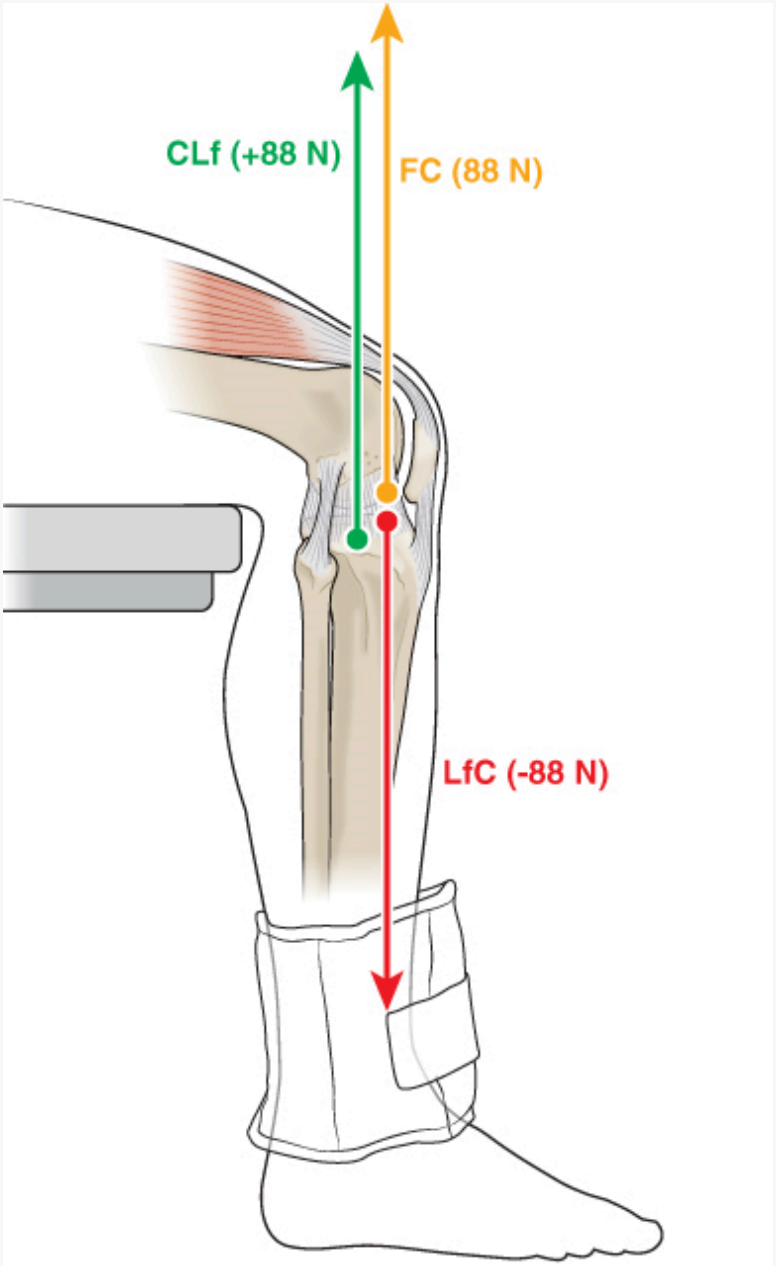
be tension in the object.

We established that the leg-foot segment is pulling on the capsule (LfC) with 88 N of force. Newton's first law (law of equilibrium) tells us that the capsule cannot be in equilibrium (at rest) if LfC is the only force acting on the capsule. Therefore, there must be at least one other force on the capsule and it must come from something "touching" the capsule. Because the capsule is attached both to the leg-foot segment distally and to the femur proximally, the femur is also touching the capsule (femur-on-capsule, or FC) (**Fig. 1–21**). If the capsule is in equilibrium, FC must also have a magnitude of 88 N (LfC + FC = 0). The forces of leg/foot-on-capsule and femur-on-capsule are opposite pulls that create tension in the capsule (each of which, then, is a tensile force). We can analogize the joint capsule to thin pieces of rope holding the two bones together. Like a rope in a tug-of-war, the capsule is getting pulled in opposite directions by forces of equal magnitude and will be under equal tension throughout. For a joint capsule and its associated ligaments to do the job of holding the bones of a joint together, the capsuloligamentous tissues must have sufficient tissue integrity to be able to withstand the tensile stresses imposed by the pulls of the contiguous bones.

FIGURE 1-21

For the knee joint capsule to be in equilibrium, there must be a second force on the capsule coming from something touching the capsule. Because the femur is the only other "object" touching the capsule, femur-on-capsule (FC) must be equal in magnitude (88 N) and opposite in direction to leg/foot-on-capsule (LfC). Because both

FC and LfC are pulling on the capsule in opposite directions, these forces create tension in the capsule and are considered to be tensile forces.



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Foundational Concepts 1–7

Tension and Tensile Forces

- Tensile forces (or the resultants of tensile forces) on an object are always equal in magnitude, opposite in direction, and applied parallel to the long axis of the object.
- Tensile forces are collinear, coplanar, and applied to the same object; therefore, tensile vectors are part of the same linear force system.
- Tensile forces applied at either end to a flexible or rigid structure of homogenous composition create the same tension at all points along the long axis of the structure (in the absence of additional contact and friction).

Joint Distraction

Joint capsule and ligaments are not always under tension. John's capsuloligamentous structures are under tension because of the downward pull of gravity and the ankle weight on John's leg-foot segment must be offset as John's unsupported leg-foot segment dangles (see Fig. 1–20). If John's foot was supported by someone's hand pushing upward on the bottom of John's foot (hand-on-leg/foot) with a force of 88 N, the tension in the capsule (and ligaments) would be eliminated because the upward pull of the capsuloligamentous structures is no longer necessary. In fact, the capsuloligamentous structures might actually become slack. If the upward support of the hand is gradually removed (and assuming the capsuloligamentous structures are initially slack), there would be at least a small net unbalanced force downward that would cause the leg-foot segment to accelerate away from the femur with the small acceleration proportional to the net unbalanced force (Newton's second law). The movement of one bony segment away from another is known as **joint distraction.**[3] As the distraction increases with the decreasing support of the hand, the tension in the capsule will concomitantly increase until, once again, the tension in the capsule

completely offsets the 88 N downward pull of gravity and the ankle weight on the leg-foot segment; equilibrium is restored once again to the leg-foot segment.

The pulls of gravity and the ankle weight (separately or together) on the leg-foot segment can be referred to as **distraction forces**[3] or **joint distraction forces.** A distraction force is directed away from the joint surface to which it is applied, is perpendicular to its joint surface, and leads to the separation of the joint surfaces. It is important to note that here the term *distraction* refers to separation of rigid non-deformable bones. Distraction across or within a deformable body is more complex and will be considered in Chapter 2.

 Expanded Concepts 1–4

Acceleration in Joint Distraction

Although not feasible in a clinical setting, the acceleration of the leg-foot segment downward can be calculated.

Gravity and the ankle weight together weigh 88 N. To calculate acceleration ($a = F_{unbal} \div m$), however, the mass (not just the weight) of the leg-foot segment and ankle weight must be known. Recalling that 1 N is the amount of force needed to accelerate 1 kg at 1 m/sec^2, weight (in newtons or equivalently in kg-m/sec^2) is mass (in kilograms) multiplied by the acceleration of gravity, or:

$$W = (m)(9.8\ m/sec^2)$$

Solving for mass, a weight of 88 N is equivalent to 88 kg-m/sec^2 ÷ 9.8 m/sec^2 = 8.97 kg. Consequently, the leg-foot segment and ankle weight together have a mass of approximately 9 kg. Assigning some arbitrary values, assume that a downward force of –88 N is offset in this static example by an upward push of the supporting hand of 50 N and a capsular tensile force of 10 N. The net unbalanced force on the leg-foot segment is –28 N (–88 + 50 + 10). Therefore:

$$a = \frac{-28 \text{ kg-m/sec}^2}{9 \text{ kg}}$$

$$a = -3.11 \text{ m/sec}^2$$

In the human body, the acceleration of one or both segments away from each other in joint distraction (the dynamic phase) is very brief unless the capsule and ligaments (or muscles crossing the joint) fail. John's leg-foot segment will not accelerate away from the femur for very long (milliseconds) before the distraction forces applied to the leg-foot are balanced by the tensile forces in the capsule. Presuming that John is relaxed as the ankle weight hangs on his leg-foot segment (we have not asked him to do anything yet), the check to joint distraction will fall entirely to tension in the capsuloligamentous structures. John presumably has a ligamentous injury that is likely to cause pain with tension in these pain-sensitive connective tissues.



Foundational Concepts 1–8

Joint Distraction and Distraction Forces

- Distraction forces create separation of joint surfaces.
- There must be a minimum of one (or one resultant) distraction force on each joint segment, with each distraction force perpendicular to the joint surfaces, opposite in direction to the distraction force on the adjacent segment, and directed away from its joint surface.
- Joint distraction can be dynamic (through unequal or opposite acceleration of segments) or static (when the tensile forces in the tissues that join the segments are balanced by distraction forces of equal or greater magnitude).

Joint Compression and Joint Reaction Forces

Supporting John Alexander's leg-foot segment with a hand underneath his foot can minimize or eliminate the tension in his injured capsuloligamentous structures. If the

supporting upward push of the hand on the leg-foot segment increases to 90 N while the magnitude of the downward pull of gravity and the ankle weight remain at –88 N, the sum of these forces will result in a net unbalanced *upward* force on the leg-foot segment of 2 N. If the 90 N support of the hand continues, the leg-foot segment will undergo a slight acceleration upward until a new downward force is encountered. The new force will emerge when the leg-foot segment makes contact with ("touches") the femur that lies above the tibial plateau. The upward acceleration of the leg-foot segment will stop when the new contact force, femur-on-leg/foot (FLf), reaches a magnitude of –2 N, at which point equilibrium of the leg-foot segment is restored.

When the two bony segments of a joint are pushed together and "touch" each other, the resulting reaction (contact) forces are also referred to as **joint reaction forces.**[3] Joint reaction forces are contact forces that result whenever there is contact between contiguous joint surfaces. Joint reaction forces are dependent on the existence of one force on each of the adjacent joint segments because the two bones must push on each other. Those forces will be perpendicular to and directed *toward* the joint surfaces. The two forces that cause joint reaction forces on each segment are known as **compression forces.** Compression forces push joint surfaces together to produce joint reaction forces just as distraction forces separate joint surfaces to produce capsuloligamentous tensile forces. In the example we have just been using, the push of the hand on the leg-foot segment is a compression force. Although there also must be a compression force on the femur to allow the femur to push on the leg-foot segment, our analysis doesn't require that we account for it.

 Foundational Concepts 1–9

Joint Compression and Joint Reaction Forces

- Joint compression forces create contact between joint surfaces.
- There must be a minimum of one (or one resultant) compression force on each contiguous joint segment, with each compression force perpendicular to and directed toward the segment's joint surface and opposite in direction to the compression force on the adjacent segment.

 Expanded Concepts 1–5

Close-Packing of a Joint

Capsuloligamentous structures are typically not under tension when there are *net* compressive forces (and no shear forces) across a joint. There is an important exception, however. With sufficient twisting of the capsuloligamentous structures of a joint caused by active or passive joint rotation, the adjacent articular surfaces can be drawn together into contact by the capsuloligamentous structures. This is called "close-packing" of the joint. Although considered an "Expanded Concept" in Chapter 1, this concept will be elaborated upon in Chapter 2 and in the examination of individual joint complexes in subsequent chapters. In those chapters, the concept of close-packing becomes a "Foundational Concept."

Revisiting Newton's Law of Equilibrium

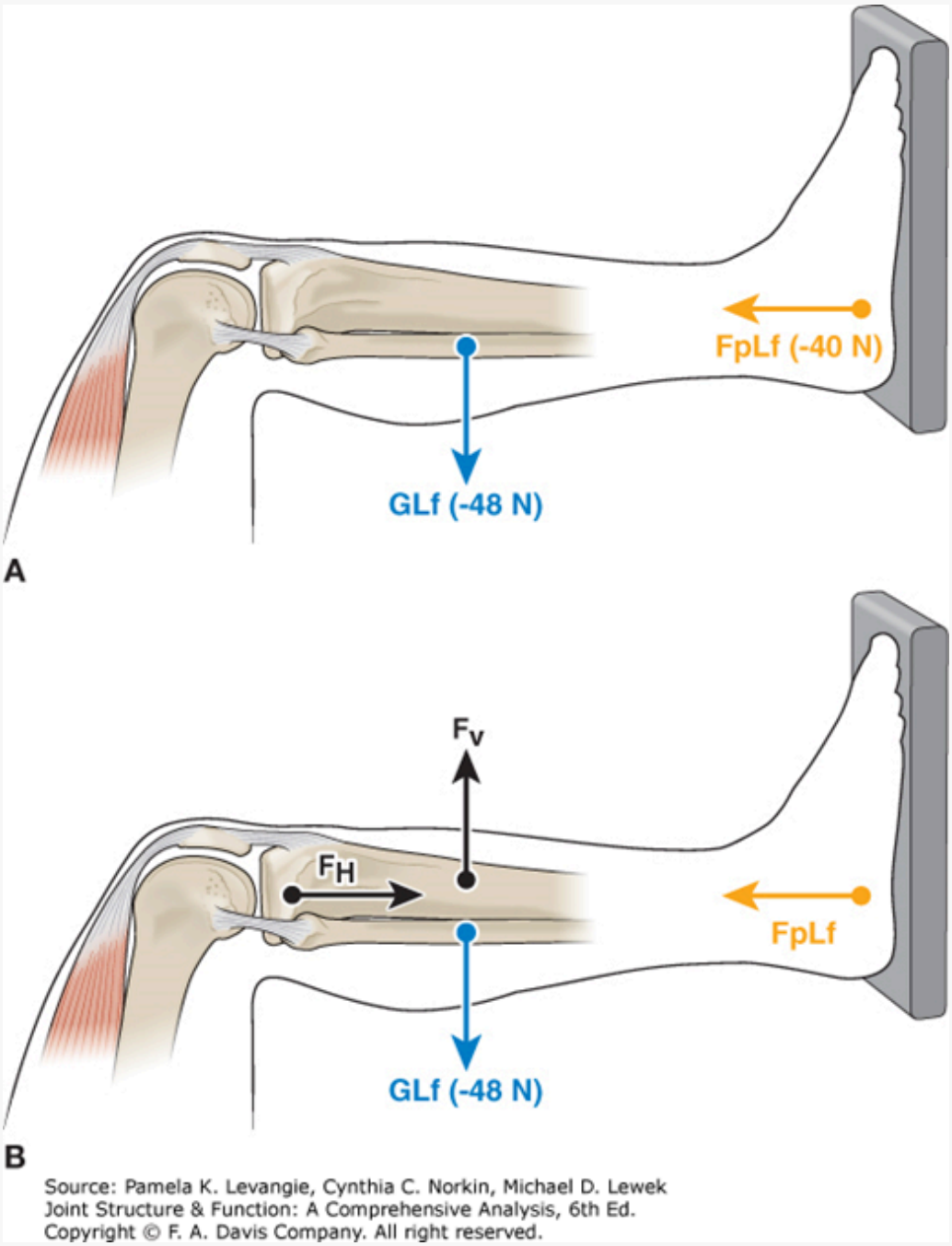
It would appear that the ankle weight is a poor option for John, given the potential tensile forces created in his injured capsuloligamentous structures, unless we plan to continue supporting his leg-foot segment with a hand (or, perhaps, a bench). Let's explore the option of the leg-press exercise to see how the change in exercise might change the forces at the knee joint.

In the leg-press exercise (**Fig. 1–22A**), the leg-foot segment is still acted upon by a –48 N gravitational force (GLf), but the change in orientation of the leg-foot segment means that the orientation of gravity has changed as well. The leg-foot segment is also contacted by the footplate (footplate-on-leg/foot, or FpLf) with a force of unknown

magnitude. Because GLf and FpLf are not part of the same linear force system in this scenario (neither collinear nor coplanar), no assumptions can be made about the magnitude of footplate-on-leg/foot based on what we know about gravity-on-leg/foot. Elaboration on Newton's law of equilibrium can assist us.

FIGURE 1-22

A. Before John begins the leg-press exercise, the leg-foot segment is contacted by gravity (GLf) and by the footplate (footplate-on-leg/foot, or FpLf). GLf and FpLf are not part of the same linear force system; the leg-foot segment cannot be in equilibrium with only these two forces. **B.** One additional vertical force (F_V) and one additional horizontal force (F_H) must exist on the leg-foot segment so that the sum of the vertical forces and the sum of the horizontal forces on the leg-foot are zero.



Vertical and Horizontal Linear Force Systems

As Newton's law of equilibrium was already defined, the sum of the forces on the object must be zero ($\Sigma F = 0$). We now can refine Newton's law of equilibrium to state that the sum of the **vertical forces (F_V)** acting on an object in equilibrium must total zero ($F_V = 0$)

and, independently, the sum of the **horizontal forces (F_H)** acting on that same object must total zero ($F_H = 0$). To meet this more specific set of conditions, there must be at least one additional vertical force (F_V) and one additional horizontal force (F_H) acting on the leg-foot segment that are equal in magnitude and opposite in direction to gravity-on-leg/foot and footplate-on-leg/foot, respectively (Fig. 1–22B). The sources of F_V and F_H are not yet established.

We know that the femur and capsule are both contacting and potentially creating forces on the leg-foot segment. Given these possibilities, F_H is likely to be the push of the femur on the leg-foot segment because the footplate can push the leg-foot segment into contact with the femur, creating a femur-on-leg/foot force. The source of the F_V is difficult to ascertain because the joint capsule (still touching the leg-foot segment) is unlikely to be a strong candidate; nothing else appears to be contacting the leg-foot segment. To identify F_V, we must acknowledge an additional property of all contact forces. Whenever there is contact between two objects, the potential exists for friction forces on both contacting surfaces. The friction forces will have magnitude, however, only if there are concomitant opposing shear forces on the contacting objects.

Shear and Friction Forces

A **shear force (F_S)** is any force (or component of a force) that has an action line parallel to contacting surfaces (or tangential to curved surfaces) that creates or attempts to create movement between surfaces. *[Note: The discussion here is about shear forces between two rigid (nondeformable) structures (e.g., bones). Shear within deformable structures will be considered in Chapter 2.]*

A **friction force (Fr)** potentially exists on an object whenever there is a contact force on

that object. Friction forces are always parallel to contacting surfaces (or tangential to curved surfaces) and have a direction that is opposite to potential movement. For friction to have magnitude, a net shear force must exist that creates or attempts to create movement between two contacting objects. Shear and friction forces share the common property that both are forces parallel to contacting surfaces, but shear forces are always applied in the direction of movement (or potential movement) whereas friction forces are always applied in the direction opposite to movement or potential movement. To understand the magnitude of a frictional force, we need to further explore the force of friction.

Static Friction and Kinetic Friction

The magnitude of a friction force on an object is always a function of the magnitude of contact between the objects and the slipperiness or roughness of the contacting surfaces. When two contacting objects with a net shear force are not moving on each other (objects are static), the magnitude of the **force of static friction (Fr_s)** on each object is less than or equal to the product of a constant value known as the **coefficient of static friction (μ_S)** and the magnitude of the contact force (F_C) on each object; that is,

$$Fr_S \leq \mu_S F_C$$

The coefficient of static friction is a constant value for given materials. For example, μ_S for ice on ice is approximately 0.05; the value of μ_S for wood on wood is as little as 0.25.[6] As the contacting surfaces become softer or rougher, μ_S increases. As the magnitude of contact (F_C) between objects increases, so too does the potential magnitude of friction. The greater the contact force on an object and the rougher the contacting surfaces, the greater the maximum potential force of friction. When you use friction to warm your

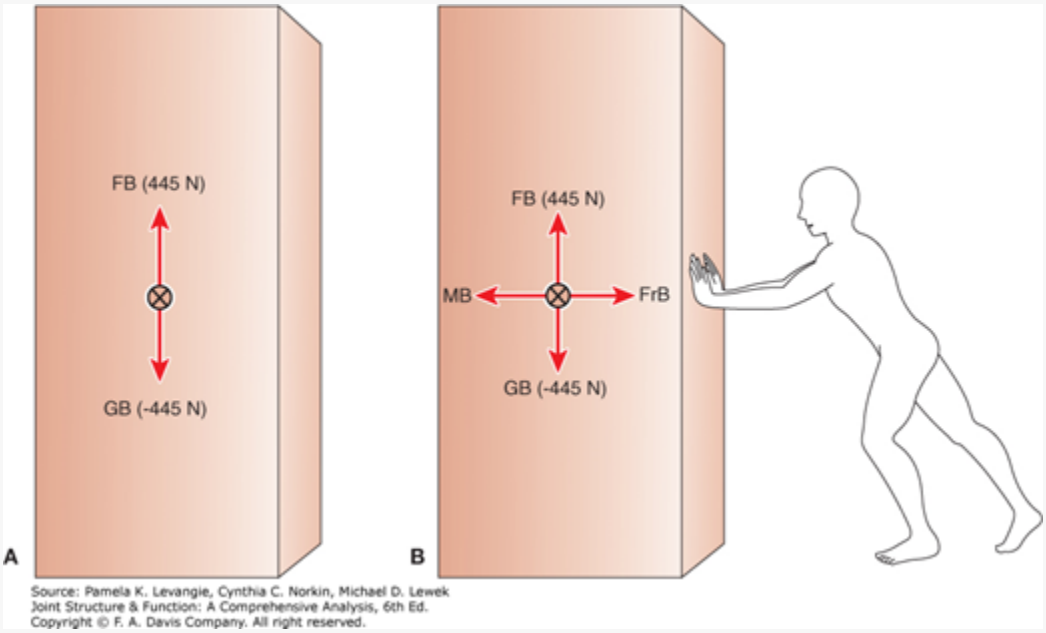
hands, the contact of the hands warms both of them (friction forces exist on both the right and left hands). If you wish to increase the friction, you press your hands together harder (increase the contact force) as you rub. Pressing your hands together harder increases the contact force between the hands and increases the maximum value of friction (the coefficient of friction remains unchanged because the surface remains skin on skin). It is commonly thought that the magnitude of friction between two surfaces is related to the amount of surface area in contact. However, the only contributing factors are the magnitude of contact and the coefficient of the contacting surfaces.[2]

In Figure 1–23A, a large box weighing 445 N (~45 kg or 100 lb) is resting on the floor. The floor must push on the box (FB) with a magnitude equal to the weight of the box (GB) because the box is not moving, therefore $\Sigma F_V = 0$. Because nothing is attempting to move the box parallel to the contacting floor, there will be no friction on either the box or the floor. However, as soon as the man begins to push on the box (Fig. 1–23B), a shear force (man-on-box, or MB) is created, with a concomitant resulting force of friction-on-box (FrB). *[Note: Although the example works, Figure 1–23B is oversimplified because it appears that all the forces are acting at the same point. Although floor-on-box does act through the center of mass of the box, it is applied at the bottom of the box; the force of friction-on-box would also act at the bottom of the box rather than as shown.]* Assuming the shear force of man-on-box is not sufficient to move the box, the maximum potential magnitude of friction-on-box can be calculated.

FIGURE 1-23

A. The box is acted on by the forces of gravity (GB) and the contact of

floor-on-box (FB). The force of friction has no magnitude (so is not shown) because there is no attempted movement. **B.** The force of the man-on-box (MB) causes an opposing friction force (FrB).



The maximum friction force on the box when the box is not moving is a product of the coefficient of static friction of wooden box on wood floor (0.25) and the magnitude (445 N) of the contact of floor-on-box:

$$FrB \leq (0.25)(445\ N)$$

$$FrB \leq 111.25\ N$$

The magnitude of the friction-on-box force will be equal to that of the shear force (man-on-box) as long as the box is not moving because $\Sigma F_H = 0$. As the man increases his push on the box without moving it, the magnitude of friction will simultaneously increase by the same amount, up to a maximum of 111.25 N as calculated in this example. The magnitude of static friction will change in response to the magnitude of shear force on

the object, but static friction cannot exceed its calculated maximum force.

If the push of the man on the box exceeds 111.25 N, the box will begin to move because friction cannot have a magnitude greater than 111.25 N; there will be a net unbalanced force in the direction of the man's push. Once an object is *moving,* the magnitude of the **force of kinetic friction (Fr_K)** must be calculated. Unlike static friction, kinetic friction in any given situation is constant in magnitude and equal to the product of the contact force (F_C) and the **coefficient of kinetic friction (μ_K):**

$$Fr_K = (\mu_K)(F_C)$$

The coefficient of kinetic friction (μ_K) is always smaller in magnitude than the coefficient of static friction (μ_S) for any set of contacting surfaces. Consequently, the magnitude of the force of friction is always greatest immediately before the object is about to move (when the shear force has the same magnitude as the maximum value of static friction). Once the shear force exceeds the maximum value of static friction and the object begins to move, the value of friction drops from its maximum static friction value to its smaller kinetic friction value, resulting in a sudden increase in the net unbalanced force on the object even if the push of the man is the same. The sudden drop in magnitude of friction results in the classic situation in which the man pushes harder and harder to get the box moving along the floor and then suddenly finds himself and the box accelerating too rapidly.



Foundational Concepts 1–10

Friction and Shear Forces

- Shear and friction forces potentially exist whenever two objects touch.

- A shear force is any force (or force component) that lies parallel to the contacting surface (or tangential to a curved surface) of an object, attempts movement between the contacting objects, and is applied in the direction of movement or potential movement.
- A friction force lies parallel to the contacting surface (or tangential to the curved surface) of any object but always opposite to the direction to potential or relative movement between contacting surfaces.
- Friction has magnitude only when there is a net (resultant) shear force applied to an object.
- The magnitude of static friction can change with an alteration in the net shear force applied to the object; the magnitude of kinetic friction remains the same regardless of the magnitude of the shear force or forces on the object or the speed of the moving object.
- The magnitude of friction can never exceed the magnitude of the net shear force it opposes.
- Shear and friction forces are always parallel to contacting surfaces, whereas the contact force (or contact force component) that must exist concomitantly is perpendicular (normal) to the contacting surfaces. Consequently, shear and friction forces are perpendicular to a contact force (or, more correctly, the component of a contact force that is "normal" to the contacting surfaces).[3]

Considering Vertical and Horizontal Linear Equilibrium

We can now return to John and the leg-press example. An understanding of shear and friction forces can be used to identify the source of F_V in Figure 1–22B and to calculate the magnitudes of FpLf, F_V, and F_H.

Because the force of gravity (GLf) lies parallel to the footplate and has the potential to slide the leg-foot segment down on the footplate (see Fig. 1–22B), GLf is a shear force (lies parallel to contacting surfaces). If there is a shear force between the vertically aligned footplate and leg-foot segment, there will also be a friction force that is vertical and in a direction opposite to the shear force (GLf). Therefore, the unknown F_V in Figure 1–22B is likely to be friction-on-leg/foot from the contact with the footplate.

The leg-foot segment is in equilibrium and there are only two vertical forces. Therefore, the magnitude of the friction-on-leg/foot force (F_V) must be equal to the magnitude (although opposite in direction) to the –48 N shear force (GLf). If the coefficient of static friction between the sole of John's shoe and the metal footplate is estimated at $\mu_S = 0.6$ (slightly more than wood-on-wood)[6], we can solve for the contact force of footplate-on-leg/foot (FpLf) given that friction-on-leg/foot (F_V) is 48 N:

$$48\ \text{N} = (0.6)(F_C)$$

$$F_C \text{ or } \text{FpLf} = 80\ \text{N}$$

If the calculated magnitude of the contact force footplate-on-leg/foot vector (FpLf) in Figure 1–22B is –80 N (directed to the left), then the magnitude of the femur-on-leg/foot force must be +80 N (directed to the right), because the sum of the horizontal forces must be zero for the leg-foot to be in equilibrium.

PART 2: Kinetics—Considering Rotary and Translatory Forces and Motions

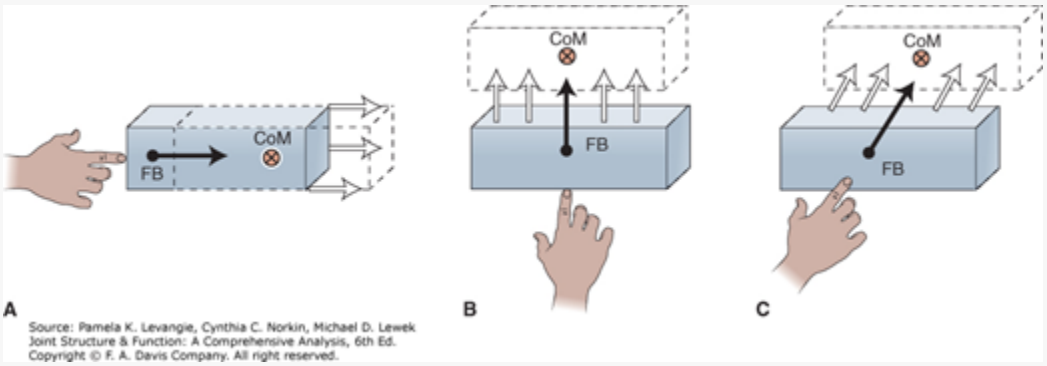
As we begin exploring rotary and general motion, we will continue to confine discussion and examples to two-dimensional analyses (as we have with examples thus far) to facilitate an understanding of the concepts. Subsequent chapters will superimpose the third dimension conceptually, although rarely mathematically.

When an object is completely unconstrained (not attached to anything), a single force applied at or through the center of mass of the object will produce linear displacement regardless of the angle at which the force is applied (Fig. 1–24A–C). However, forces applied to bony segments in the body are rarely applied through the center of mass of

the object. When the force applied to an unconstrained object does not pass through its center of mass, a combination of rotation and translation will result (Fig. 1–25). Consequently, isolated linear movement of a bony segment is atypical (if it occurs at all), with rotary and general motions much more common.

FIGURE 1-24

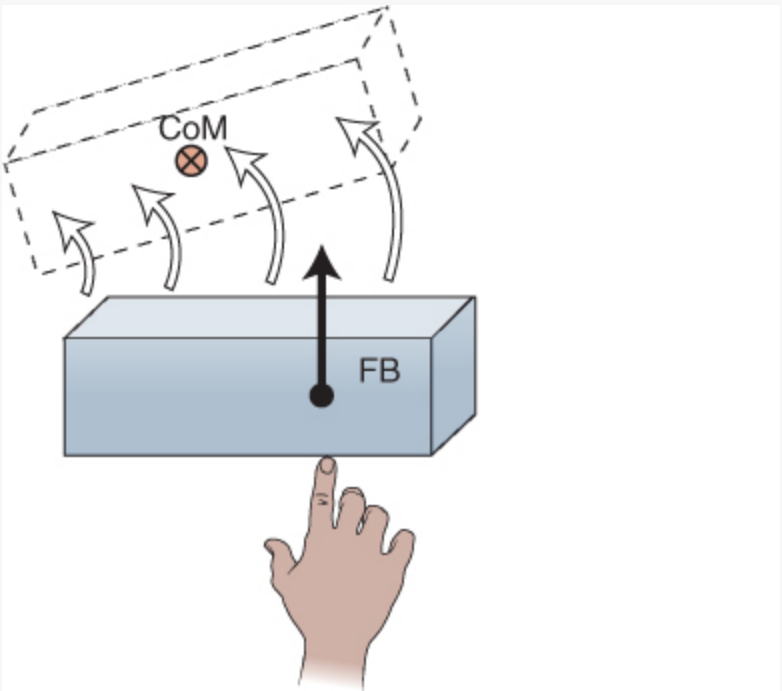
An isolated force (finger-on-block, or FB) applied to the block that passes through its center of mass (CoM) will produce linear displacement (translatory motion) of the block in the direction of the unbalanced force regardless of the direction from which the force is applied.



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FIGURE 1-25

When an isolated force (finger-on-block, or FB) that does not pass through the center of mass (CoM) is applied to the block, a combination of rotary and translatory motion of the block will occur (general motion).



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Torque, or Moment of Force

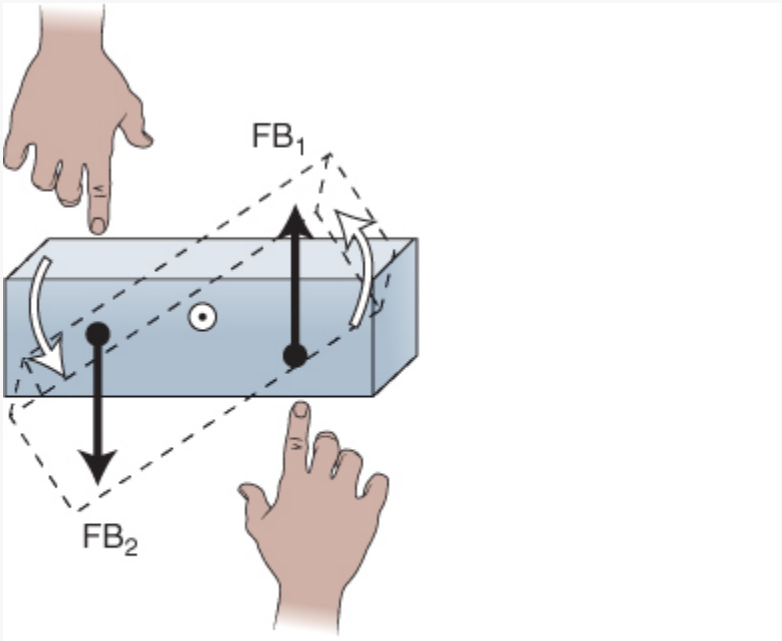
To produce isolated rotary motion (angular displacement) of an object or segment, a *second force* that is parallel to the original force must be applied to that object or segment. When a second force equal in magnitude, opposite in direction, and parallel to the original force (Fig. 1–26) is applied, the translatory effects of these forces will offset each other and pure rotary motion will occur. If the object is unconstrained, as is the block in Figure 1–26, the rotation of the segment will occur around a point midway between the two forces. If the object is constrained by one of the forces (if the point of application of FB_2 is fixed), the rotation will occur around the point of application of the constraining force (Fig. 1–27). Two forces equal in magnitude, opposite in direction, parallel, and applied to the same object at different points are known as a **force couple.** A force couple acting at some distance from each other on the same object will always

produce a **moment of force,** or torque **(τ).** Torque (used interchangeably with moment of force or "moment") is the product of the magnitude of one of the forces in a force couple and the shortest perpendicular distance between the forces, referred to as the **moment arm (MA):**

$$\tau = (F)(MA)$$

FIGURE 1-26

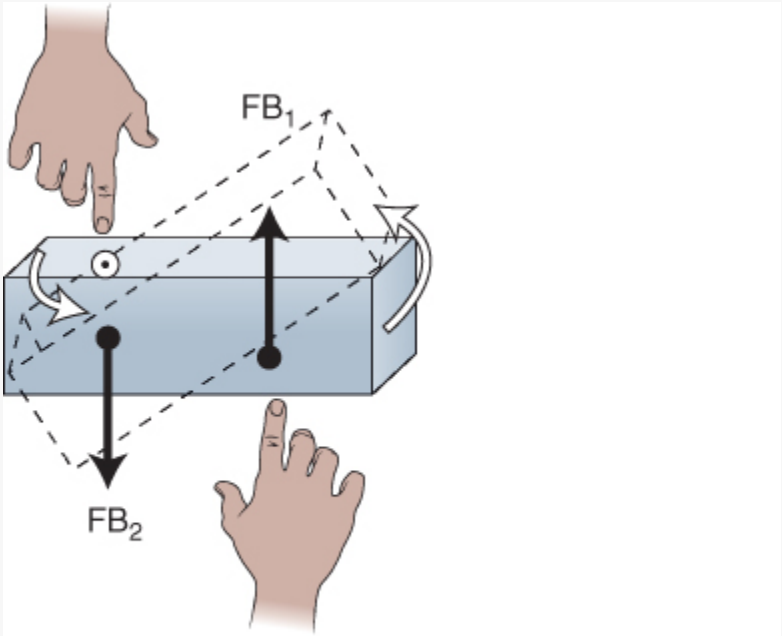
Two forces of finger-on-box (FB_1 and FB_2) that are equal in magnitude are applied to the block in opposite directions. FB_1 and FB_2 constitute a force couple and will create isolated rotation about a point midway between the forces, presuming no constraints on the object.



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FIGURE 1-27

Two forces of finger-on-box (FB_1 and FB_2) that are equal in magnitude are applied to the block in opposite directions. However, the point of application of FB_2 is fixed. FB_1 and FB_2 constitute a force couple with isolated rotation occurring around the fixed point of application of FB_2.



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Because torque is directly proportional to both the magnitude of the applied force (the magnitudes of the forces in a force couple are equivalent) and the distance between the force couple, torque can be thought of as the strength of rotation. Therefore, the greater the magnitude of the force couple, the greater the strength of rotation; the farther apart the forces of a force couple (the greater the MA), the greater the strength of rotation.

Force is measured in newtons and distance in meters; therefore, the unit for torque is

the **newton-meter (Nm)** (or the foot-pound in the U.S. system). The direction of potential rotation or torque at a joint segment can also be labeled by using the terms flexion/extension, medial/lateral rotation, or abduction/adduction. Consequently, a torque in the direction of joint flexion may be referred to either as a **flexion moment** or a flexion torque.

Parallel Force Systems

Because the forces in a force couple are parallel to each other, the two forces are part of a **parallel force system,** that exists whenever two or more forces applied to the same object are parallel to each other. The torque generated by each of the parallel forces is determined by multiplying the magnitude of the force by its moment arm, with the moment arm being the shortest distance from the force to either the point of constraint of the segment or to an arbitrarily chosen point on the segment (as long as the same point is used for each of the parallel forces).

Determining Resultant Forces in a Parallel Force System

The net or resultant torque produced by forces in the same parallel force system can be determined by adding the torques contributed by each force, considering their positive or negative signs. By convention, clockwise torques are considered negative whereas counterclockwise torques are considered positive.

Three forces are applied to an unconstrained segment in Figure 1–28. The magnitudes of forces F_1, F_2, and F_3 are 5 N, 3 N, and 7 N, respectively. The shortest distance (moment arms) between F_1, F_2, and F_3 and an arbitrarily chosen point on the object are 0.25 m, 0.12 m, and 0.12 m, respectively. F_1 and F_2 are applied in a clockwise direction, whereas F_3 is applied in a counterclockwise direction (in relation to the chosen point of rotation).

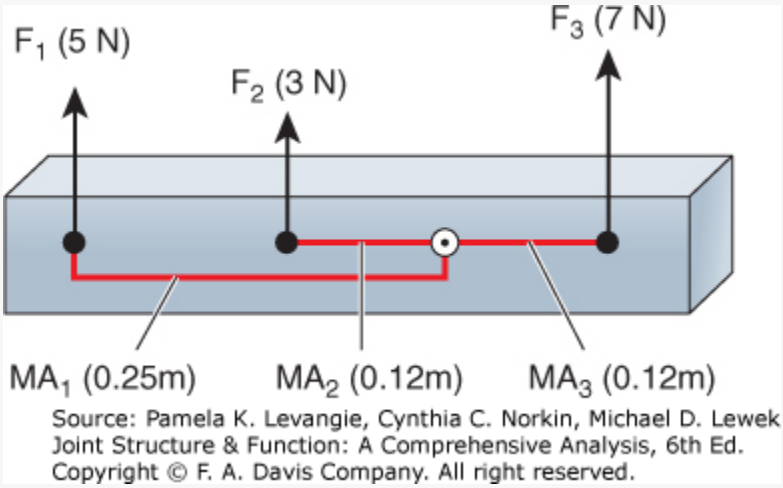
The net (resultant) torque would be:

$$\tau_{net} = (0.25\ m)(-5\ N) + (0.12\ m)(-3\ N) + (0.12\ m)(+7\ N)$$

$$\tau_{net} = -0.77\ Nm$$

FIGURE 1-28

The resultant of three parallel forces is found by the sum of the torques, with the torques being the product of each force (F_1, F_2, and F_3) and its motion arm (MA; distance from the specified point of rotation).



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That is, there will be a net rotation of the segment in a clockwise direction with a magnitude of 0.77 Nm.

Because the net torque in Figure 1–28 is calculated around a point that is *not* at the center of mass of the segment, the segment will not only rotate but will also translate. This is evident when we consider that all three forces (F_1–F_3) are upward vertical forces; these forces not only produce rotation (torque) but also produce translation. The sum

of the vertical forces yields a net upward translatory force of 15 N, with that upward translation occurring concomitantly with the net torque of -0.77 Nm.



Foundational Concepts 1–11

Revisiting Newton's Law of Equilibrium

Based on Newton's law of equilibrium, for an object or segment to be in equilibrium, $\Sigma F = 0$. We then elaborated to specify that for an object or segment to be in equilibrium, $\Sigma F_V = 0$ and $\Sigma F_H = 0$. We can now further specify that for an object or segment to be in equilibrium, $\Sigma \tau = 0$. All three of the following conditions must be met for an object or segment to be in equilibrium:

$$\Sigma F_V = 0$$

$$\Sigma F_H = 0$$

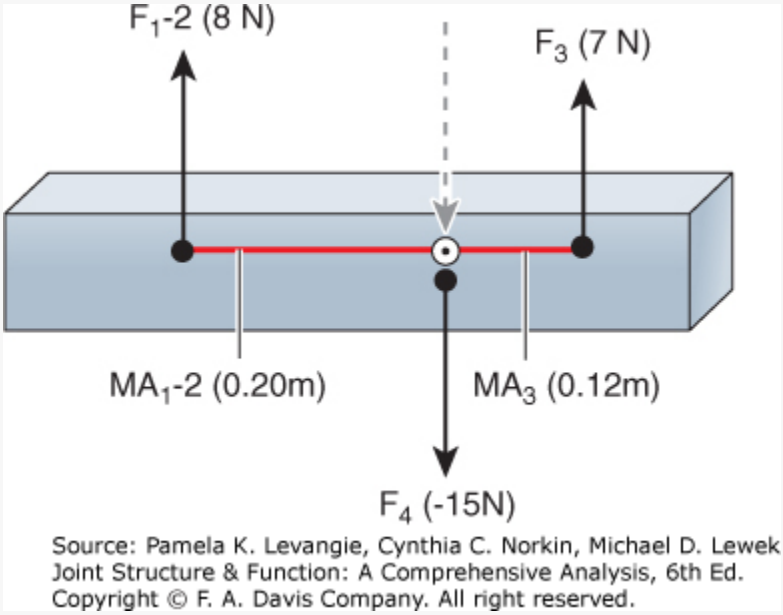
$$\Sigma \tau = 0$$

If the goal in Figure 1–28 is to rotate rather than translate the segment (as is the goal of joints in the human body), the unwanted upward translation of the segment must be eliminated by the addition of a new force. However, let us first simplify by composing F_1 and F_2 into a single resultant force, given that both are producing clockwise rotation of the segment. In **Figure 1–29**, forces F_1 and F_2 have been composed into resultant force F1-2, and a new vector (F4) has been added. *[Note: Force F4 might be easier in this particular figure to visualize as a push downward, as shown by the dotted gray arrow. However, we will continue to use the convention that the base of the arrow is on the point of application, as shown for the vector labeled F_4.]*

FIGURE 1-29

Forces F_1 and F_2, originally shown in Figure 1–28, have been

composed into force F_{1-2}. The addition of F_4, applied through the point of rotation, will produce vertical equilibrium without producing any additional torque because F_4 passes through the axis (has no moment arm). The block will now rotate around a fixed axis in the direction of unbalanced torque.



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Force F_4 (Fig. 1–29) has been assigned a magnitude of –15 N. With the addition of F_4, the sum of vertical forces (F_{1-2}, F_3, and F_4) is now zero. Force F_4 passes through the hypothetical point of rotation for the segment. Because it passes through that point, it is applied at 0 m from the point; the torque produced by F_4, therefore, is zero (–15 N x 0). Any force applied *through* a reference point or point of rotation will not produce torque. Although the addition of vector F_4 results in vertical equilibrium, the net torque is still –0.77 Nm. The effect produced by the addition of F_4 is what happens in the human body. A body segment will translate (with or without a concomitant rotation) until an equal and opposite constraint is encountered, at which point the translatory

forces on the segment are balanced and pure rotation is produced.

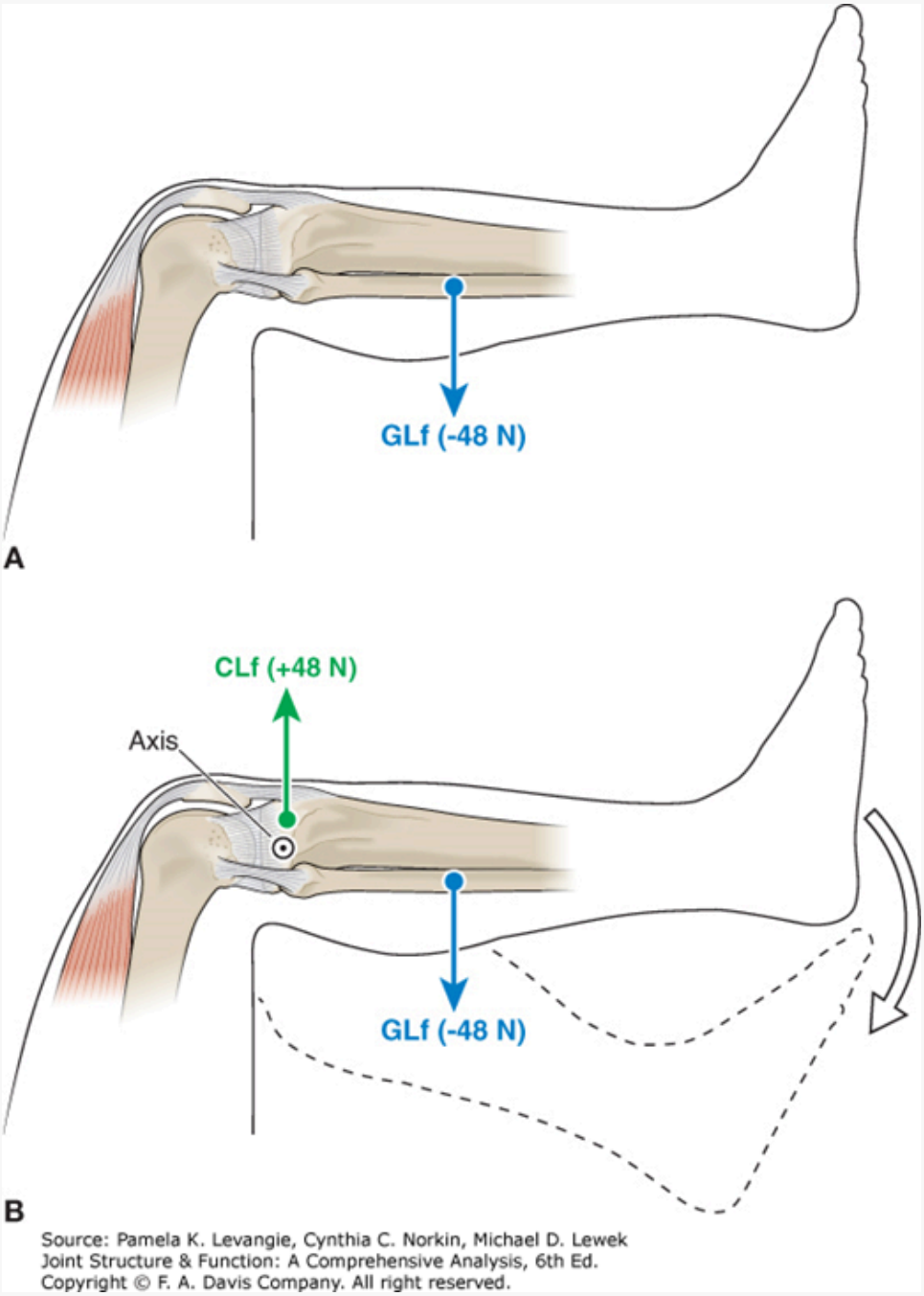
Identifying the Joint Axis About Which Body Segments Rotate

Figure 1–30A shows John's leg-foot segment in the same position as in the leg-press exercise but without the contact of the footplate. If John is still relaxed, his leg-foot segment cannot remain in equilibrium. Initially, the leg-foot segment would translate downward linearly because of the downward force of gravity (GLf). Gravity in this instance is a shear force because it lies parallel to the contacting surfaces of the femoral condyles and the tibial plateau. Although a shear force has the potential to generate an opposing friction force, the very small coefficient of friction that exists in a synovial joint (with estimates as low as 0.005) means that the magnitude of friction would be negligible.[3] The leg-foot segment, therefore, would translate downward until the knee joint capsule became tensed as we saw previously, creating the force of capsule-on-leg/foot (CLf) (Fig. 1–30B). As the leg-foot segment translates downwardly, the magnitude of tension in the capsule would increase until CLf reached 48 N. We can now see that capsule-on-leg/foot (CLf) forms a force couple with the gravity-on-leg/foot (GLf). Motion of the leg-foot segment then continues as an isolated rotation around the point of application (axis) of the constraining capsule-on-ligament force.

FIGURE 1-30

A. Gravity-on-leg/foot alone would cause downward translation of the leg-foot segment. **B.** As the leg-foot segment translates downward, the capsuloligamentous structures of the knee joint (CLf) become increasingly tight. When CLf reaches 48 N and constrains further

downward movement, CLf and GLf form a force couple that produces clockwise rotation of the knee joint around the point of application of the constraining force, CLf.



In the human body, the motion of a segment at a joint is ultimately constrained by the

articular structures, either by joint reaction forces (bony contact) or by capsuloligamentous forces. The point of application of the joint reaction or capsuloligamentous force that constrains further translation becomes the fixed pivot point for rotation (one half of a force couple). The net effect of translation (to the point of constraint) followed by rotation (around the point of constraint) is a subtle curvilinear motion of the segment. Because the motion is curvilinear, the axis shifts slightly during the motion. If the normal articular constraints at a joint are inadequate (as for damaged tissues), there will be excessive translatory motion and an excessive shift in the instantaneous center of rotation can occur.

Although it is acknowledged that the instantaneous center of rotation of a joint shifts with motion of a body segment, it is common practice to identify the joint axis as a fixed point about which the joint rotation *appears* to occur. This is most often assumed to be approximately in the center of the sequential instantaneous centers of rotation. It must be acknowledged, however, that this practice somewhat oversimplifies the assessment of torques on a body segment.

Bending Moments and Torsional Moments

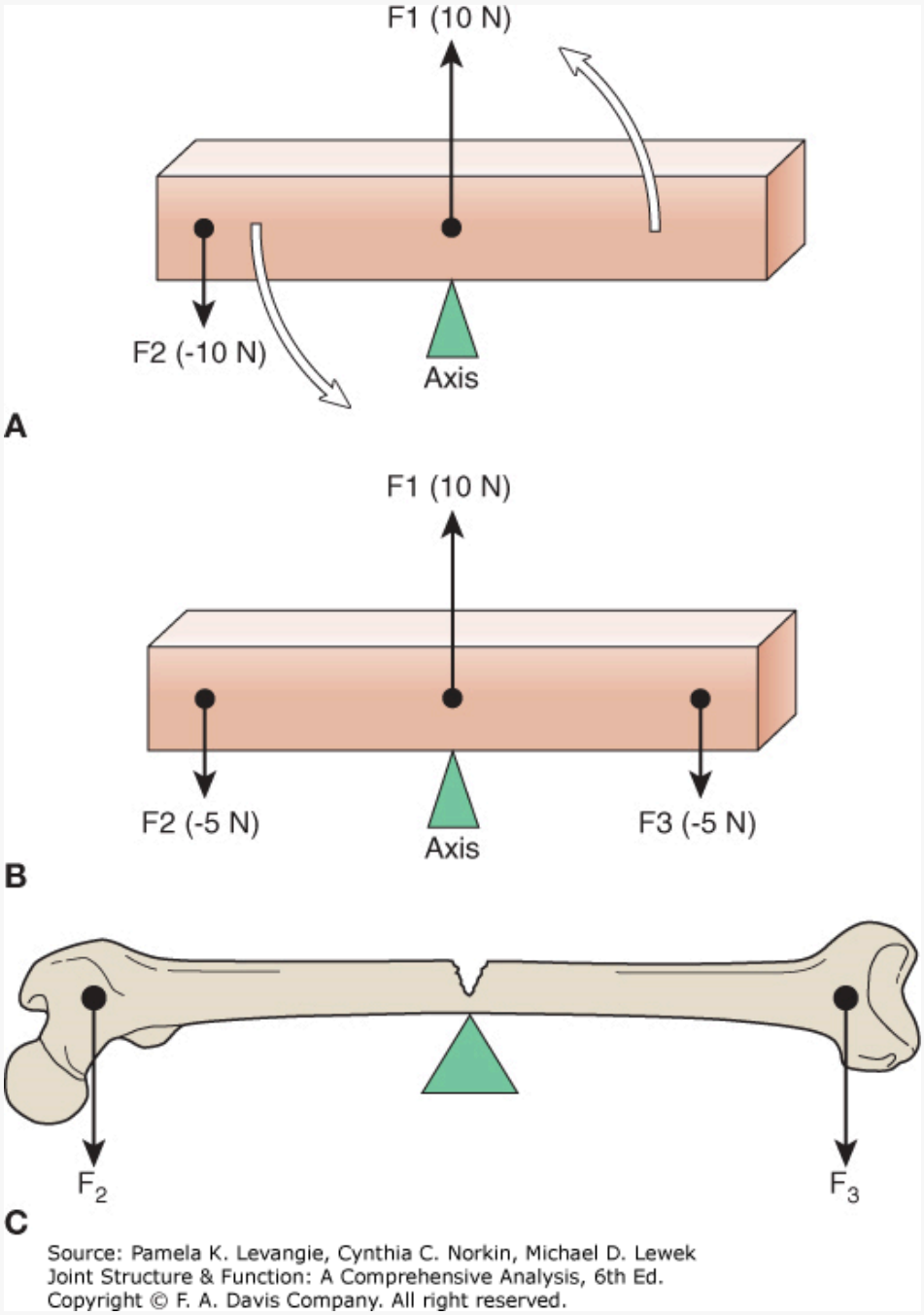
When parallel forces are applied to a rigid object in a way that the object is in equilibrium (neither rotation nor translation of the segment), the torques (moments of force) applied to a particular point on the object may also be considered to be **bending moments.**[3,4]

If parallel forces F1 and F2 with a magnitude of 10 N each are applied to a block (Fig. 1–31A), the block would rotate around the fixed point of rotation (point of application of F1) because F1 and F2 are a force couple. Introducing a new force (F3) can restore

equilibrium to the block, with F1 and F3 each having half the magnitude of F2 ($\Sigma F_V = 0$) (Fig. 1–31B). Although the segment is neither rotating nor translating, F1 and F3 would "bend" the segment around the apex of the triangle if the block was not rigid; hence, force couples in an equilibrium situation can also be considered to be bending moments. Because it took all three parallel forces to create the bending effect, bending moments may also be referred to as **three-point bending.** Occasionally, a bony segment is constrained in a way that results in substantial bending forces that cause damage, that is, fracture. Bone strength is often tested in experimental conditions using three-point bending (Fig. 1–31C).

FIGURE 1-31

A. A force couple (F_1 and F_2) will produce rotation around the fixed force (F_1). **B.** A bending moment is created when a third force (F_3) is added to a force couple (F_1 and F_2), resulting in rotary and translatory equilibrium but tending to "bend" the object around the point of application of the fixed force (F_1). **C.** The principle of bending moments (or three-point bending) is often used in biomechanical testing of long bones (e.g., the femur).



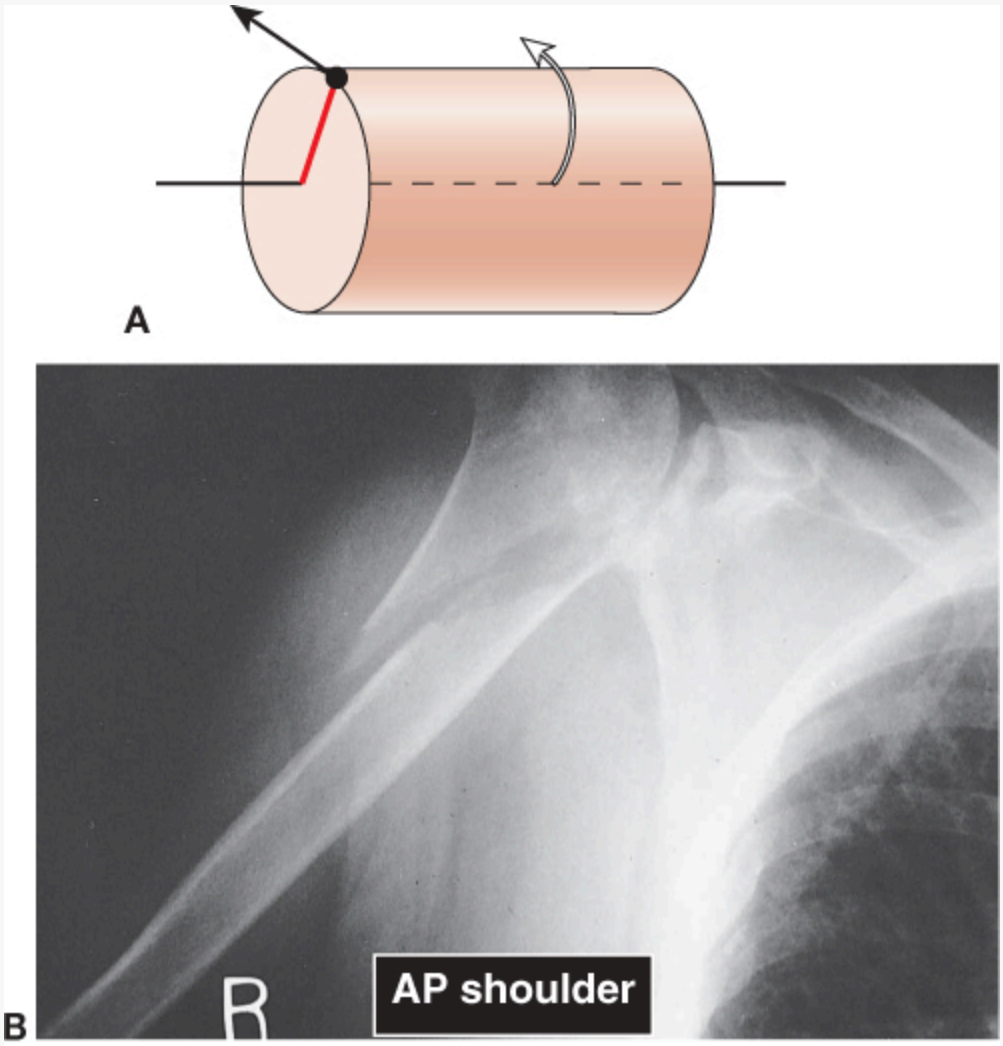
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A **torsional moment** is sometimes considered a special case (or subcategory) of a torque or moment of force. A torsional moment is produced when a so-called torsional force applied tangential to the periphery of a segment and at a distance from the segment's axis of rotation creates (or tends to create) rotation *within a segment* around its long

axis (Fig. 1–32A). A common example of the result of such a torsional moment is a spiral fracture in a long bone of the body (Fig. 1–32B).

FIGURE 1-32

A. A tangential force applied to the periphery of a long segment (at some distance from the axis that passes longitudinally) produces a "torsional moment" that is directly proportional to the magnitude of the force and its distance from the longitudinal axis. **B.** A spiral fracture of a humerus results from a torsional moment on the bone.



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Muscle Forces

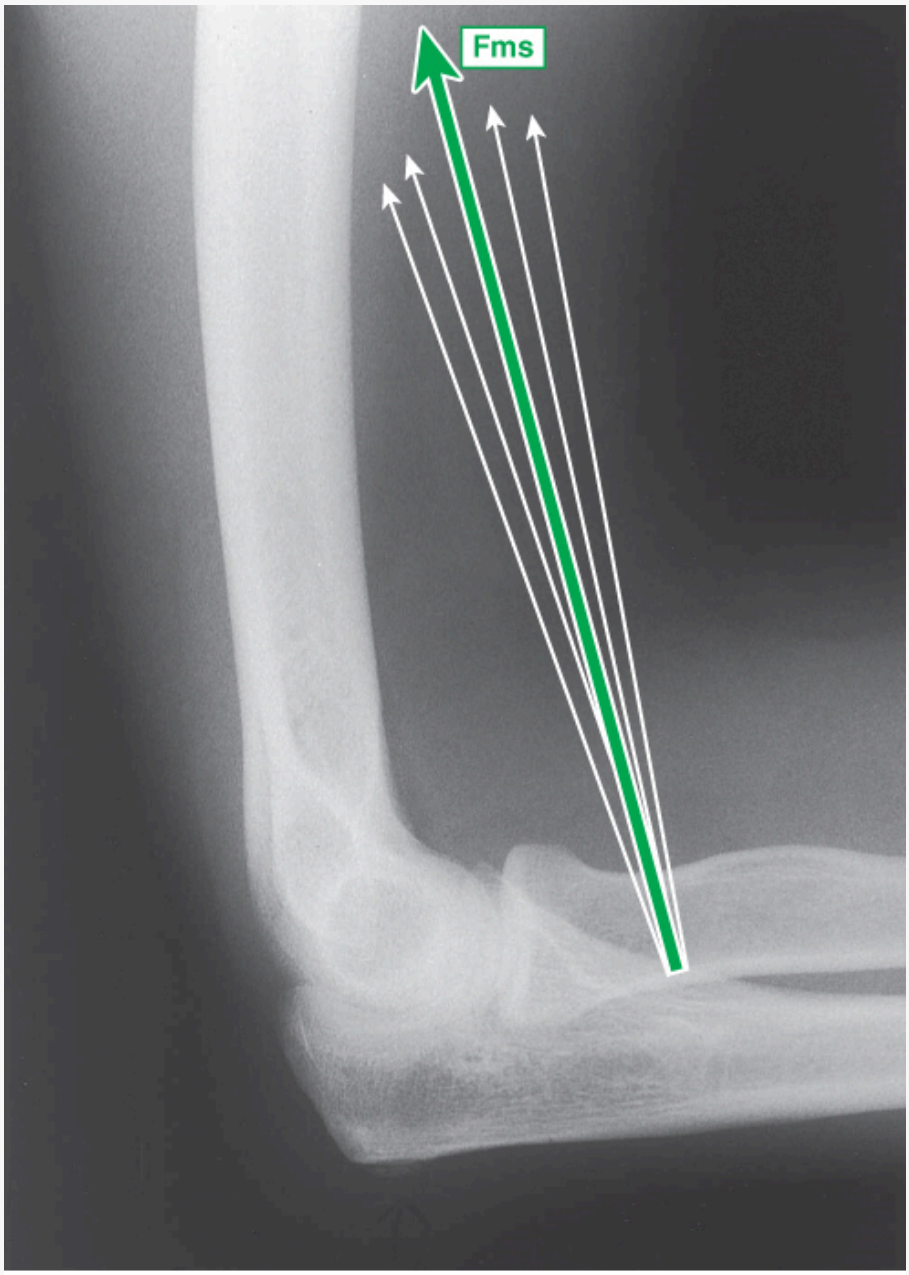
Total Muscle Force Vector

The force applied by a muscle to a bony segment is actually the resultant of many individual forces (muscle fibers) pulling on a common point of application. Each fiber in the muscle can be represented by a force vector that converges with the other fiber vectors at the muscle's attachment, forming a concurrent force system with a resultant represented by the total muscle force vector (Fms) (Fig. 1–33). The vector of a muscle

force can be approximated by drawing an action line symmetrically through the middle of the muscle's fibers in the direction of the middle of the muscle. The direction of pull for any muscle is *always* toward the center of the muscle. Every muscle exerts a force (a pull) on each of its attachments simultaneously. Therefore, every muscle creates a minimum of two force vectors, one on each of the two (or more) segments to which the muscle is attached; each of the two (or more) vectors is directed toward the middle of the muscle. The type and direction of motion that results from an active muscle contraction depends on the net forces and net torques acting on each of its bony attachments. The muscle will rotate a segment in its direction of pull only when the torque generated by the muscle exceeds opposing torques.

FIGURE 1-33

A representative sample of brachialis muscle fibers converge on the ulna, with the total muscle force (Fms) being the resultant of all the individual muscle fiber pulls.



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Expanded Concepts 1–6

Measuring Muscle Force

The magnitude assigned to a muscle force in a living human is typically estimated because the actual magnitude (the pull of a muscle on its attachment) cannot be

determined in a person without invasive measurement tools. Electromyography (EMG) can measure the electrical activity of a muscle. The electrical activity is directly proportional to the motor unit activity, which is, in turn, directly proportional to isometric muscle force (see Chapter 3). However, neither electrical activity nor the number of motor units is a measure of absolute force because muscle is exquisitely sensitive to its mechanical environment (e.g., length, velocity). The clinical "strength" of a muscle is measured by using devices such as weights, force transducers, or isokinetic devices. However, what is actually being measured directly or indirectly is the torque being applied to the joint. Although the net internal torque on the segment can be estimated if the moment arms of the known external forces (gravity, weights, and so on), the magnitudes of individual internal forces contributing to the net internal torque are difficult, if not impossible, to quantify. Even if a single muscle is active (which is almost never the case), it would be difficult to separate out the influence of forces such as joint reaction forces, capsuloligamentous forces, and small friction forces. The result is that the actual magnitude of pull of a single muscle cannot be assessed in a living person (in vivo) without surgically implanting a device directly onto the tendon, which may itself alter the force normally produced by a muscle. It should be noted that individual muscle forces are often approximated using mathematical models of varying levels of sophistication. The outputs of these models are the best estimates of muscle force.

In a space diagram, we will often identify only the pull of the muscle on the segment under consideration. However, it should be recognized that we are purposefully ignoring that same muscle's concomitant pull on one or more *other* segments.

Anatomic Pulleys

Frequently, the fibers of a muscle or a muscle's tendon wrap around a bone or are deflected by a bony prominence. When the direction of pull of a muscle is altered, the bone or bony prominence causing the deflection forms an **anatomic pulley.** Pulleys (if they are frictionless) change the direction of a force without changing the magnitude. As we will see, the change in action line produced by an anatomic pulley will have implications for the ability of the muscle to produce torque.

Anatomic Pulleys, Action Lines, and Moment Arms

The function of any pulley is to redirect a force to make a task easier. The "task" in human movement is to rotate a body segment. Anatomic pulleys (in the majority of instances) make this task easier by deflecting the action line of the muscle farther from the joint axis, thus increasing the moment arm of the muscle force. By increasing the moment arm (MA) of a muscle force, a force (F) of the same magnitude will produce greater torque (remembering that $\tau = F \times MA$).

Sesamoid bones lie where tendons pass over joints and function to change the direction of a muscle or tendon action line and, as a result, increase the ability of a muscle to produce torque around one or more joint axes. There are several sesamoid bones in the human body, the largest being the patella.



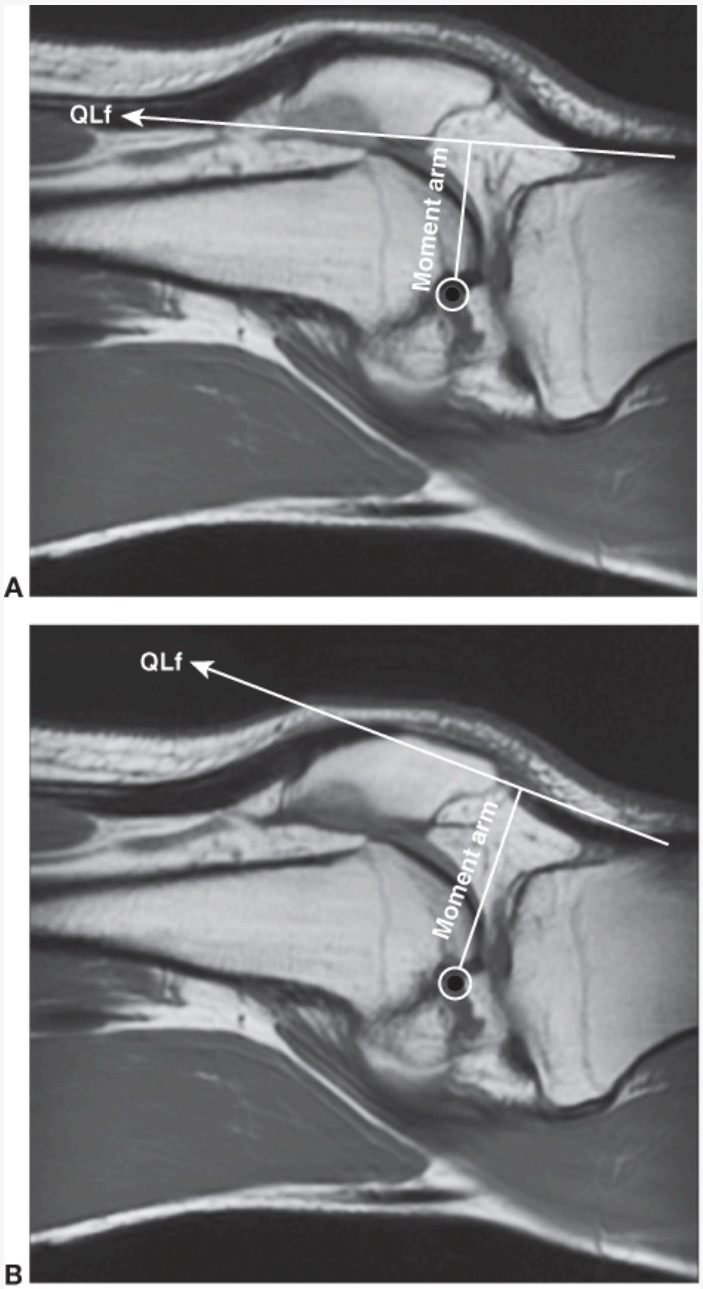
Example 1–2

The Patella as an Anatomic Pulley

The classic example of an anatomic pulley is that formed by the patella. The quadriceps muscle belly lies parallel to the femur. The tendon of the muscle passes over the knee joint and attaches to the tibia via the patellar tendon at the tibial tuberosity. For knee joint extension, the joint axis is considered to be located through the femoral condyles. The moment arm for the quadriceps muscle force (QLf) is the shortest distance from the vector to the joint axis. Without the patella, the line of pull of the quadriceps muscle on the leg-foot segment (tibia) would follow the patellar tendon at the tibia 1 tubercle and would lie parallel to the leg-foot segment (**Fig. 1–34A**). However, the patella is embedded in the quadriceps tendon and pushes the tendon away from the femur, changing the angle that the patellar tendon makes with the tibia and changing the line of pull of the quadriceps muscle away from the knee joint axis (**Fig. 1–34B**). The effect of changing the line of pull of the quadriceps muscle on the tibia is to increase the moment arm. Given an increased moment arm, the same magnitude of quadriceps force will produce greater torque (and a greater angular acceleration) in Figure 1–34B than in Figure 1–34A.

FIGURE 1-34

Sagittal T1-weighted MRIs of the knee. **A.** The line of pull and moment arm of the quadriceps muscle without the patella. **B.** With the patella's pulley effect, the line of pull of the muscle is deflected away from the joint axis, increasing the moment arm of the muscle force.



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Foundational Concepts 1–12

Muscle Force Vectors

- Active or passive tension in a muscle creates a force (pull) on all segments to which the muscle is attached, although one may choose to consider only one

segment at a time.

- The point of application of a muscle force vector is located at the point of attachment on a segment.
- The muscle action line is in the direction of the fibers or tendon *at the point of application.*
- Muscle vectors (like all vectors) are represented by a straight line. Once the action line of the muscle approximates the alignment of the tendon or muscle fibers at the point of application of the muscle, the vector continues in a straight line regardless of any change in direction of muscle fibers or tendons beyond the point of application.
- The magnitude of a muscle force is generally a hypothetical or theoretical value because the absolute force of a muscle's pull on its attachments cannot be measured noninvasively.

Torque Revisited

We are now ready to add the quadriceps muscle force to the leg-foot segment at the point at which knee extension is to be initiated in the ankle weight example; that is, it is time for John Alexander to attempt to extend his knee.

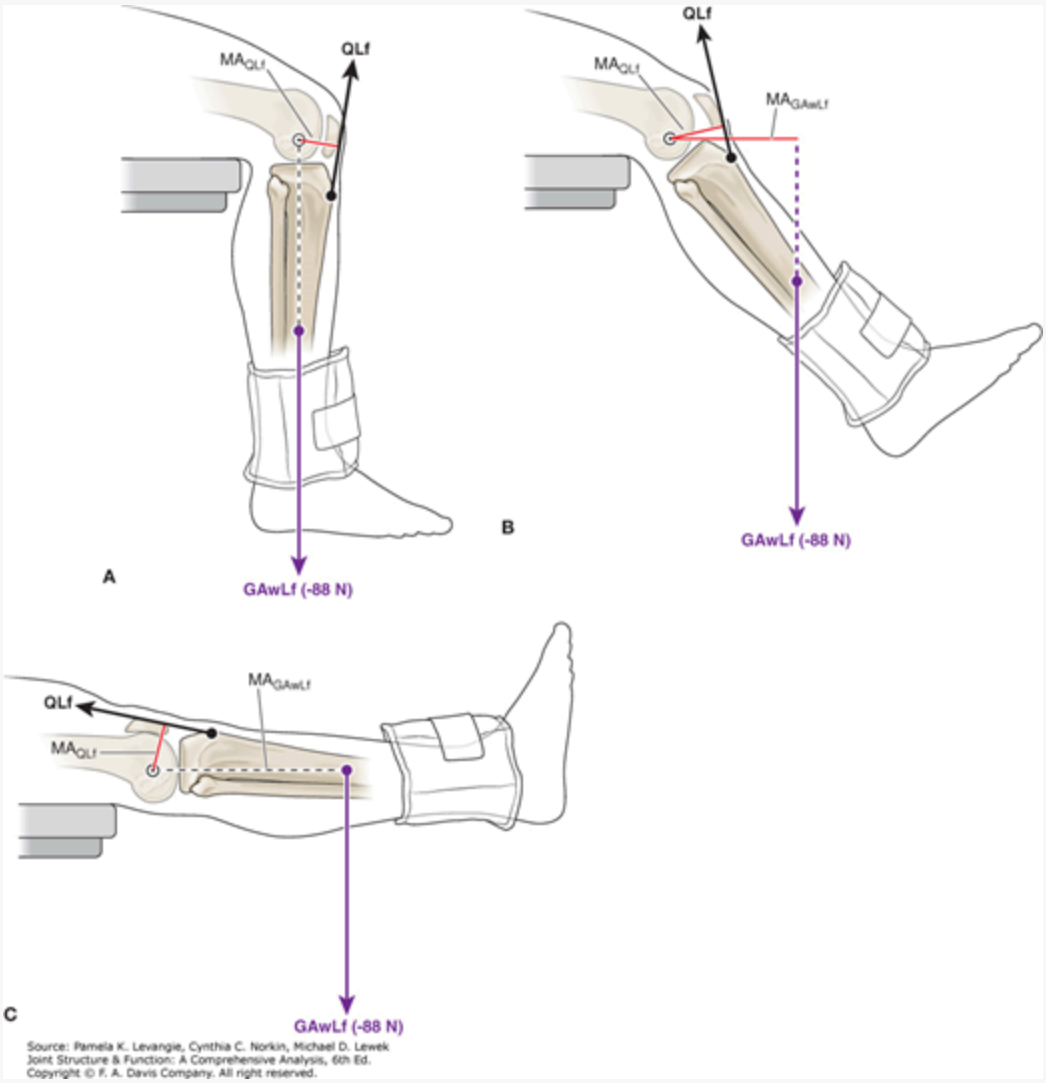
Changes to Moment Arm of a Force

To understand the effect of initiating a quadriceps contraction on John Alexander's leg-foot segment, we need to identify the moment arms for the relevant forces. Figure 1–35A shows the pull of the quadriceps muscle (QLf) on the leg-foot segment as well as the resultant pull (GAwLf) of both gravity and the ankle weight. For some vectors, finding the shortest (or perpendicular) distance (moment arm) between a force and a joint requires that the force vector be extended. The effect of a force is not changed by extending the vector graphically—nor should it be considered to change the magnitude of the force. When GAwLf is extended in Figure 1–35A (dashed line), the force still has a magnitude of –88 N, but its moment arm (MA) is effectively zero because the extended vector passes through the knee joint axis (MA ≈ 0). Therefore, gravity and the ankle

weight create negligible torque on the leg-foot when the knee joint is still at 90° of knee flexion. Given that GAwLf produces no torque in this knee joint position, a relatively small force by the quadriceps muscle (applied through its relatively larger moment arm estimated in this example to be 0.03 m) will yield a net resultant torque in the direction of knee extension. The scenario will change as soon as the knee is no longer at 90° because the torques created by the forces applied to the leg-foot segment will change as the orientation of the leg-foot segment changes.

FIGURE 1-35

A. At 90° of knee flexion, the moment arm (MA_{QLf}) for the quadriceps (QLf) is shown. The moment arm for GAwLf is effectively zero because the extended vector (dashed line) passes through the knee joint axis. **B.** At 45° of knee flexion, MA_{QLf} increases slightly but the moment arm for GAwLf (MA_{GAwLf}) increases substantially, thus considerably increasing the torque against which the quadriceps must now work to extend the knee. **C.** At 0° of knee flexion, MA_{QLf} has gotten slightly smaller whereas MA_{GAwLf} has increased substantially. The quadriceps will now have to tremendously increase the force of its contraction to generate sufficient torque to maintain the knee at full extension.



In Figure 1–35B, the leg-foot segment has been brought farther into the knee extension (~45° of knee flexion). As the segment moves in space, the relation between the forces applied to the segment and the segment itself change. The gravitational force (GWbLf) now lies at a substantially greater distance from the knee joint axis (the moment arm has increased). The quadriceps muscle force also has a larger moment arm (increasing the estimated distance from 0.03 m to 0.05 m), but the increase is minimal in comparison with the increase in the moment arm of GAwLf (increased from 0 m to an

estimated 0.27 m). The magnitude of the quadriceps force necessary to continue knee extension from this new point in the ROM is now considerably greater.

If the moment arm for GAwLf in Figure 1–35B is 0.27 m, then a force of 288 N would create a clockwise (knee flexion) torque of 23.76 Nm. The quadriceps muscle would have to create a counterclockwise (extension) torque of 23.76 Nm to maintain rotary equilibrium. If the moment arm for the quadriceps muscle is 0.05 m, the quadriceps force would have to be 475.2 N (~107 lb) just to maintain the leg-foot segment at 45°. The quadriceps force in fact has to be greater than 475.2 N in order to create a net unbalanced torque in the direction of continued knee extension.

Figure 1–35C shows the leg-foot segment at the end of knee extension (0° of knee flexion). GAwLf is now perpendicular to the leg-foot segment and farther away from the joint axis (its moment arm increasing to an estimated 0.36 m). The line of pull of the quadriceps is now closer to the joint axis than when the knee was at 45°, decreasing its moment arm. The magnitude of GAwLf remains unchanged at –88 N. However, GAwLf now creates a clockwise (flexion) torque of 31.68 Nm. If the reduced moment arm for the quadriceps is now 0.01 m, the muscle will have to generate a force of 3,168 N (712 lb) to maintain this fully extended knee position (rotary equilibrium).

Whenever any force is applied at 90° to the long axis of a segment (as GWbLf is in Fig. 1–35C), the length of the moment arm for that force is maximal. The moment arm will lie parallel to the long axis of the segment (the lever) and, under these conditions, can also be referred to as the **lever arm** of the force. Because the moment arm of a force is greatest when that force is at 90° to the segment, the torque *for a given magnitude of force* will also be greatest when the force is applied at that angle. As John Alexander

uses his quadriceps to extend his knee, the quadriceps will have to exert the most force on the fully extended knee, making this the hardest knee position to maintain.

 Foundational Concepts 1–13

Moment Arms and Torque

- As the moment arm of a force increases, its potential to produce torque increases.
- The moment arm of a force is at its maximum when the force is applied at 90° to its segment.
- The moment arm of a force is minimal (0.0) when the action line of the force passes through the center of rotation of the segment to which the force is applied.
- The force of gravity will be applied at 90° to a segment when that segment is horizontal to the ground, and will be applied at 0° when the segment is vertical.
- When the moment lies parallel to the segment, the term *lever arm* may also be used to describe the shortest distance from the force vector to the joint axis.

 Expanded Concepts 1–7

Work, Energy, and Power

Work, energy, and power are combinations of kinematic and kinetic variables. Although these terms are not the focus of this chapter, these important concepts in biomechanics are related to muscle force production, and warrant brief discussion. **Work** is defined as force applied over some distance and is expressed in units of joules (or Nm).

$$W = F \times D$$

If an examiner applied a load of 10 N to the distal forearm when testing elbow flexion (see Fig. 1–10) and the distal forearm moved 2 cm at the point of application of the force, the examiner would have completed 0.2 J (10 N × 0.02 m) of work. **Power** is the *rate* of work production and is expressed in watts (or J/sec).

$$P = W \div \text{time}$$

If the examiner created the 2 cm movement of the distal forearm over a 2 second

period, he or she would have generated 0.1 watt of power. Although this concept may seem trivial, power is frequently misused when talking about force. Power is the rate of work production and is much more complicated than simple force production. The final concept is energy. **Energy** is the ability to perform work and is expressed in the same units as work (joules).

Angular Acceleration With Changing Torques

We approached our analysis of the ankle weight exercise in Figures 1–35A–C as a series of freeze-frames or single points in time. Human motion, of course, is not a series of points of equilibrium but is rotation of the bony segment around a joint axis with a changing rotary (angular) acceleration as the segment moves through its arc of motion.

We established that the quadriceps muscle would need to contract with a force of approximately 3,168 N at 0° of knee flexion to hold the limb against the flexion (clockwise) torque of the weight of the limb and ankle weight (Fig. 1–35C). If a quadriceps contraction of 3,168 N is generated at 90° of knee flexion (Fig. 1–35A), the ankle weight and weight of the limb (GAwLf) would provide almost no resisting flexion torque because of the small moment arm. There would be a large net extension torque (ΣT) produced by the quadriceps contraction. The net torque would produce a substantial angular acceleration of the leg-foot segment. *[Note: Newton's second law (law of acceleration) indicates not only that linear acceleration is directly proportional to the net unbalanced force, but that angular (rotary) acceleration is proportional to the net unbalanced torque.]* If the quadriceps muscle continues to contract with a force of 3,168 N as knee extension occurs, the net unbalanced extension torque will gradually decline because the flexion torque generated by GAwLf gradually increases as the moment arm of GawLf increases, reaching its maximum moment arm and flexion

torque at full knee extension. As the net unbalanced torque decreases, so too does the angular acceleration of the leg-foot segment. The angular acceleration will reach zero at the point of full knee extension when posterior knee structures, along with gravity and the weight boot, contribute to checking further knee extension (with equilibrium to the leg-foot segment restored when $\Sigma\tau = 0$ and $\Sigma F = 0$). Even if the force of the quadriceps contraction is constant and weight of the leg-foot and ankle weight are constant, the angular acceleration of the limb will vary with the changes in moment arm of the forces. *[Note: It is a characteristic of the quadriceps muscle that its moment arm is greatest in, approximately, the middle of the knee ROM. The point in the joint ROM where the moment arm of a muscle is largest, thereby producing its greatest potential torque, is essentially unique to each muscle and is not predictable by any rules.]*

Moment Arm and Angle of Application of a Force

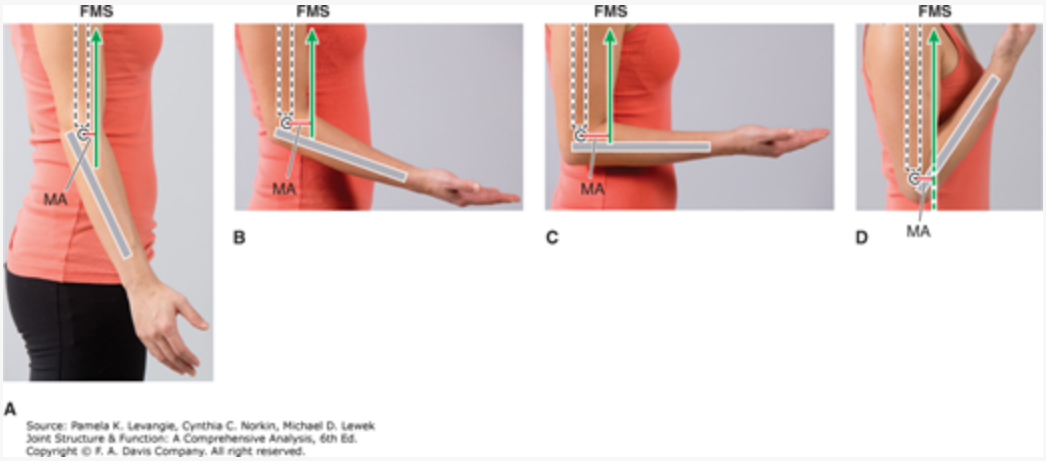
We have seen that the moment arm of a force can change as a segment rotates around its joint axis and as the segment changes its orientation in space. The length of the moment arm is directly related to the **angle of application of the force** on the segment. The angle of application of a force is the angle made by the intersection of the force vector and the segment to which it is applied *on the side of the joint axis under consideration.* Any time a force is applied to a segment, at least two angles are formed. The viewer's eye will automatically tend to find the acute (rather than the obtuse) angle. To identify the angle of application of a force, the angle *on the side of the joint axis* is the angle of interest, regardless of whether that angle is acute (less than 90°) or obtuse (greater than 90°).

Figure 1–36 shows a force vector (e.g., brachialis) at four different angles of application

in relation to the forearm. It can be seen how the angle of application of the force affects the size of the moment arm. When the angle of application of a force is small (~35°), the moment arm will be small (**Fig. 1–36A**). As the angle of application for the force increases (~70°), the moment arm increases because the vector lies farther from the joint axis (**Fig. 1–36B**). When the angle of application of the force is at 90°, the moment arm is as large as it will get (**Fig. 1–36C**). When the angle of application moves beyond 90° (e.g., 145°) (**Fig. 1–36D**), the moment arm is measured from the extended tail of the vector; the extended tail swings closer to the joint axis as the angle of application of the force continues to increase beyond 90°. As the moment arm of a muscle force changes, so too does its ability to generate torque as we saw already with the quadriceps in Figures 1–35A–C.

FIGURE 1-36

Changes in the angle of application of the force (the angle between the force vector and lever *on the side of the axis*) on the forearm result in changes to the moment arm (MA) of the force (Fms). **A.** The moment arm is smallest when the angle of application is smallest (~35°). **B.** As the angle of application of Fms increases (70°), the MA increases. **C.** The MA is maximal when the force is applied at 90° to the lever. **D.** When the angle of application of Fms is greater than 90° (145°), the vector must be extended to find the length of the MA.



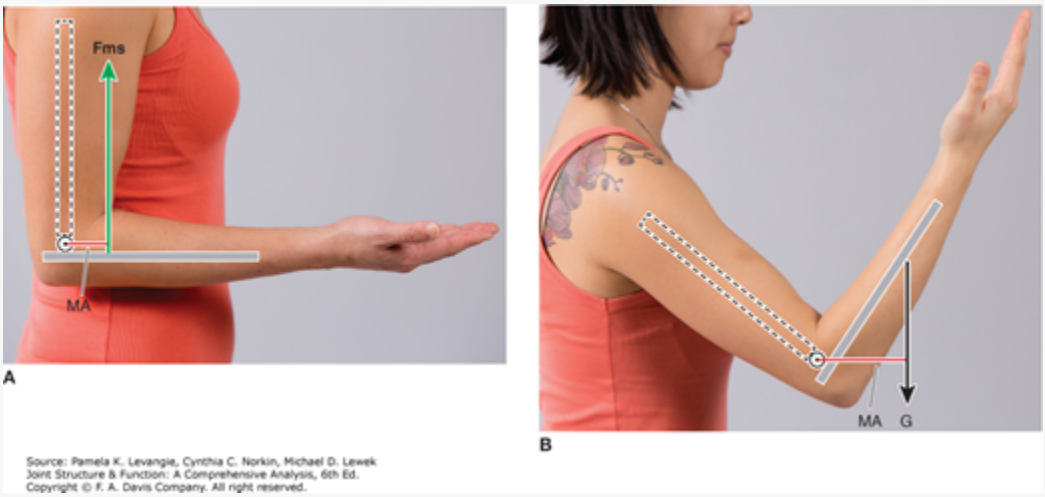
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In Figures 1–36A–D the moment arms and torques changed as a segment moved through ROM. There will also be a change in moment arm and angle of application for gravity as a segment moves through its joint ROM. However, the basis for the change in angle of application of a gravitation force is different than for a muscle. A gravitational force is always vertically downward. Consequently, it is the orientation of the *segment in space* rather than the joint angle that causes a change in the angle of application of gravity.

In **Figure 1–37A**, the elbow joint is at 90° and the forearm is parallel to the ground (horizontal). Therefore, gravity-on-forearm will be applied at 90° to the forearm and the moment arm of gravity-on-forearm will be at its greatest. In **Figure 1–37B**, the elbow joint is still at 90° but the forearm is no longer parallel to the ground. Consequently, the moment arm for gravity-on-forearm (and its torque) will be less. Because the *magnitude* of the gravitational force on a segment does not change as the segment moves through space, the torque produced by the weight (G) of a segment is directly a function of the position of the segment in space rather than the joint angle.

FIGURE 1-37

A. When the forearm is parallel to the ground (horizontal), gravity (G) will always have an angle of application of 90° and the moment arm (MA) for the force will be as large as it can get. **B.** Although the elbow is still at 90° of flexion, the angle of application of G is now applied at 45° to the forearm and the MA is smaller than in position A.



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Unlike gravity that has a fixed center of mass in an unsegmented object, other external forces (e.g., a manual resistance, mechanical resistance, or applied load) may be able to change the point of application of the force to different points on the body segment. When the point of application of an external force on the segment changes, so too does the moment arm (and torque) of that force. As a manual resistance to a joint motion is shifted away from the joint axis, production of the same torque requires less force because of the larger moment arm.



Foundational Concepts 1–14

Moment Arms and Angle of Application

For all forces (internal and external):

- The moment arm of a force is greatest when the force is applied at, or as close as it will get to, 90° (perpendicular) to the segment it is acting on.
- The moment arm of a force is least when the force is applied at, or as close as it will get to, 0° or 180° (parallel) to the segment.
- The angle of application (and moment arm) of a muscle force will vary with the angle of the joint it is crossing because the segment moves in space.
- The angle of application (and moment arm) of gravity will vary with the orientation of the segment in space, regardless of the joint angle.
- Most muscles are aligned fairly parallel to the bony segments to which they attach; consequently, muscles that have an angle of application that comes close to 90° are the exception rather than the rule.

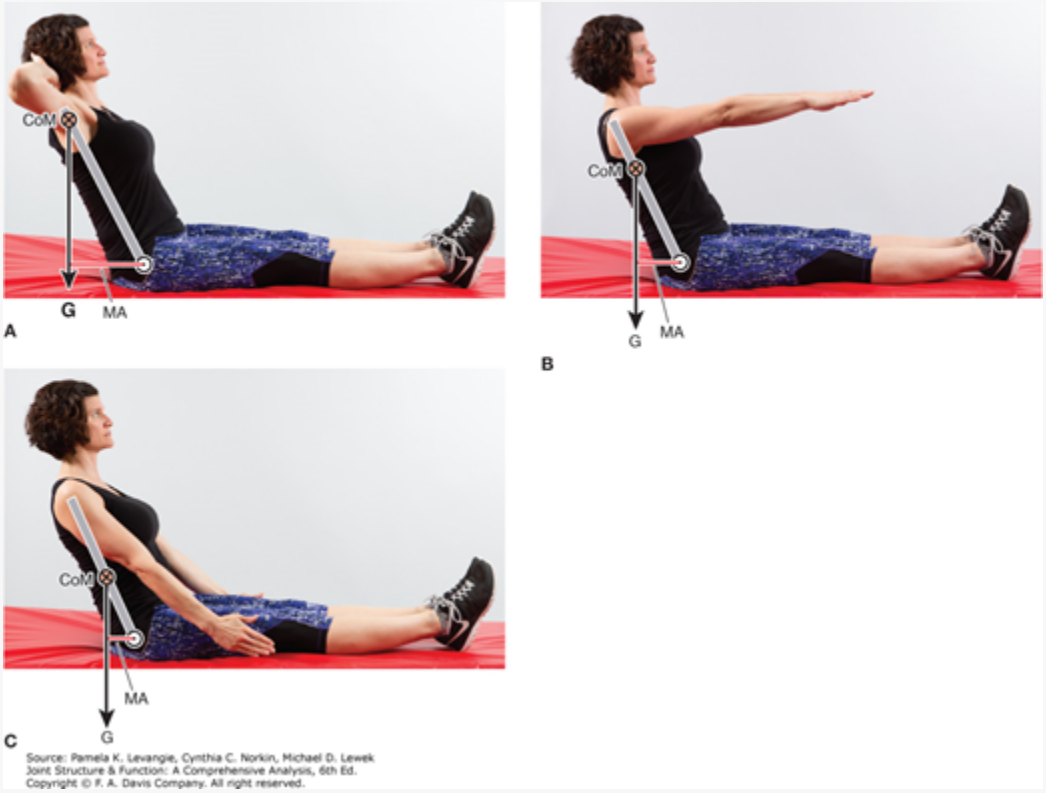
Changing the Gravitation Moment Arm by Changing the Center of Mass

Figure 1-38 shows three graded exercises for the abdominal muscles. The vertebral interspace of L5 to S1 is considered here to be a hypothetical axis about which the head-arms-trunk (HAT) rotates. In each figure, the arms are positioned slightly different, which results in a rearrangement of mass and a shift in the center of mass. In Figure 1–38A, the arms are raised above the head. As a result, the center of mass of the head-arms-trunk is located closer to the head (cephalad), with a line of gravity that is farther from the axis of rotation (greater moment arm) than when the arms are in the other two positions. The torque generated by gravity is counterclockwise (a trunk extension torque or moment). To maintain this position (rotational equilibrium), the abdominal muscles must generate an equal torque in the opposite direction (an equivalent flexion moment). In Figures 1–38B and C, the centers of mass move caudally as the arms are lowered. The relocation of the center of mass of the head-arms-trunk brings the line of gravity closer to the axis. Because the weight of the upper body does not change when the arms are lowered, the magnitude of torque applied by gravity to the upper body

diminishes in proportion to the reduction in the moment arm. The decreased gravitational torque requires less opposing torque by the abdominal muscles to maintain equilibrium. Consequently, it is easiest to maintain the position in Figure 1–38C and hardest to maintain the position in Figure 1–38A.

FIGURE 1-38

Changes in arm position in a sit-up cause the center of mass (CoM) of the upper body segment to move, the moment arm (MA) to change, and the torque of gravity (G) to decrease from position A to position C.



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Lever Systems, or Classes of Levers

One perspective used to assess the relative torques of internal and external forces is

that of **lever systems,** or **classes of levers.** Although applying the terminology of lever systems to human movement requires some important oversimplifications, the terms (like those of the cardinal planes and axes) provide a useful frame of reference and a common terminology that permit us to break complex kinetics into describable component parts.

A lever is any rigid segment that rotates around a fulcrum. A lever system exists whenever two forces are applied to a lever in a way that produces opposing torques. To apply concepts of levers to a bony segment, we must also consider the joint axis of the bony segment to be relatively fixed (a problematic assumption, as we have already discussed). However, it is common to apply the concepts of levers to bony segments when looking at the net rotation produced by two forces, typically (1) a muscle force and (2) a gravitational or external force.

In a lever system, the force that is producing the resultant torque (the force acting in the direction of rotation) is called the **effort force (EF).** Because the other force must be creating an opposing torque, it is known as the **resistance force (RF).** Another way to think of effort and resistance forces acting on a lever is that the effort force *is always the winner* in the torque game, and the resistance force *is always the loser* in producing rotation of the segment. The moment arm for the effort force is referred to as the **effort arm (EA),** whereas the moment arm for the resistance force is referred to as the **resistance arm (RA).** Once the effort and resistance forces are identified and labeled, the position of the axis and relative sizes of the effort and resistance arms determine the class of the lever.

A **first-class lever** is a lever system in which the axis lies somewhere between the point

of application of the effort force and the point of application of the resistance force (Fig. 1–39A). A **second-class lever** is a lever system in which the resistance force has a point of application between the axis and the point of application of the effort force (Fig. 1–39B); this always results in the effort arm being larger than the resistance arm. A **third-class lever** is a lever system in which the effort force has a point of application between the axis and the point of application of the resistance force (Fig. 1–39C); this always results in the resistance arm being larger than the effort arm.



Foundational Concepts 1–15

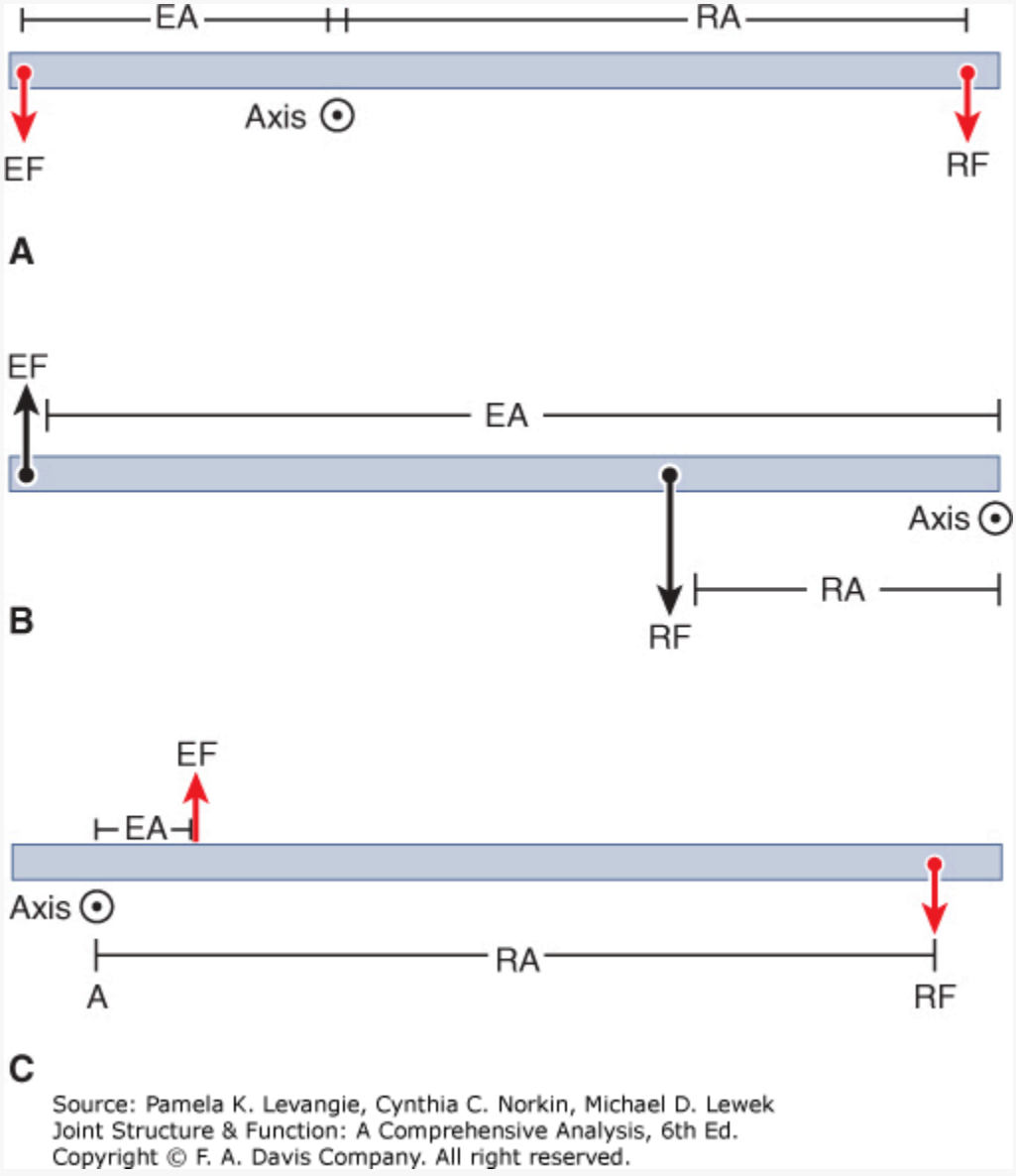
Muscles in Lever Systems

- When a muscle is contracting **concentrically** (actively shortening), the muscle must be moving the segment to which it is attached in the direction of its pull. Therefore, the muscle is "winning" and will be the effort force. In most instances, these muscles are acting on third-class levers.
- When a muscle is contracting **eccentrically** (actively lengthening), the muscle must be acting in a direction opposite to the motion of the segment; that is, the muscle must be "losing" and is the resistance force. When a muscle is contracting eccentrically, it generally serves to *control* (slow down) the acceleration of the segment produced by the effort force and, in most instances, is acting on a second-class lever.
- When a lever is in rotational equilibrium, the muscle acting on the lever is contracting **isometrically.** In such a case, labeling the muscle as the effort or resistance force is arbitrary so the class of lever on which the muscle is acting depends on the labeling.
- There are a few muscles that lie on one side of an axis while the center of mass of the limb being moved by the muscle acts on the other side of the axis. These muscles act on, in the human body, fairly unusual first-class levers.

FIGURE 1-39

A. First class lever: the axis lies between the effort force (EF) and the

resistance force (RF). **B.** Second class lever: The RF lies between the axis and the EF so that the effort arm (EA) is always larger than the resistance arm (RA). **C.** Third class lever: The EF lies between the axis and the RF so that RA is always larger than EA.



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Mechanical Advantage

Mechanical advantage (M Ad) is a measure of the mechanical efficiency of the lever

system (the relative effectiveness of the effort force in comparison with the resistance force). Mechanical advantage is related to the classification of a lever and provides an understanding of the relationship between the torque of an external force (which we can roughly estimate) and the torque of a muscular force (which we can estimate only in relation to the external torque). Mechanical advantage of a lever is the ratio of the effort arm (EA; moment arm of the effort force) to the resistance arm (RA; moment arm of the resistance force), or:

$$\text{M Ad} = \frac{\text{EA}}{\text{RA}}$$

When the effort arm is larger than the resistance arm (as in the second-class lever in Fig 1–39B), the mechanical advantage will be greater than one. When the mechanical advantage is greater than one, the magnitude of the effort force is working through a larger moment arm and can be smaller than the magnitude of the resistance force while still creating greater torque to "win." The "advantage" is that a small force can defeat a larger force.

When the effort arm is smaller than the resistance arm (as in the third-class lever in Fig. 1–39C), the mechanical advantage will always be less than one. A third-class lever is "mechanically inefficient" or is working at a "disadvantage" because the magnitude of the effort force *must* always be *greater* than the magnitude of the resistance force for the torque of the effort force to "win" (produce a greater torque) while working through a smaller moment arm.

It is important to note the mechanical advantage of a lever is determined by the lengths

of the moment arms and not by the *magnitudes* of the effort and resistance forces. If an effort force is winning (as it must be to earn the "effort force" title) and has a magnitude smaller than that of the resistance force, the muscle *must* be working through a larger moment arm than the resistance or it couldn't "win." However, if the moment arm of the effort force is larger than that of the resistance (e.g., acting on a second-class lever), the magnitude of the effort force is *not necessarily* smaller than that of the resistance force. If both the magnitude of the effort force and the moment arm of the effort force are larger than those of the resistance force, the net torque and angular acceleration will be large.



Foundational Concepts 1–16

Mechanical Advantage and Classes of Levers

- In all second-class levers, the mechanical advantage (M Ad) of the lever will always be greater than one. The magnitude of the effort force can be (but is not necessarily) less than the magnitude of the resistance.
- In all third-class levers, the mechanical advantage of the lever will always be less than one. The magnitude of the effort force *must* be *greater* than the magnitude of the resistance for the effort to produce greater torque.
- The mechanical advantage of a first-class lever can be greater than, less than, or equal to one. However, it is often true of first-class levers in the body that the moment arm of the muscle will be shorter than the moment arm of the external force, thus rendering the lever inefficient.
- When the muscle is the effort force in a lever with a mechanical advantage of less than one, the necessary expenditure of energy to produce sufficient muscle force to "win" is offset by the need for minimal linear displacement and velocity of the muscle to produce proportionally greater angular displacement and angular velocity of the distal portions of the segment.
- When an external force is the effort force in a lever with a mechanical advantage greater than one (e.g., a second-class lever), the magnitude of the effort force can be small in comparison with the resistance force, but less is gained in angular displacement and velocity.

Most muscles have a moment arm that is smaller than that of the external force(s) against which they are working because most muscles lie closer to joint axes than do external forces. Consequently, the muscle must be able to create large forces not only to "win" (work concentrically as the effort force) but also to eccentrically control external forces when the external force is "winning." As shall be shown in Chapter 3, a muscle is typically structured to optimize production of the large force required to produce sufficient torque when working through a small moment arm, whether functioning eccentrically as a resistance force or concentrically as an effort force.

Limitations of Analysis of Forces by Lever Systems

Although the conceptual framework of lever systems described here provides useful terms and some additional insights into rotation of segments and muscle function, there are distinct limitations to this approach. Our discussion of lever systems ignored the established fact that the rotation of a lever requires at least one force couple. An effort force and a resistance force are *not* a force couple because effort and resistance forces produce rotation in the opposite direction (whereas a force couple works together to produce rotation in the same direction). The second force in the "couple" is generally an articular constraint (a joint reaction force or capsuloligamentous force) that may serve as the pivot point for the rotation. Consequently, a simple lever-system approach to analyzing human motion requires oversimplification that fails to take into consideration key elements that affect human function and structural integrity. Torques on human segments are not simply produced by one muscle group and one external force but result from many forces *including* vertical and horizontal translatory forces that are typically ignored in a simple lever approach.

Force Components

In the analysis of the quadriceps force producing an extension torque on the leg-foot segment (Fig. 1–35A–C), we did not identify the second part of the force couple that is actually required to produce the knee extension torque. We also did not identify the mechanism by which translatory equilibrium ($\Sigma F_V = 0$ and $\Sigma F_H = 0$) of the leg-foot segment was established—a necessary condition for rotation of a joint around a relatively fixed axis. To examine the degree to which these conditions are met (net extension torque and translatory equilibrium), we need to understand how to resolve forces into their component parts.

A force of constant magnitude produces its greatest torque when the force is applied at 90° to the lever. At other angles of application for that same force, the torque will be less even if the magnitude of the force is unchanged. If the *same* magnitude of force produces *less* torque when the angle of application is not 90°, some of that force must be doing something other than producing rotation. Torque, in fact, is typically considered to be produced only by that *portion* of the force that is directed toward rotation. When the force is applied to a lever at some other angle (greater or less than 90°), the component of force that *is* applied at 90° to the lever will contribute to rotation. Consequently, the portion of a force that is applied at 90° to the segment is known as the **perpendicular** (**rotary** or **"y"**) **component** of the force **(Fy).** The rotary component is the "y" component because the long axis of the body segment is usually the reference line or essentially the x-axis. Consequently, the component that is perpendicular to the segment is the y-axis.

The magnitude of Fy can be found graphically by the process of **resolution of forces.** In resolution of forces, the original (or **total**) force (F_{TOT}) is broken down into two components that are perpendicular to each other. Just as two concurrent forces can be composed into a single resultant vector (composition by parallelogram), a single vector (F_{TOT}) can be resolved into two concurrent components. In this instance, the components will be specifically constructed so that one component (Fy) lies perpendicular to the segment and the second component (Fx) is drawn parallel to the segment.

Resolving Forces into Perpendicular and Parallel Components

In Figure 1–40, three steps are shown to resolve the quadriceps force (QLf or F_{TOT}) into its perpendicular (Fy_{QLf}) and parallel (Fx_{QLf}) components:

Step 1 (see Fig. 1–40A). A line with the *same point of application* as the original force (QLf) is drawn perpendicular to the long axis of the lever in the direction of the original force (this is the "draft" line of component Fy). A second line with the *same point of application* as QLf is drawn parallel to the long axis of the lever in the direction of the original force (this is the "draft" line of component Fx). The draft Fx and Fy component lines should be drawn long enough to reach or go past the arrowhead of the original vector QLf.

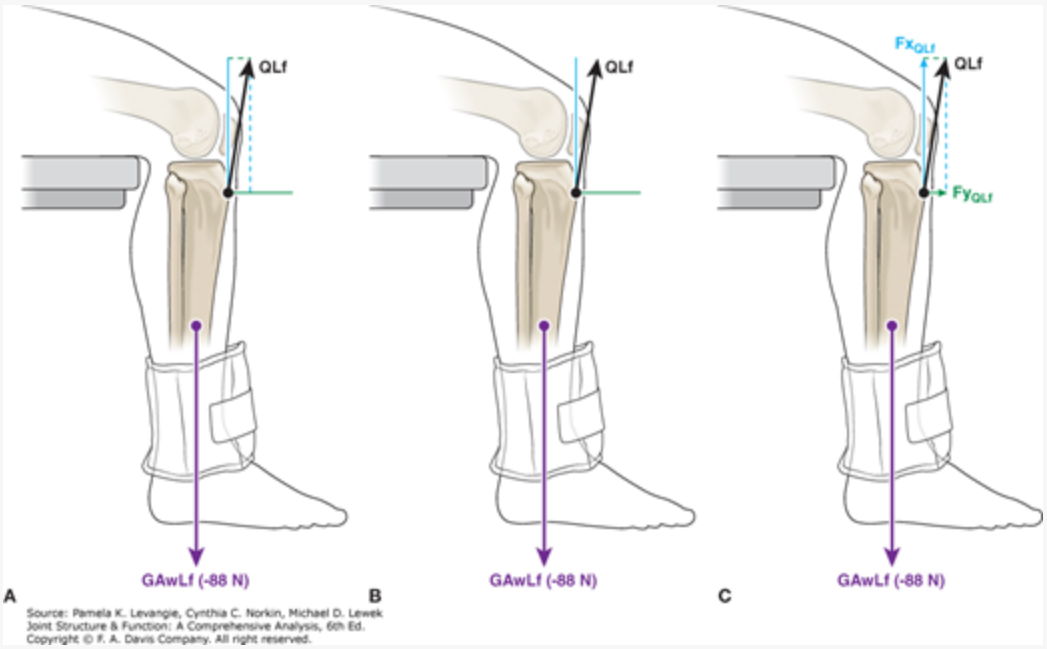
Step 2 (see Fig. 1–40B). The rectangle is completed (closed) by drawing (from the arrowhead of the FTOT or QLf vector) lines that are parallel to each of the draft Fx and Fy components.

Step 3 (see Fig. 1–40C). All the lines are "trimmed," leaving only the completed

rectangle. Components Fy (the rotary component) and Fx (the translatory component) each form one side of the rectangle, have a common point of application, and have arrowheads at the corners of the rectangle; force QLf (F_{TOT}) is now the diagonal of the rectangle. It is important that the lengths of vectors Fy and Fx are “trimmed” to the confines of the rectangle to maintain the proportional relation among Fx, Fy, and F_{TOT}. This proportional relation will permit determination of the predominant and relative effects of F_{TOT}.

FIGURE 1-40

When a force (e.g., QLf) is resolved into its components, **A.** two lines that are perpendicular and parallel to the long axis of the bone are drawn from the point of application of the original QLf force; **B.** a rectangle is completed by drawing parallel lines from the arrowhead of the original QLf force; and **C.** the perpendicular (Fy) and parallel (Fx) component vectors are the adjacent sides of the rectangle that have a common point of application with the original force. The original force (QLf) is the diagonal of the rectangle.



Perpendicular and Parallel Force Effects

Focusing on the components of the quadriceps force (Fy_{QLf} and Fx_{QLf} in Fig. 1–40C) rather than on the quadriceps "total" force (QLf), it can be seen more readily that the components of QLf have the potential to create three different motions of the leg-foot segment: vertical motion, horizontal motion, and rotary motion. Component Fx_{QLf} (Fig. 1–40C) will tend to create vertical translatory motion, while component FyQ_{Lf} will both create rotation and tend to create horizontal translation. Because a force component (e.g., FyQ_{Lf}) may create both rotation and translation, labeling components as "rotary" and "translatory" can be confusing. For the remainder of this chapter, therefore, we will refer to Fy exclusively as the perpendicular component and to Fx exclusively as the parallel component; the effects of Fy and Fx (rotation or translation) will depend on the scenario.

We have already established that determining whether a segment is in equilibrium or

not requires assessment of ΣF_V, ΣF_H, and Σ_τ (Newton's law of equilibrium). Consider, however, that a force that is perpendicular to the segment and also horizontally oriented in space(e.g., Fy_{QLf} in Fig. 1–40C), with movement of the segment in space, may become vertically oriented in space while still remaining perpendicular to the segment (e.g., if the knee moves to full extension). The same shift in spatial orientation may happen with an Fx component. Because the relationship of the force or force component to the segment is the critical factor in understanding the effect of the force(s) on the joint (not whether a force is vertical or horizontal in space), we will proceed to assess translatory equilibrium of a body segment by considering the sum of the Fx (ΣFx) and Fy (ΣFy) components. The rotary equilibrium of the body segment will be assessed by determining the sums of the torques contributed by the forces or force components (Σ_τ).

Determining Magnitudes of Component Forces

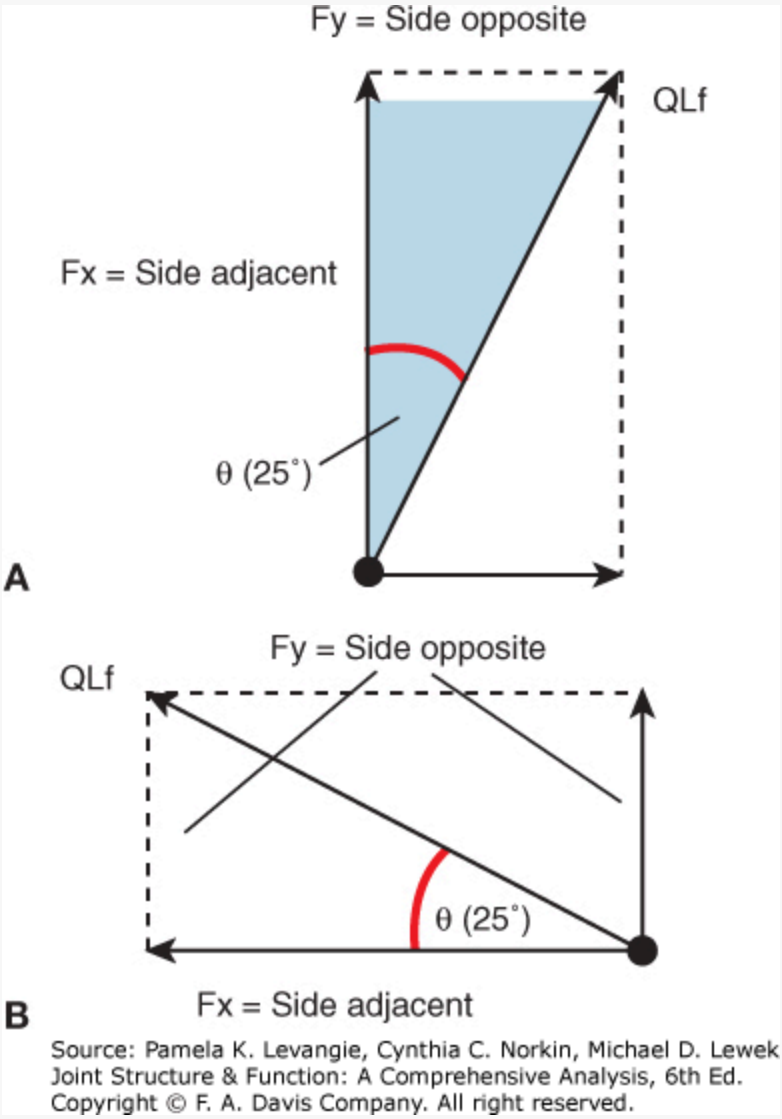
In a figure drawn to scale, the relative vector lengths can be used to estimate the net unbalanced forces. If the vectors are not drawn to scale, the relative magnitudes of Fy and Fx must be determined by using trigonometric functions of sines (sin) and cosines (cos).

Component forces Fx and Fy are always two sides of a rectangle that is divided into two right triangles by their resultant force (F_{TOT}). Based on the example of the quadriceps force (QLf) in **Figure 1–41**, part A of Figure 1–41 shows the quadriceps vector (QLf) and its components in isolation and enlarged. The shaded half of the rectangle is the triangle of interest because the angle (θ) is the angle of application of the quadriceps (the angle between QLf and the long axis of the leg-foot segment on the side of the knee

joint axis). Vector QLf is the hypotenuse (longest side) of the triangle and is now assigned an arbitrary magnitude of 1,000 N. The angle of application θ is 25° in this example. Vector Fx is the side adjacent to angle θ, and vector Fy is the side opposite angle θ. **Figure 1–41B** shows the same rectangle as in **Figure 1–41A**, but reoriented to one more commonly seen when assessing elements in a right triangle. It should be noted that the "side opposite" the angle θ is not *literally* the perpendicular component (Fy) as we initially labeled it but is identical in magnitude (see Fig. 1–41B) because these are opposite sides of a rectangle and, therefore, are equivalent in length.

FIGURE 1-41

Review of trigonometry: **A.** Vector QLf and its Fx and Fy components are replicated (although enlarged) in the same orientation shown in Figure 1–40C. The reference triangle (shaded) shows that QLf is the hypotenuse, Fx is the side adjacent to angle θ, and Fy (or its equivalent length) is the side opposite to angle θ. **B.** The same figure is reoriented in space to provide an alternative visualization.



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Trigonometry now can be used to solve for the magnitudes of Fx (Fx_{QLf}) and Fy (Fy_{QLf}), where the angle of application θ is given as 25° and QLf is 1,000 N. *[Note: The interested reader is referred to Expanded Concepts 1–8 for details of the trigonometric solution.]*

$$Fx_{QLf} = (\cos 25°)(1{,}000\ N) = (0.91)(1{,}000\ N) = 910\ N$$

$$Fy_{QLf} = (\sin 25°)(1{,}000\ N) = (0.42)(1{,}000\ N) = 420\ N$$

It should be recognized that the sum of the magnitudes of the Fx and Fy components

will always be greater than the magnitude of the resultant force (F_{TOT}). As with composition of forces, the resultant is more "efficient." However, analysis of the translatory or rotary effects of any set of forces will produce mathematically equivalent results, whether the total force or the force components are used in appropriate analyses.



Expanded Concepts 1–8

Trigonometric Resolution of Forces

The relation between the lengths of the three sides in a right (90°) triangle is given in the Pythagorean theorem: $A^2 + B^2 = C^2$, where C is the length of the hypotenuse of the triangle (the side opposite the 90° angle) and A and B are, respectively, the lengths of the sides adjacent to and opposite the angle θ (Fig. 1–42A). This equation is useful when the lengths (magnitudes) of at least two of the three sides are known. However, in resolution of forces, we often know at best the length of the hypotenuse and one angle. In these cases, the sine and cosine rules allow the unknown lengths in the triangle to be computed:

$$\sin \theta = \text{Side opposite/hypotenuse}$$

$$\cos \theta = \text{Side adjacent/hypotenuse}$$

These formulas may be used to solve for the length of the adjacent side (Fx) or opposite side (Fy) when angle θ (the angle of application of the force) and the hypotenuse (the magnitude of the force) are known:

$$\text{side opposite} = (\sin \theta)(\text{hypotenuse})$$

$$\text{side adjacent} = (\cos \theta)(\text{hypotenuse})$$

The values of the sine and cosine functions in trigonometry are not arbitrary numbers. The sine and cosine values reflect a fixed relationship between the lengths of the sides of a triangle for any given angle. In Figure 1–42B, a right triangle with an angle θ of 25° is drawn. The large triangle is divided into three smaller triangles of different sizes; the hypotenuses are assigned scaled values of 10 cm, 15 cm, and 20 cm for the smallest to largest triangles, respectively. The corresponding values of Fy (side opposite) will be 4.2 cm, 6.3 cm, and 8.5 cm, respectively; the corresponding

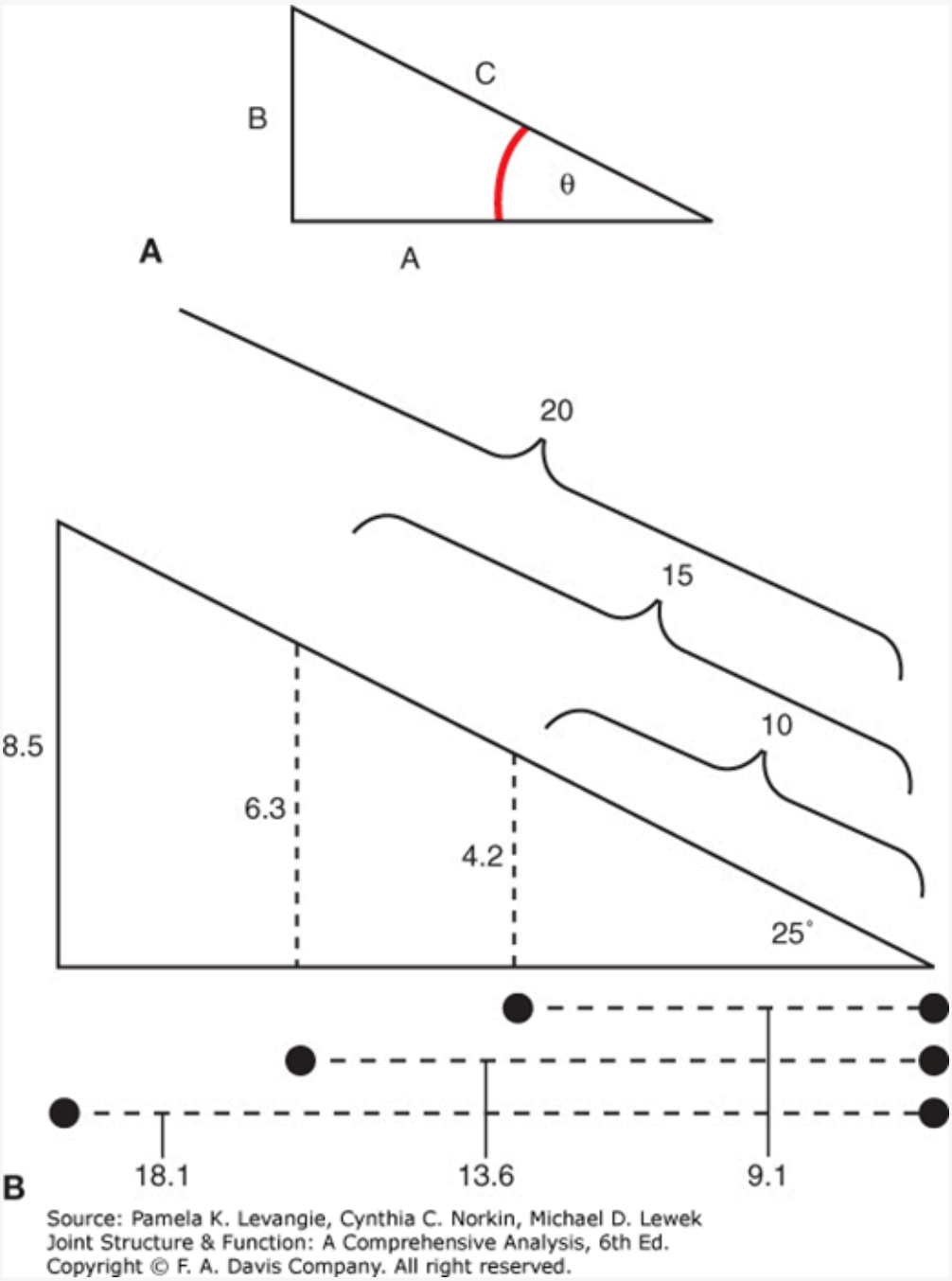
values of Fx (side adjacent) will be 9.1 cm, 13.6 cm, and 18.1 cm, respectively. The ratio of any two sides of one of these 25° triangles will be the same, regardless of the size of the triangle, and that ratio will be the value of the trigonometric function for that angle.

1. For the (side opposite/hypotenuse) for each of the three triangles, the value of sin 25° is 0.42: (4.2/10 = 0.42); (6.3/15 = 0.42); and (8.5/20 = 0.42).
2. For the (side adjacent/hypotenuse) for each of the three triangles, the value of cos 25° is 0.91: (9.1/10 = 0.91); (13.6/15 = 0.91); and (18.1/20 = 0.91).
3. For the (side opposite/side adjacent) for each of the three right triangles, the value of the tangent (tan, or sin/cos) 25° is 0.46: (4.2/9.1 = 0.46); (6.3/13.6 = 0.46); and (8.5/18.1 = 0.46).

This is the "proof" that, for a given angle of application of F_{TOT}, Fx and Fy have a fixed proportional relationship to F_{TOT} *and* a fixed relationship to each other, regardless of the magnitude of F_{TOT} (and regardless of the orientation of the segment in space or whether the force is internal or external).

FIGURE 1-42

Review of trigonometry: **A.** The Pythagorean theorem states that $A^2 + B^2 = C^2$, where A, B, and C are the lengths of the sides of a right triangle. **B.** For a given angle θ (e.g., 25°), the lengths of the side opposite and side adjacent to the angle will have a fixed relationship to each other and to the original force, regardless of the size of the triangle.



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Force Components and the Angle of Application of the Force

We have established that the moment arm (and, therefore, the torque) of a force of constant magnitude will change as the segment rotates around its joint axis and that the change in the moment arm is a function of the change in the angle of application of

the force as the segment rotates. Because the perpendicular component (Fy) of a force is effectively the portion of the total force that produces most if not all of the torque, a change in torque produced by a force of constant magnitude must mean that the magnitude of Fy is changing. This is quite logical because the magnitude of Fy is a function of the angle of application of the force (Fy = [sin θ][hypotenuse]) and we've already established that the angle of application of a force changes as the segment moves around the joint axis.

When a force is applied at 90° to the segment, we've established that the moment arm is as large as it can be for that force. Similarly, the Fy component will be as large as it can be and is equivalent in magnitude to the total force (F_{TOT}); a line drawn perpendicular to the segment (Fy) at the point of application of F_{TOT} will coincide completely with F_{TOT} when F_{TOT} is also at 90°. Consequently, both moment arm and Fy have their greatest magnitude when a force is applied at 90° to a segment. Importantly, there will also be a proportional decrease in Fx as Fy increases. As the angle of application of F_{TOT} gets closer to 90° (perpendicular to the segment), its perpendicular component (Fy) will get longer and its parallel component (Fx) will get shorter. Correspondingly, as F_{TOT} gets closer to an angle of application of 0° (parallel to the segment), its parallel component (Fx) will get longer and its perpendicular component will get shorter. That is, the magnitudes of Fx and Fy are inversely proportional.

The majority of muscles in the body: (1) lie nearly parallel to the segment to which they attach, (2) have relatively small angles of pull (Fx is greater than Fy) regardless of the joint angle, and (3) almost always pull toward (in the direction of) the joint axis. The effect of this arrangement of muscles is that a muscle force generally has a relatively

small Fy component contributing to rotation, with a larger Fx component that is nearly always compressive. Therefore, most of the force generated by a muscle contributes to joint *compression,* rather than joint *rotation.* This arrangement enhances joint stability but means that a muscle must generate a large total force to produce the sufficient torque to move the lever through space.



Foundational Concepts 1–17

Components of Muscle Forces

- The angle of application (angle of pull) for the majority of muscles is small, with an action line more parallel to the bony lever than perpendicular to the bony lever.
- The parallel (Fx) component of a muscle force is larger than the perpendicular (Fy) component for most muscles.
- The parallel component (Fx) of most muscle forces contributes to joint compression, making muscles important joint stabilizers.

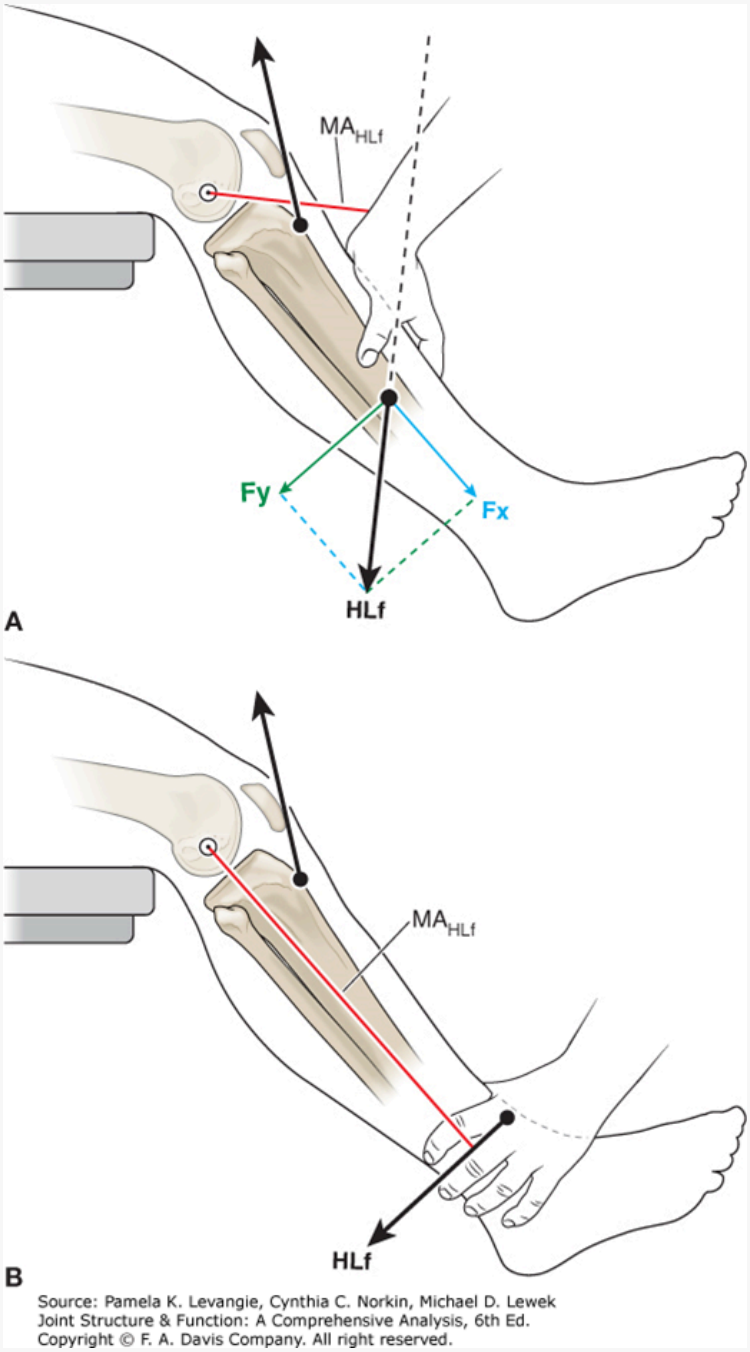
The generalizations that can be made about the majority of muscle forces do not apply to external forces. Gravity is constrained in its direction (always vertically downward) and therefore, has some predictability (the torque of gravity is always greatest when the limb segment is parallel to the ground). Other external forces (e.g., manual resistance) have few if any constraints and, therefore, most often do not have predictable relationships to or effects on a segment. However, the *principles* of forces in relation to the angle of application and the consequential magnitudes of the moment arm, Fy component, Fx component, and torques apply to any and all forces, including those commonly encountered in clinical situations.

Manipulating External Forces to Maximize Torque

In **Figure 1–43**, a manual external force (hand-on-leg/foot [HLf]) is applied to two different points of application on the leg-foot segment with the same amount of force both times. If the goal is to maximize the quadriceps (extension) force needed to maintain the position of the leg-foot segment ($\Sigma_\tau = 0$) with minimum effort on the part of the person applying the opposing flexion force (strengthening the quadriceps with less work by the therapist), one strategy has distinct advantages over the other. In **Figure 1–43A**, HLf has a substantially smaller moment arm than does HLf in **Figure 1–43B** because the resisting hand is placed closer to the knee joint. By moving the resisting hand down the leg-foot segment (Fig. 1–43B), the moment arm of HLf is substantially increased. The flexion torque produced by HLf in Figure 1–43B is going to be much greater than the flexion torque produced by the same magnitude of force in Figure 1–43A. Because the joint angle and angle of pull of the quadriceps is the same in both scenarios (its moment arm doesn't change), the muscle must increase its force of contraction in Figure 1–43B to produce the greater extension torque needed to maintain equilibrium of the leg-foot segment. A similar manipulation of the resistance force (and corresponding quadriceps force) can take place by modifying the angle of application of the manual resistance. If the resisting hand in Figure 1–43B was applied to the leg at 45°(rather than 90°), only about half of the applied force (HLf) would resist rotation ($Fy_{HLf} << HLf$); when the manual resistance is applied at 90° to the leg, HLf = Fy_{HLf}. An understanding of the ability to manipulate both moment arm and angle of application of an external manual force will allow the person applying the force to either increase or decrease the challenge to the quadriceps muscle, depending on the goal of the exercise.

FIGURE 1-43

A. A manual force (HLf) is applied at 90° to the leg-foot segment but at a point relatively close to the knee joint axis. **B.** A manual resistance of the same magnitude produces substantially greater torque when the point of application of HLf is farther from the knee joint axis because the moment arm (MA) is substantially larger.



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Foundational Concepts 1–18

Manipulating External Forces to Maximize Torque Production

- The torque from an external force can be increased by adding to the magnitude of the applied force.

- The torque from an external force can be increased by applying the force perpendicular (or closer to perpendicular) to the lever.
- The torque from an external force can be increased by increasing the distance of the point of application of the force from the joint axis.

Translatory Effects of Force Components

Let us return once again to John and the ankle weight exercise. The goal is to have the segment in translatory equilibrium ($\Sigma Fx = 0$ and $\Sigma Fy = 0$) while having the quadriceps muscle produce a net extension torque. To understand the complex interaction of forces, we must identify the translatory effects of the forces already applied to the leg-foot segment, as well as determine what additional forces, if any, are necessary to create translatory equilibrium.

In Figure 1–44, the forces of gravity and the ankle weight on the leg-foot segment (GAwLf) and the Fx component of the quadriceps (Fx_{QLf}) are both parallel to the leg-foot segment, in opposite directions and sufficiently coplanar and collinear to be considered as part of a linear force system. GAwLf is a joint distraction force because it is away from the knee joint, whereas vector Fx_{QLf} is a joint compression force because it is toward the knee joint. Given the magnitude of GWbLf (-88 N) and that of Fx_{QLf} (determined by our earlier trigonometric calculation to be 910 N for this angle of application of the quadriceps force), there is an apparent net unbalanced parallel force of 822 N (upward). Given this upward pull of the quadriceps, the leg-foot will translate upward until it contacts the adjacent femur (joint reaction force), resulting in a femur-on-leg/foot (FLf) force that is in a similar line and should be able to offset the net upward pull of the quadriceps (FLf = -822 N).

Fy_{QLf} was calculated trigonometrically to be 420 N to the right. In Figure 1–44, there

must be another force (or forces) of 420 N to the left before Fy_{QLf} can produce rotation rather than translation. The most obvious source of that force is the pull of capsuloligamentous structures on the leg-foot segment (CLf) that are positioned to check translation of the tibia out from under the femoral condyles. *[Note: There are other possible forces that might be the source of the missing Fy component(s), but the key here is to recognize that additional forces are needed for translatory equilibrium rather than to identify the source(s).]*

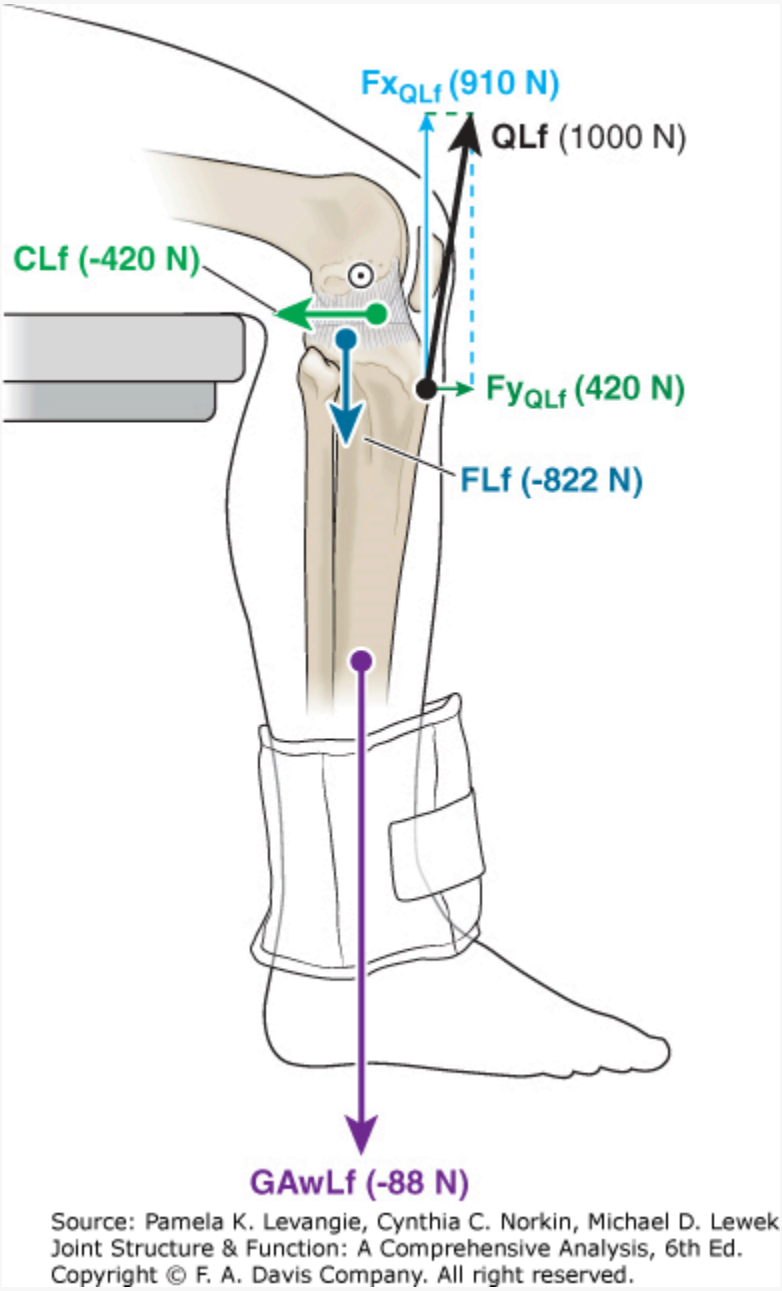
Rotary Effects of Force Components

In Figure 1–44, CLf and Fy_{QLf} are perpendicular to the leg-foot segment. As is true for any forces perpendicular to a segment, these forces will contribute to rotation of the segment if translation is prevented. These two forces qualify as a force couple because they are applied to the same segment in opposite directions, have equal magnitudes, and produce rotation of the leg-foot segment in the same direction (extension). Once the tension in the capsule reaches 420 N (after a small movement of the tibial plateau to the right, or anteriorly), rotation of the leg-foot segment will be initiated around the pivot point of the capsular attachment.

FIGURE 1-44

The forces of GAwLf and Fx_{QLf} (-88 N and 910 N, respectively) are both applied parallel to the leg-foot segment with a net upward force of 822 N. *The leg-foot segment will translate upwardly* until it hits the femur, at which point femur-on-leg/foot (FLf) will push down with a force of -822 N. The perpendicular force of Fy_{QLf} (420 N) will translate the leg-

foot segment to the right (anteriorly) until tension in the capsuloligamentous tissues (CLf) checks further translation. When CLf reaches 420 N of force, CLf and Fy_{QLf} will constitute a force couple and the leg-foot segment will pivot (rotate) around the point of constraint of the capsuloligamentous structures.



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Thus far in this chapter, torque has been computed as F x MA (or F_{TOT} x MA). When a force is *not* applied at 90° to the segment (as is true for the quadriceps force in Fig. 1–44), the torque can equivalently be computed as Fy x MA, although the moment arm (MA) for the Fy component is not the same as the moment arm for F_{TOT} (other than when Fy = F_{TOT}). In Figure 1–44, it can be appreciated that the shortest distance between QLf (F_{TOT}) and the knee joint axis is different than the shortest distance between Fy_{QLf} and the joint axis. Regardless of whether the torque of a force is computed using F_{TOT} x MA or using Fy x MA, the product (magnitude) of torque will be the same. If the moment arm of Fy_{QLf} is 0.06 m, FyQ_{Lf} would generate a torque of 25.2 Nm (420 N x 0.06 m).

$$(F_{TOT} \times MA_{FTOT}) = (Fy \times MA_{Fy})$$



Expanded Concepts 1–9

Rotation Produced by Parallel (Fx) Force Components

Typically, only forces (or force components) that are perpendicular to a bony segment produce torques on that segment. However, *any force* (including an Fx component) with an action line that lies at some distance from a joint axis will produce rotation around that axis (e.g., Fx_{QLf} in Fig. 1–44). Because the contribution of an Fx component to torque (as opposed to its contribution to translation) for any given force is generally small (or smaller than that of Fy), estimating torque based on Fy alone is generally considered to be a reasonable (albeit conservative) estimate of torque produced by the total force.



Foundational Concepts 1–19

Force Components and Joint Motion

- Rotation around a joint axis requires that ΣFx = 0. If ΣFx ≠ 0, there will be an initial translatory motion of the segment (alone or in combination with rotation) in the

direction of the Fx resultant until the translation is checked either by a capsuloligamentous force or by a joint reaction force.

- Rotation around a joint axis requires that the translation be checked ($\Sigma Fy = 0$). If $\Sigma Fy \neq 0$, there will be an initial translatory motion of the segment (alone or in combination with rotation) in the direction of the net Fy resultant until the translation is checked either by a capsuloligamentous force or by a joint reaction force. Once translation is checked, rotation will take place (there is now a force couple).
- The majority of torques on a segment will be produced by forces or force components that are applied at 90° to the segment (Fy) and at some distance from the joint axis.
- Whenever the goal is the rotation of a joint, a net unbalanced torque in the direction of desired movement ($\Sigma_\tau \neq 0$) is necessary.
- The greater the net unbalanced torque on a segment, the greater the angular acceleration of the segment.

Multisegment (Closed-Chain) Force Analysis

The primary difference between John Alexander's ankle weight and leg-press exercises is that the leg-foot segment is "fixed," or weightbearing at both ends when using the leg press. The distal end of the leg-foot segment is constrained by its contact with the footplate and is not free to move in space; the proximal end is connected to the femur and also is not free to move in space. Whenever one end of a segment or set of segments is free to move in space, this is referred to as an **open chain.** When *both* ends of a segment or set of segments are constrained in some way (and not free to move in space), this is referred to as a **closed chain.**

 Expanded Concepts 1–10

Open and Closed Chains

The adjectives "kinetic" or "kinematic" are often used to modify the terms *open chain* and *closed chain.* Although either might be justified, there is no consensus

about which is preferred. At this juncture, the terms “open chain” and “closed chain” are now in such common use that modifiers no longer seem necessary. What is necessary, however, is to avoid *misuse* of the term “closed chain” as equivalent to “weightbearing” (unfortunately, a commonly used but inappropriate synonym). A segment *may* be fixed at both ends by proximal joint attachments and distal weight bearing. However, a segment or set of segments can be weight bearing *without* being fixed at both ends (and without being subjected to the constraints of a closed chain). The effects of open and closed chains on two or more segments will be encountered in subsequent chapters because there are substantial implications for understanding forces that create or limit human movement.

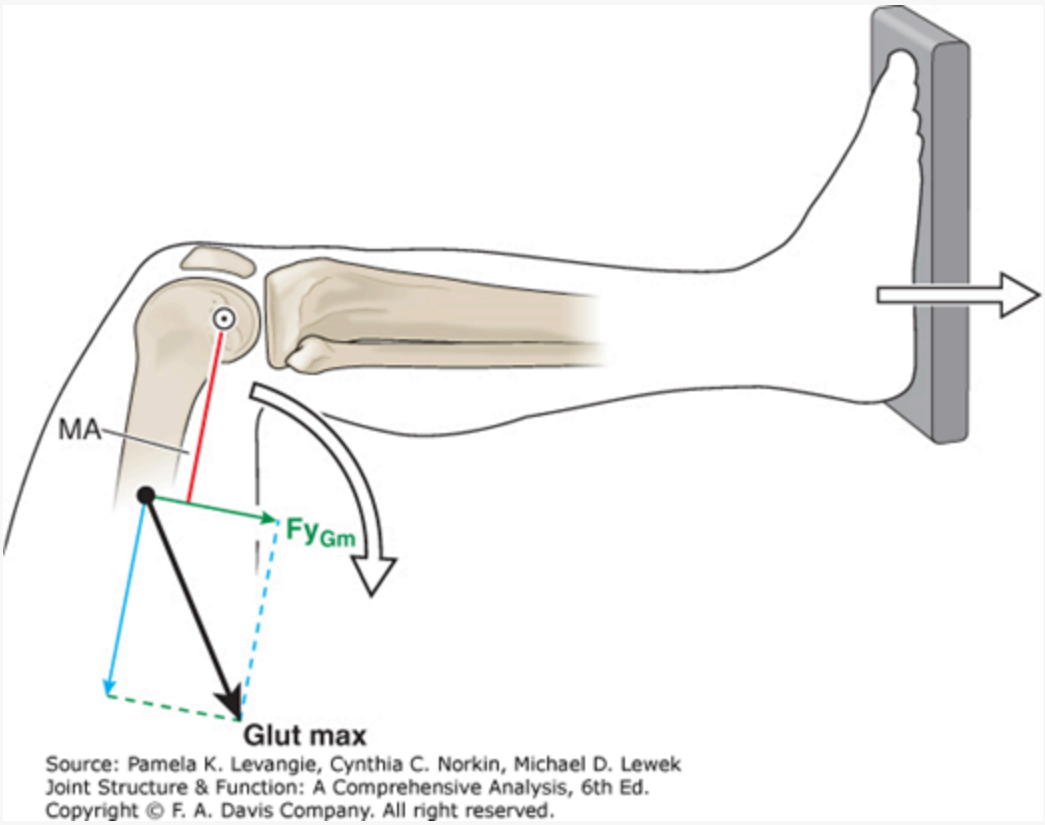
When the leg-foot segment is in a closed chain leg-press exercise, the analysis of forces becomes substantially more complex. In a closed chain, motion of one segment in the chain can be produced by forces applied elsewhere in the chain. Figure 1–45 is an oversimplified representation of the leg-press exercise, showing *only* the force of the gluteus maximus muscle acting on the femur. The femur is in a closed chain with the leg-foot segment because the femur is attached proximally to the pelvis and the leg-foot segment is attached distally to the footplate, with the hip and knee joints in between. In this example, the segment of interest shifts from the leg-foot segment to the femur.

In Figure 1–45, the perpendicular component of the gluteus maximus (Fy_{Gm}) will tend to create clockwise (extension) rotation of the femur. The gluteus maximus is a large muscle that can apply a force of considerable magnitude to the femur, with an Fy component that lies at a considerable distance from the knee joint axis (has a large moment arm). That is, Fy_{Gm}—although not crossing the knee joint—will create a substantial clockwise rotation (torque) of the femur around the knee joint axis (knee extension); this occurs in a closed chain because clockwise rotation of the femur will

cause the femur to contact the leg-foot segment and push the leg-foot segment to the right (the goal of the leg-press exercise). In the leg-press exercise, both the quadriceps force on the leg-foot and the gluteus maximum force on the femur will create a simultaneous knee extension torque. *[Note: Given the limitations on the figure proportions in Figure 1–45, the magnitude of the gluteus maximus vector and its components are substantially underestimated by the length of the vectors in the figure. Furthermore, the actual point of application of the gluteus maximus on the femur is more proximal (further to the left) than is shown because the correct point of application is beyond the limits of the figure. Consequently, the effect of Fy_{Gm} on the femur and knee joint is considerably underestimated in the figure.]*

FIGURE 1-45

The force of the gluteus maximus muscle (Glut max) is applied to the femur in a closed chain. The resulting extension of the hip from component Fy_{Gm} will push the leg-foot segment to the right against the footplate and create an extension torque at the knee joint.



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Because the leg-press exercise involves forces on the femur as well as on the leg-foot segment, a complete analysis would involve (at a minimum) identification of the sources, magnitudes, and effects of all forces (and their components) on both the leg-foot and femur segments—a task beyond the scope or intent of this chapter. However, the complexity of the closed chain leg-press exercise means that John Alexander will be using his gluteus maximus as well as his quadriceps, although the target is the quadriceps. It is impossible for John to isolate the exercise to the quadriceps muscle alone. This is an important concept in designing exercises to strengthen a muscle or muscle group. Although weightbearing exercises are more “functional” (more closely approximating activities of daily living), the combinations of forces that may be used to perform such exercises are large in number and not always predictable (or

controllable). That is, the person may avoid the use of a weak or injured muscle (or muscles) simply by shifting the load to another muscle or muscle group.

The larger moment arm and force-generating capability of the gluteus maximus as compared with the quadriceps would make it likely that the gluteus maximus will contribute more knee extension torque in the leg-press exercise than will the quadriceps. However, we will proceed with an analysis of the leg-press exercise as if the quadriceps were the prime mover because this assumption simplifies the presentation while quite adequately conveying the important principles.

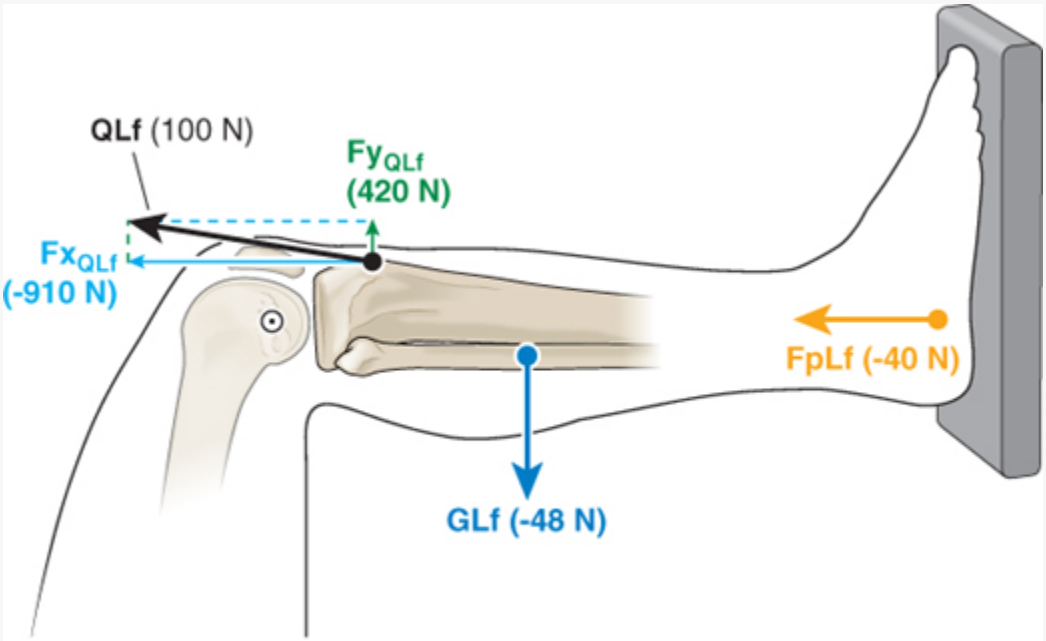
Figure 1–46 shows the leg-foot segment in the leg-press exercise once again. Gravity (GLf) is acting on the leg-foot segment with a magnitude of 48 N. The quadriceps muscle (QLf) is shown generating the same 1,000 N contraction with the same Fx_{QLf} and Fy_{QLf} components shown for the ankle weight exercise (see Fig. 1–44) because the angle of application of the quadriceps remains the same as long as the knee joint is at 90°. The footplate is contacting and creating a force (footplate-on-leg/foot, or FpLf) on the leg-foot segment. Let's assume that there is a 40 N weight on the leg-press machine (the same weight that was used in the ankle weight exercise). The push of the footplate on the leg-foot segment (FpLf) cannot be greater than the amount of weight on the machine, so FpLf is assigned a magnitude of 40 N. Although a friction force might exist between the foot and the footplate (opposing the net upward shear), its magnitude would be minimal and is, therefore, not accounted for. We will identify any additional forces acting on the leg-foot segment by balancing the forces already in place.

Figure 1–46 indicates a net compressive force of -950 N (ΣFx), given the magnitudes of Fx_{QLf} and FpLf as -910 N and -40 N (to the left), respectively. There is a net upward force

of 372 N (ΣFy), given the magnitudes of Fy_{QLf} and GLf, which are 420 N and -48 N, respectively. These net Fx and Fy forces would result in the leg-foot segment moving to the left and upward. The goal, however, is to move the leg-foot segment to the right against the resistance of the footplate without any upward sliding (shear) between the tibial plateau and femoral condyles. Therefore, some additional forces must be applied to the leg-foot segment for John Alexander to get the job done.

FIGURE 1-46

The forces of quadriceps muscle (QLf) and its components, gravity (GLf), and the footplate (FpLf) on the leg-foot segment are shown for the leg-press exercise at 90° of knee flexion. Although a friction force might exist between the foot and the footplate (opposing the net upward shear), its magnitude would be minimal and is, therefore, omitted from the figure.

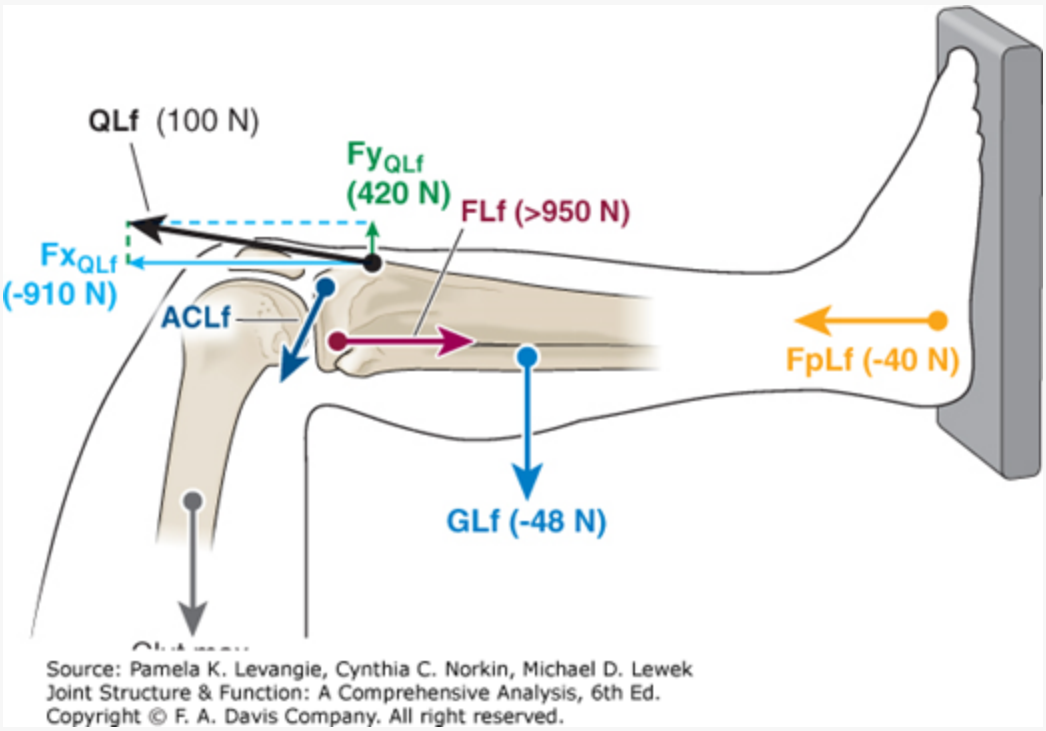


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Given the net Fx component of -950 N, the leg-foot will translate to the left until it hits the femur, at which point there will be a femur-on-leg/foot (FLf) force (**Fig. 1–47**). Although an FLf force of 950 N will be sufficient to prevent the footplate from pushing the leg-foot to the left, the magnitude of FLf must *exceed* 950 N to actually translate (push) the footplate to the right as is appropriate for this exercise. *[Note: The force of the gluteus is shown in Figure 1–47 as a shaded vector to remind the reader that the analysis is more complicated than we are showing.]*

FIGURE 1-47

The contact of the femur with the leg-foot segment (FLf) and its components have been added to the force analysis, along with the force of the anterior cruciate ligament and the joint capsule on the leg-foot segment (ACLf). The force of the gluteus maximus (Glut max) on the femur is shaded and remains only as a reminder of one of the potential sources of FLf.



There is a net upward (Fy) force of 372 N in Figure 1–47. Assuming again that friction between the foot and footplate is of insufficient magnitude to help, we must now identify a structure that will prevent this anterior slide (upward shear) of the tibial plateau on the femoral condyles. The possible source is a capsuloligamentous force—more specifically, tension in the anterior capsule and in the anterior cruciate ligament (ACL). Consequently, a composite capsuloligamentous force of anteriorcruciate/capsule-on-leg/foot (ACLf) has been added to the figure. Although no attempt has been made to resolve ACLf into its components, its Fy component (posterior displacement) should be approximately 588 N. There will also be an Fx component of ACLf, with a magnitude that will depend on the angle of pull but will contribute further to the 950 N joint compression force already created by Fx_{QLf} and FpLf. The joint compression in the leg-press exercise, therefore, will substantially

exceed the 822 N compression estimated for the ankle weight exercise at the same knee joint position and with the same magnitude of quadriceps muscle contraction.

Although we didn't estimate the net torques that would result from all the forces on the leg-foot segment (to confirm that there is a net knee extension torque), the example was sufficient for demonstrating the complexity of force analyses even in very oversimplified situations while also demonstrating that applying the principles of Newton's laws is fairly straightforward if approached methodically.

In spite of very rough estimates of angles of application of forces (and concomitant Fy components and moment arms), our findings with regard to net effects for both the ankle weight and leg press are reasonably in line with findings from at least one group of researchers (although we used a minimum resistance in both the weight boot and leg-press exercises, in comparison with their substantially heavier loads).[7,8] This group of researchers also demonstrated the imprecise nature of biomechanical analyses (even using sophisticated instrumentation) because they found differing results (e.g., magnitudes of joint compression and joint shear forces) using the same data from the same subjects on the basis of different mathematical modeling variables. Their results also contradicted some of the findings in at least one previous study.[9]

Summary

- The goal of this chapter was to use John Alexander to present the biomechanical principles necessary to establish a conceptual framework for looking at the forces and their effects on joints at various points in a joint's ROM (using a predominantly simplistic two-dimensional approach that was based on sequential static rather

than dynamic analyses).

- We identified many (but not all) of the limitations of this conceptual framework when attempting to understand or explain the extremely complex phenomena of human function (laying the foundation for an understanding of dysfunction). We paid particular attention to the interdependence of muscular forces, gravitational forces (or other external forces), and articular constraints.

- The need for articular constraints (joint reaction forces or capsuloligamentous forces) to accomplish joint rotation is too often underappreciated, assessed only when there are problems with these constraints and ignored when they are effective in their function. To avoid this omission, we will next explore the structural composition and properties of the articular constraints (bone, cartilage, capsule/ligament/fascia), as well as those of the muscles and their tendons (Chapters 2 and 3).

- The composition and behavior of various tissues are keys to understanding the stresses (force per unit area) that these tissues may create on other tissues or to which the tissues must characteristically respond.

- With chapter concepts in hand, the reader should understand the basis of both normal and abnormal function at each of the presented joint complexes.

Study Questions

1. Name three types of motion, and provide examples of each.

2. In what plane and around what axis does rotation of the head occur?

3. Is naming the plane of motion considered part of kinetics or part of kinematics? Why?

4. How does a center of rotation (or instantaneous center of rotation) differ from a fixed axis? How does the distinction apply to motion of human joints?

5. What is the definition of a force, what are the units of measure for force, and what are the sources of forces applied to a segment?

6. What are the characteristics that are common to all force vectors? What characteristics are common to all gravitational vectors?

7. What generalizations can be made about the line of gravity (gravity vector) of all stable objects?

8. What happens to the center of mass and line of gravity of a rigid object when the object is moved around in space?

9. What happens to the center of mass and line of gravity of the body when the body segments are rearranged? What happens to the body's center of mass if the right upper extremity is amputated? Provide a simple drawing to support your answer.

10. A student is carrying all of her books for the fall semester courses in her right arm. What does the additional weight do to her center of mass? To her line of gravity? How will her body most likely respond to this change?

11. Describe the typical gait of a child just learning to walk. Why does the child walk this way?

12. Give the name, point of application, magnitude, and direction of the contact force applied to a man weighing 90 kg as he lies on a bed.

13. Are the two forces of an action-reaction pair (reaction forces) part of the same linear force system? Defend your answer.

14. What conditions must exist for friction on an object to have magnitude? What are the conditions under which the magnitude of the force of friction is always greatest?

15. How do a contact force, shear force, and friction force differ?

16. A man who weighs 90 kg (882 N, or ~198 lb) is lying on a bed in cervical traction with a weight of 5 kg (49 N, or ~12 lb), suspended from a horizontal rope. Assuming that the entire body is a single unsegmented object and that the body is in equilibrium, identify all the forces acting on the body (assuming that μ_S for skin on bed is 0.25).

17. A man who weighs 90 kg (882 N, or ~198 lb) is lying on a bed. A nurse standing at the foot of his bed has grasped his ankles and is pulling him down toward the foot of the bed. Treating the man as a single unsegmented object, determine the minimum force that the nurse must apply to the man to *initiate* the movement (assuming that μ_S for skin on bed is 0.25).

18. Assuming now that the leg-foot segment of the man in Question 17 is joined to the rest of his unsegmented body by capsuloligamentous structures at his knee, what must be the magnitude of tension in the knee joint capsuloligamentous structures just as the man begins to be pulled toward the foot of the bed by the nurse? What are the names of the forces causing tension in the capsuloligamentous structures? What are the names of the forces causing distraction of the knee joint segments?

19. How do you graphically determine the net (resultant) effect of two forces applied to the same segment that intersect at an angle to each other? What is this process called?

20. What are the *minimum* requirements to produce rotation of a segment? What are the terms used to refer to the magnitude of rotation?

21. Define moment arm. How does moment arm affect the ability of a force to rotate a segment?

22. If two forces exist on a body segment on the same side of the joint axis but in opposite directions, how does one determine which is the effort force and which is the resistance force?

23. How do anatomic pulleys affect the magnitude and direction of a muscle force (Fms)?

24. Muscle “strength” and muscle “power” are often used interchangeably. What is the difference between these two terms?

25. What factors can cause the net torque on a segment to change?

26. What kind of contraction is a muscle performing when that muscle is the effort force on a rotating segment? What kind of contraction is a muscle performing when it is the resistance force on a rotating segment?

27. What are the benefits of a mechanical advantage greater than one? Do muscles most often work on levers with or without mechanical advantage? What challenges does this present to muscle function?

28. Using the values below, identify the class of the lever, its mechanical advantage, what kind of contraction the muscle is performing, and the point of application of the resultant force of gravity-on-forearm and ball-on-forearm (the hand will be considered part of the forearm). Here Fms = muscle force, MA = moment arm, G = gravity-on-forearm, and B = ball-on-forearm (assume that all forces are applied perpendicular to the forearm lever):

    Fms = 500 N (counterclockwise), MA = 2 cm

    G = 32 N (clockwise), MA = 18 cm

    B = 20 N (clockwise), MA = 28 cm

29. Describe how the perpendicular component of a force and the moment arm of that force are related. When is the moment arm of any force applied to a segment potentially greatest?

30. Describe how you would position the leg-foot segment in space so that gravity

exerts the least torque on the leg-foot segment. How would you position the leg-foot segment so that gravity exerts the greatest torque?

31. A muscle (Fms) is rotating a segment around a joint against the resistance of gravity (G). Identify at least one force other than Fms and G that *must* be applied to the segment for rotation of the segment to occur.

32. If not all of a muscle's force is contributing to rotation, what happens to the "wasted" force?

33. What effects at a joint may a perpendicular force have on a segment other than rotation?

34. When a force is applied at 135° to a segment, what proportion of the force will rotate the segment? What proportion of the force will translate the segment, and will the direction of that force be compressive or distractive?

35. The quadriceps muscle is acting on the free leg-foot segment with a force of 500 N at an angle of 45° (unrealistic, but simplified). Identify the forces (not the magnitudes or locations) that would be required for the leg-foot segment to successfully extend at the knee joint.

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Chapter 2: Complexity of Human Joint Design

Susan M. Sigward

Introduction

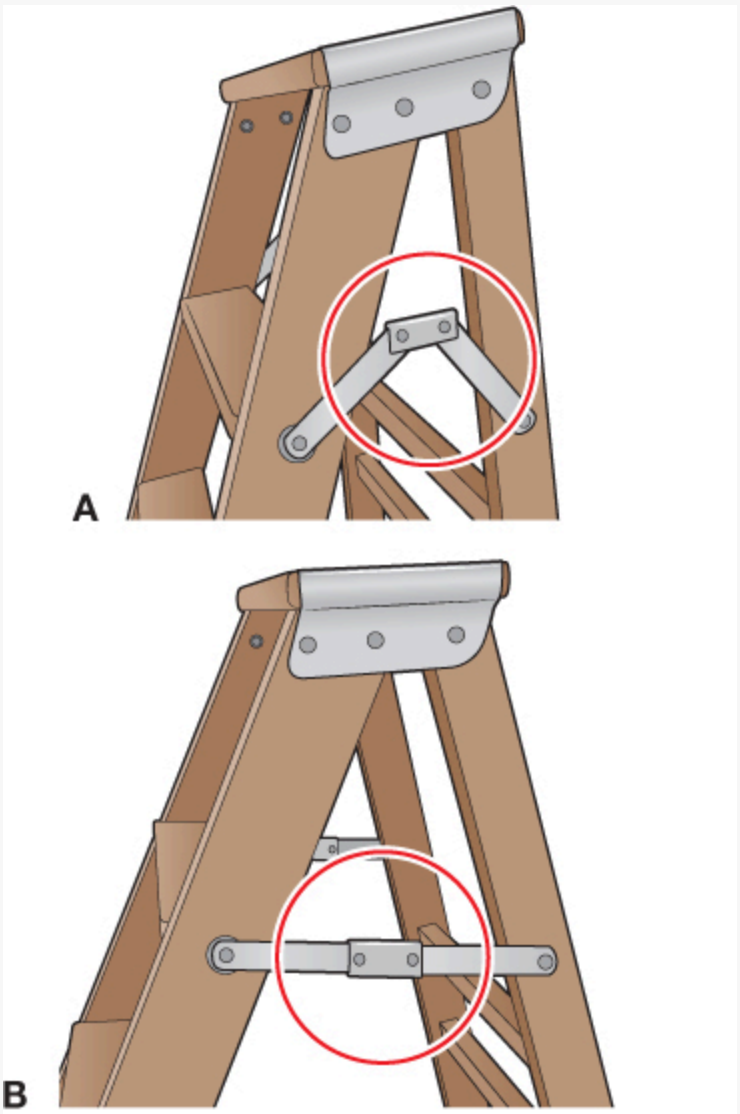
Form Follows Function

The joints of the human body, like those used in the construction of buildings, furniture, and machines, connect different segments together and often allow movement between those segments. The design of the joint reflects these demands. The dictum *form follows function,* coined by the American architect Louis Sullivan and promoted by the Bauhaus school of design of post–World War I Germany,[1] suggests that the appearance (form) of an object or building is governed by its function. Although a wooden stool and a chair with a padded seat, arms, and back are both for sitting, the differences in form hint at the intended functions, respectively, of short-term versus longer-term support. The function of a step stool is to support weight so the elements are joined to serve stability and can be made of a sturdy material such as a heavy wood. However, if the step stool needs to be portable and collapsible while still stable when opened, the design and material composition will become much more complex. An aluminum stepladder is lighter and has a metal strut with a locking device as well as hinges at the intersection of the two sides of the ladder. The ladder can be folded when the brace is unlocked; when the brace is locked, the ladder is still stable (Fig. 2–1). If the ladder is opened but the brace is not locked, stability is compromised by excessive mobility. Human joints that must meet both stability and mobility demands are more complex than joints that favor stability. The more complex the demands on and design

of a human joint, the more stresses the joint must withstand and the more at risk the joint is for failure.

FIGURE 2-1

Folding stepladder joint. **A.** The sides of the stepladder are free to move (mobile) when the brace is unlocked. **B.** The sides of the stepladder are prevented from moving when the brace is engaged, providing necessary stability.



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Form includes an object's structure as well as its composition, and a change in either structure or materials (or both) can influence function. The prosthetic foot shown in Figure 2–2 reflects the relationship between physical form (structure and composition) and function. The design of the curved foot allows the structure to bend as body weight is applied whereas the heel extension provides stable support. The bending of the foot can both absorb shock and provide an elastic recoil that contributes to the subsequent

propulsion. Alterations in the material (usually a carbon fiber composite) affect the amount of elastic recoil. A person with a lower extremity amputation can order limbs made of different materials, depending on their functional demands. Springier materials that are harder to stretch and have more recoil are used for activities with large loads and high speeds (e.g., running, jumping), whereas materials with less elastic recoil are used for walking. A track athlete with lower extremity amputations can use feet that are less stable (no heel extension) but have greater recoil for increased mobility (speed) (Fig. 2–3).[2]

FIGURE 2-2

The design of this prosthetic foot facilitates gait through its design and composition. The curved blade bends during loading and then assists with propulsion as the blade recoils. The heel piece increases stability. *Photo courtesy of Freedom Innovations.*



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FIGURE 2-3

The form of a prosthetic foot changes when the goal is to maximize mobility (speed), even at the expense of stability. With running blades, the heel can be eliminated and the elastic recoil of the foot is increased for stronger propulsion.

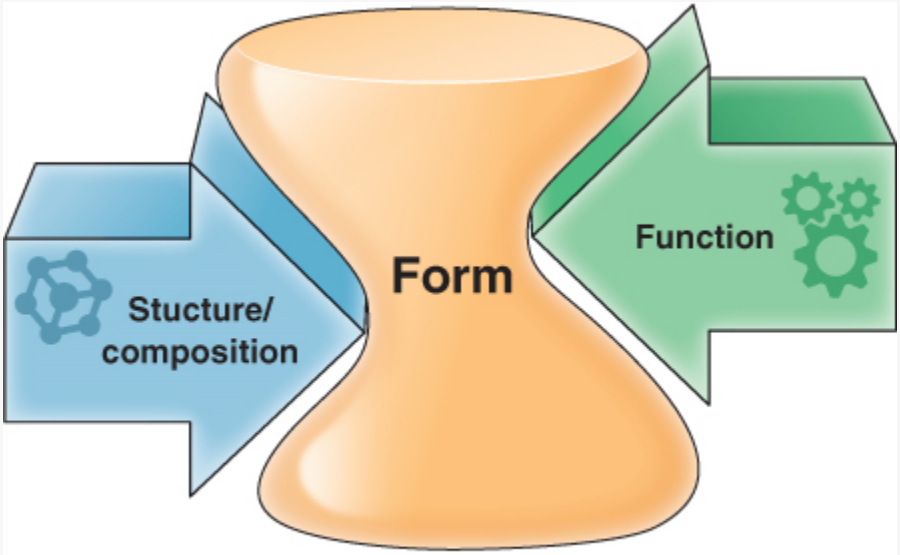


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In man-made structures, form for the most part is relatively static. Although the stepladder can be collapsed, the shapes of all the component parts remain unchanged. Structure, function, and form in humans are dynamic and interactive. Cellular composition and the structure of a tissue will affect the form that a tissue will take which, in turn, is affected by the type and amount of stresses applied to a tissue; stresses are determined by the functional demands placed on a tissue. As function is altered, the stresses on the tissue will change and may signal cells and extracellular elements to alter composition and shape and, therefore, the form that the tissue takes (Fig. 2–4).

FIGURE 2-4

The form of human tissues is determined both by the overall composition and structure of the connective tissues and by functional use. The size of the tissue and its composition will determine the types of loads the tissue can bear. However, tissues may adapt to altered functional demands in response to shifting stresses that lead to cellular and extracellular events affecting the form of the tissue.



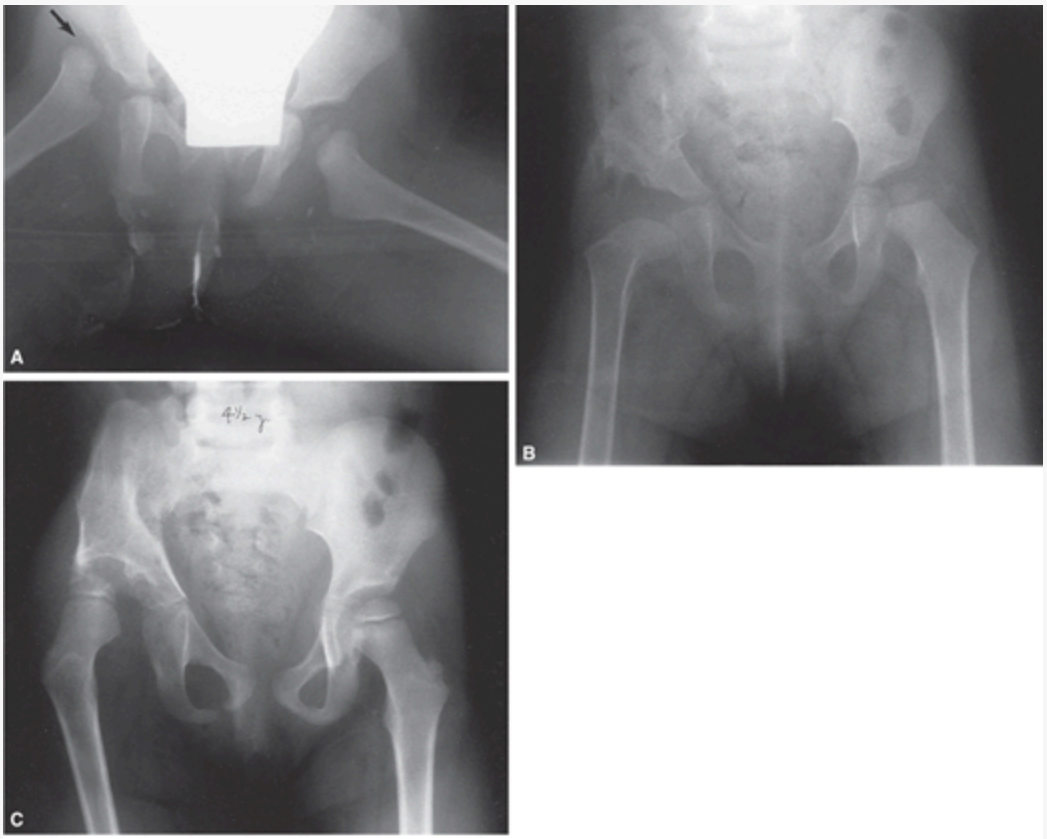
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Changes in the material properties of tissues and shapes of human joints occur as we develop and can result from demands imposed during typical and atypical movement. This relationship is illustrated in the case of developmental hip dysplasia. The structural elements of the hip joint develop before birth. The mature shape of the head of the femur and the acetabulum is determined by the functional interaction between these two structures over time. Decreased contact between the acetabulum and femoral head that can occur during fetal development may lead to a shallow acetabulum or less

spherical femoral head (alteration in form), as well as to deficiencies in the acetabular cartilage (alteration in composition).[3,4] Changing the stresses across the hip joint in the infant by creating a deeper acetabulum or repositioning the femoral head in the acetabulum (e.g., through surgical intervention) generally will lead to adaptive changes in the femoractetabular joint structure and composition;[5] the changes will lead to improvements in both the mobility and stability of the hip joint (Fig. 2–5). Julius Wolff described the adaptation of bone to changes in stresses in the late 1800s (now known as Wolff's law[6]). However, the interaction between form and function isn't unique to bone but is common to all living tissues.

FIGURE 2-5

Developmental dysplasia of the hip in a toddler. **A.** Arrow points to the congenital deformity of the right hip joint seen pre-operatively in this 16-month-old boy. For comparison, note the configuration of the child's normal left hip joint in the same film (much of the femoral head and acetabulum are cartilage so are transparent on radiograph). **B.** A film six months after surgical creation of an acetabular fossa (posterior iliac osteotomy) shows improved location of the femoral head relative to the new acetabulum. **C.** At 4 ½-years-old, the femoral head is sufficiently seated in the now rounded acetabulum to support all ambulatory activities.



All the elements of human joints, including bones, cartilage, ligaments, muscles, and tendons, adapt their appearance and composition in response to functional demands. These demands can change with immobilization, inactivity, or training. Knowing the functional demands and the tissues' responses to those demands, therapists can modify the functional demands (stresses) on joint structures during rehabilitation to optimize targeted tissue structure and function.

Foundational Concepts 2–1

Interaction of Joint Function, Structure/Composition, and Form

- The form that a joint takes is dependent upon the function that the joint is serving as well as its structure (design) and composition (materials).

- Most human joints serve a dual purpose of stability and mobility although one purpose may predominate.
- As demands for both mobility and stability increase, the design and composition of the joint becomes more complex.
- The elements of structure/composition, function, and form in human tissues and joints are dynamic and interactive; changes in one element can affect one or both of the other elements.

The Complexity of Human Joint Design

An appreciation of the complexities of human joint design may be gained by considering the nature of the bony components and the functions that the joints must serve. The human skeleton has about 200 bones that are connected by joints. Bones vary in size from the pea-sized distal phalanx of the little toe to the long femur of the thigh. The shape of the bones varies from round to flat, and the contours of the ends of the bones vary from convex to concave. The task of designing joints that are capable of working together to provide both mobility and stability for the total structure represents an engineering problem of considerable magnitude.

Joint designs in the human body vary from simple to complex. Simple human joints usually have stability as a primary function; the more complex joints usually have mobility as a primary function but cannot completely sacrifice stability, ideally achieving what is commonly referred to as *dynamic stability.* Stability joints may be contoured either to fit snuggly into each other or to lie flat against each other with relatively little motion between. As joints require increasing amounts of mobility without fully sacrificing stability, additional structural elements, such as joint capsules, ligaments, and accessory joint structures, help make the compromise between the

mobility and stability demands. Classifications of human joints are named based on structure of the joint or amount of movement. Structurally, joints can be classified as **fibrous, cartilaginous,** or **synovial,** with each referring to the connective tissue that binds the bony elements together or the presence or absence of a synovial joint cavity. The joint categories of **synarthrosis, amphiarthrosis,** and **diarthrosis** refer to the degree of movement a joint affords. Synarthrosis joints are those that have little or no movement, amphiarthrosis joints allow minimal to moderate movement, and diarthrosis joints are freely moveable joints. The two different classification schemes actually have considerable overlap because joints with little movement (synarthroses) typically are united by fibrous tissue, those with moderate movement are typically united directly by **hyaline** (**articular**) **cartilage** or **fibrocartilage,** and those that have a lot of movement (diarthrodial) are united by a synovial joint structure. Consequently, we will identify joint classifications using the simpler structural nomenclature where both structure and function (movement) for each classification are described.

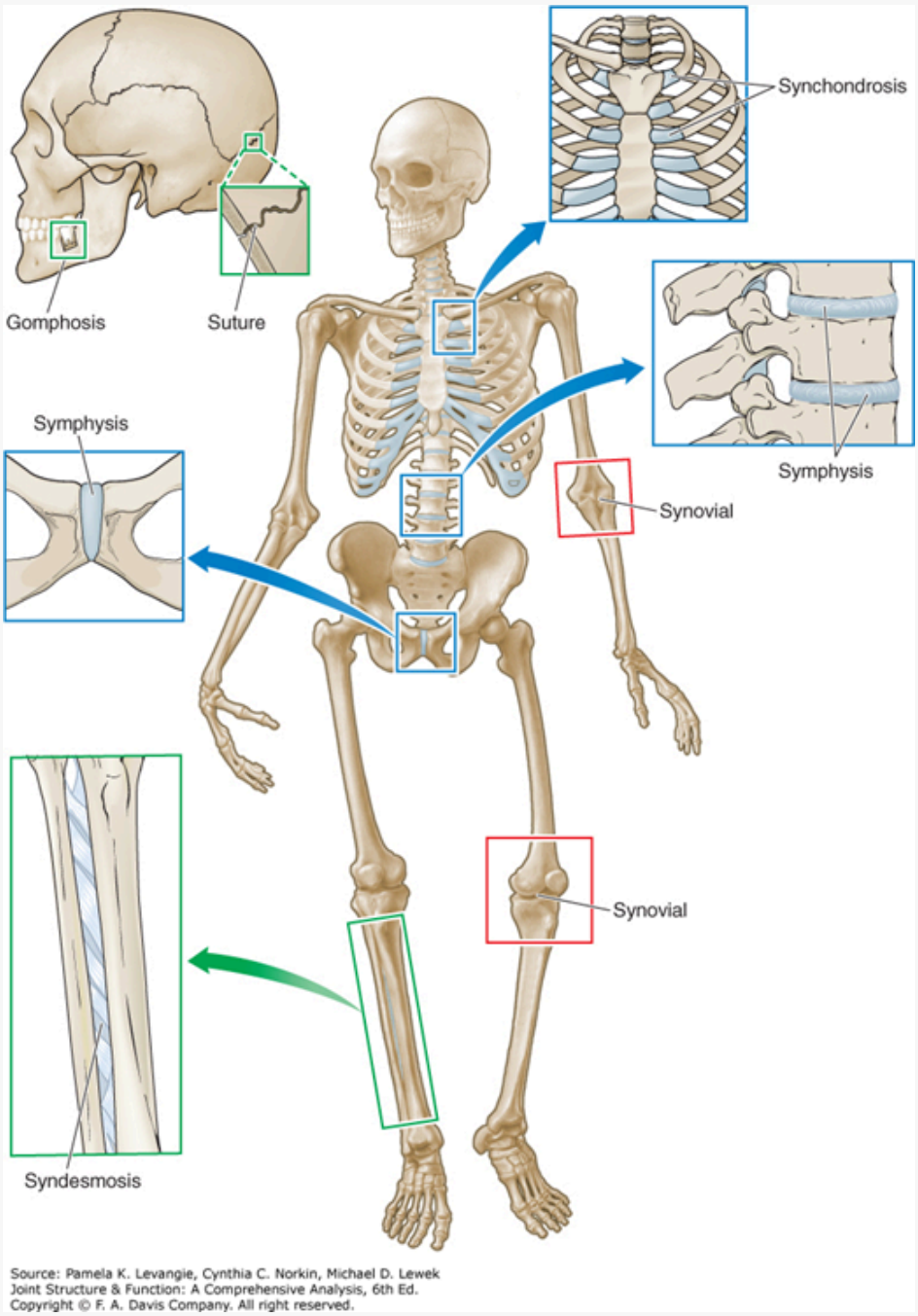
Joint Classification

Examples of fibrous, cartilaginous and synovial joints are illustrated in Figure 2–6.

FIGURE 2-6

Fibrous joints allow for little or no movement between the bones; examples (outlined in green) include sutures (e.g., between the frontal and parietal bones of the skull), gomphoses (e.g., between each tooth and the mandible), and syndesmoses (e.g., between the distal tibia and fibula). Cartilaginous joints (outlined in blue) directly unite one

bony surface to another via hyaline or fibrocartilage; examples include symphyses (e.g., between the vertebral bodies and between the pubic bones) and synchondroses (between the first 7 ribs and the sternum). In synovial joints (outlined in red) such as the knee and elbow joints, the bones are indirectly united by a fibrous capsule lined with synovium.



Fibrous Joints

The bony components of a fibrous joint are joined together by fibrous interosseous connective tissue that directly unites one bone to another. This creates a solid connective tissue-bone interface that allows for little or no movement, thus classifying

them functionally as synarthroses. Three different types of fibrous joints are found in the human body: sutures (e.g., between the frontal and parietal bones of the skull), gomphoses (e.g., between each tooth and the mandible), and syndesmoses (e.g., between the distal tibia and fibula; see Fig. 2–6).

A suture joint is one in which two bony components are united by a collagenous sutural ligament or membrane. The ends of the bony components are shaped so that the edges interlock or overlap one another. This type of joint is found only in the skull early in life and allows a small amount of movement. Fusion of the two opposing bones in suture joints occurs later in life and leads to the formation of a bony union.

A gomphosis joint is one in which the surfaces of bony components join each other like a peg in a hole and are connected by fibrous connective tissue. The only gomphosis joint that exists in the human body is the joint between a tooth and either the mandible or maxilla.

A syndesmosis joint is a type of fibrous joint in which two bony components are joined directly by an interosseous ligament, a fibrous cord, or an aponeurotic membrane. These joints usually allow a small amount of motion and can be seen between the shafts of the radius and ulna and the shafts of the tibia and fibula.

Cartilaginous Joints

The materials connecting the bony components in cartilaginous joints are fibrocartilage or hyaline cartilage. These materials directly unite one bony surface to another, creating a bone-cartilage-bone interface that allows for some movement between the bones. The two types of cartilaginous joints are symphyses and synchondroses (see Fig. 2–6).

In symphysis joints, the two bony components are covered with a thin lamina of hyaline cartilage and directly joined by fibrocartilage in the form of discs or pads. Examples of symphysis joints include the intervertebral joints between the bodies of the vertebrae, the joint between the manubrium and the body of the sternum, and the symphysis pubis in the pelvis.

A synchondrosis joint is one in which hyaline cartilage connects two bony components. An example would be the articulation of the first seven ribs with the sternum. Synchondroses also refer to the hyaline cartilage bond between two ossifying centers of bone in a developing skeleton. In Figure 2–5C, the cartilaginous ossification centers appear as spaces between the femoral head and femoral neck. These joints function to permit bone growth while also providing stability and allowing a very small amount of mobility. Epiphyseal growth plates near the ends of long bones as well as in the pelvis are also synchondrosis unions. When bone growth is complete, these joints ossify and convert to bony unions.



Foundational Concepts 2–2

Fibrous and Cartilaginous Joints

- Fibrous joints include sutures, gomphoses, and syndesmoses.
- The two types of cartilaginous joints are symphyses and synchondroses.
- Bones directly connected by fibrous or cartilaginous connective tissue afford less movement and greater stability than do synovial joints, with cartilaginous joints typically being somewhat more moveable than fibrous joints.

Synovial Joints

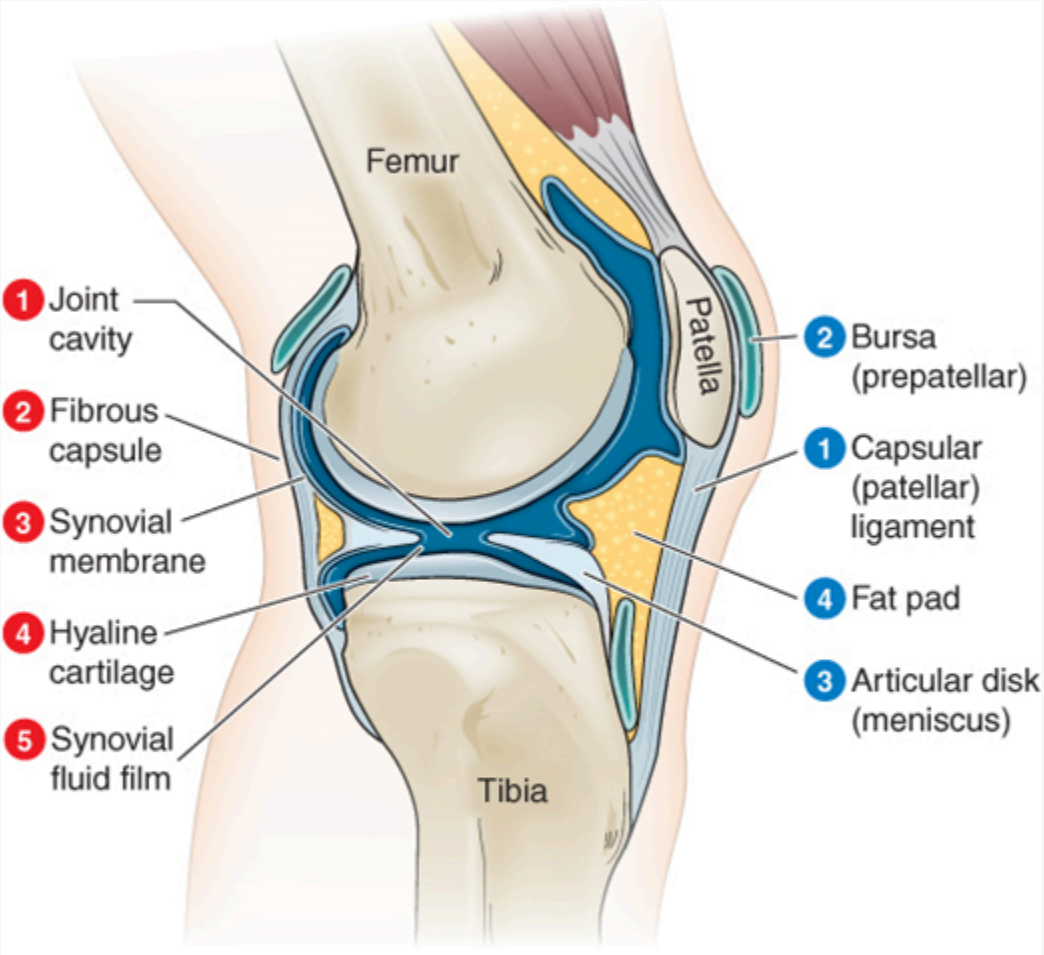
The joint construction in synovial joints differs from that found in fibrous and cartilaginous joints. In synovial joints, the ends of the bony components are free to

move in relation to one another because no connective tissue directly unites the adjacent bony surfaces. The bony components are indirectly connected to one another by means of a fibrous synovium-lined joint capsule that encloses the joint. Examples of synovial joints include the elbow and knee joints (see Fig. 2–6). Synovial joints are constructed in a similar manner (Fig. 2–7) and have the following common features numbered in red: (1) a **joint cavity** that is enclosed by (2) **a fibrous joint capsule** that surrounds the joint; (3) a **synovial membrane** that lines the inner surface of the fibrous capsule; (4) hyaline cartilage that covers the articular surfaces of the contiguous bones; and (5) synovial fluid that forms a film over the joint surfaces within the capsule.[7] Synovial joints may also include associated structures such as capsular ligaments that reinforce the capsule, bursae to reduce friction between structures, fibrocartilaginous accessory structures (discs, menisci, plates or labrums) that typically contribute to joint stability while minimizing loss of mobility, and fat pads where cushioning is needed. All synovial joints are crossed by muscles or their associated tendons. The capsule and other structures associated with the joint typically keep joint surfaces aligned and can actually guide motion. Stabilization by the passive joint structures and active stabilization by muscles and their associated tendons will limit joint distraction and excessive translation of the contiguous bones.[8]

FIGURE 2-7

Elements of a synovial joint such as the knee joint will always include (listed in red): (1) a joint cavity that is enclosed by (2) a fibrous joint capsule that surrounds the joint; (3) a synovial membrane that lines the inner surface of the fibrous capsule; (4) hyaline cartilage that

covers the articular surfaces of the contiguous bones; and (5) synovial fluid that forms a film over the joint surfaces within the capsule. A synovial joint may also include associated structures (as the knee does) such as (listed in blue): (1) capsular ligaments, (2) bursae, (3) accessory structures such as menisci, discs, plates, or labra, and (4) a fat pad.



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Patient Application 2–1

Sione Tanielu is a 16-year-old football player who hyper-dorsiflexed his ankle when another player tackled him from behind. He tore the ligaments supporting his distal tibiofibular joint. This joint is structurally classified as a fibrous joint (syndesmosis) and is supported by anterior and posterior tibiofibular ligaments as well as the interosseous membrane. Although the structure allows relatively small but important movements between the distal tibia and fibula, the stability of this joint is critical because the tibia and fibula together form the proximal segment of the synovial (diarthrodial) talocrural (ankle) joint. Disruption (hypermobility) of Sione's distal tibiofibular joint will greatly affect the stability of the talocrural joint, limiting if not completely preventing effective weightbearing.

JOINT CAPSULE

The synovial joint cavity is an area between two bones that is enclosed by a joint capsule that consists of two layers: an outer fibrous layer and an inner synovial layer (see Fig. 2–7). The outer fibrous layer inserts circumferentially around the contiguous ends of the articulating bones. Its primary functions are to provide an enclosure for the joint cavity and enhance joint stability through its passive restraints.[9] The inner synovial layer lines the joint cavity, providing lubrication and nutrition to the articular cartilage.[10] Both layers of the capsule house sensory nerve fibers that provide important information to the central nervous system about, for example, position sense and the need for active muscular support of the joint.

The fibrous layer of the joint capsule is composed of dense irregular connective tissue with varying thickness and composition from joint to joint, thus affording different amounts of passive restraint. For example, the hip joint capsule is thick and dense, providing stability to this weightbearing joint that undergoes considerable stress. Other joint capsules such as that of the glenohumeral joint are thin and loose, providing less stability while allowing increased mobility. The thickness, fiber orientation, and

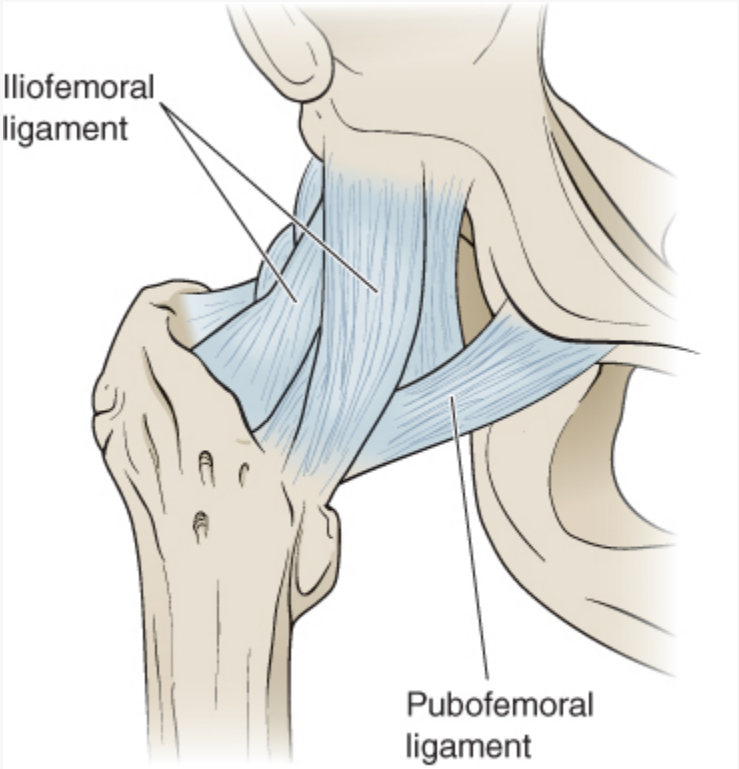
composition of the fibrous capsule depend on the stresses placed on it, illustrating the dynamic nature of the joint capsule. In patients with shoulder instability (a chronically subluxing glenohumeral joint), the joint capsule is subjected to repeated tensile stresses that alter tissue composition; capsular collagen fibrils and elastin fibers are larger and denser when compared with typical capsules. These adaptations may increase capsular strength and resistance to stretching deformations.[8] In contrast, portions of an articular capsule that are subjected to repeated compression forces may show development of fibrocartilaginous tissue.[9,11]

Capsular ligaments are localized thickenings of the capsule with distinct fiber orientation that distinguish it from the capsule. These passive tissues provide additional joint stability or restraint to movement based on their orientation, with that orientation influenced by typical stresses at that joint. For example, the iliofemoral ligament of the hip reinforces the anterior joint capsule as it runs from the anterior inferior iliac spine and fans out to attach to the intertrochanteric line of the femur (**Fig. 2–8**). The ligament forms an inverted Y shape as the ligament fans into superior and inferior bands.[12] This reinforcement of the anterior capsule of the hip provides needed extra restraint to hip extension that enhances passive joint stability and reduces the need for active hip flexor activity to check strong extension torques that occur at the hip.

FIGURE 2-8

The iliofemoral ligament reinforces the anterior hip joint capsule, providing added passive restraint to strong hip joint extension

torques.



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The synovial layer of the capsule is the inner lining of the fibrous joint capsule. The synovium does not cover the articular cartilage or menisci/disks (if present), but will line other intra-articular structures that may be present (e.g., the cruciate ligaments within the knee joint). The synovial layer is divided into two layers: the intima and the subintima. The **intima** consists of a thin layer of surface cells that line the joint space.[10] These cells are responsible for the removal of debris from the joint cavity, structural repair of the synovium, response to antigens and tissue destruction, and synthesis of hyaluronic acid (a component of synovial fluid).[10] The intima is richly endowed with capillary vessels, lymphatic vessels, and nerve fibers. The **subintima** lies outside

(superficial to) the intima and is a loose network of highly vascularized fibrous connective tissue that allows the membrane to move freely. The subintima supports the intima and merges on its external surface with the fibrous capsule. The blood vessels in the subintima transport oxygen, nutrients, and immunologic cells to the joint.

Branches of nearby sensory nerves and branches of nerves from muscles near the joint penetrate the joint capsule through the fibrous layer.[9] Large-diameter sensory efferent nerves and thinly myelinated nerves are present in the fibrous capsule; non-myelinated C-type fibers are found in the synovium.[13] The joint receptors found in the fibrous joint capsule are sensitive to stretching or compression of the capsule, as well as to any increase in internal pressure due to increased production of synovial fluid (joint swelling). Using the knee joint and its capsule as an example, most of the joint receptors are located in the subintima layer of the capsule close to the insertions of the anterior cruciate ligament (ACL). Mechanoreceptors (predominantly Ruffini receptors) in the subintima capsule and ACL respond primarily to the stretch (e.g., with terminal knee extension). Pacini receptors are reported less frequently and are thought to be activated by compression. Free nerve endings are more numerous than mechanoreceptors and function as nociceptors that react to inflammation and pain stimuli (Table 2–1).[14,15] Together, sensory stimuli, among other functions, contribute to active joint stability by regulating resting muscle tension and initiating reflexive changes in muscular contractions.[13]

Table 2-1

Joint Receptors

SYNOVIAL FLUID

Synovial fluid, a thin film that covers the inner surface of the synovial membrane of the joint capsule and the articular cartilage, helps to keep the joint surfaces lubricated and reduces friction.[16,17] Synovial fluid also provides nourishment for the hyaline cartilage that covers articular surfaces as the fluid moves in and out of the cartilage with joint compression and release.

The composition of synovial fluid is similar to that of blood plasma, except that synovial fluid also contains hyaluronate (hyaluronic acid) and a glycoprotein called lubricin.[18] The hyaluronate component of synovial fluid is responsible for the viscosity of the fluid and is essential for joint lubrication. Hyaluronate reduces friction between the synovial folds of the capsule and the articular surfaces.[10] Lubricin is the component of synovial fluid thought to be responsible for cartilage-on-cartilage lubrication.[1,16,19,20]

The minimal wear shown by normal articular cartilage, despite varied loads, is the result of the structure of the cartilage matrix and the presence of lubricating fluid.[17,19,20] Although a number of models have been proposed to explain how synovial joints are lubricated under varying load conditions (e.g., “boundary lubrication” and “fluid-film lubrication”), the general consensus is that no single model is adequate to explain human joint lubrication.[16] It is recognized, however, that changes in the concentration of hyaluronate or lubricin in the synovial fluid will affect the overall lubrication and the

amount of friction that is present in the joint. Many experiments have confirmed that articular coefficients of friction in synovial joints are far lower than those created by currently available manufactured lubricants.[17] The lower the coefficient of friction, the lower the resistance to movement. There is some evidence to support the use of injections of hyaluronate and lubricin to alleviate symptoms of hip or knee osteoarthritis.[21,22]

Normal synovial fluid appears as a clear, pale yellow, viscous fluid present in small amounts at all synovial joints.[20] There is a direct exchange between the vasculature of the synovial layer and synovial fluid in the intracapsular space, where nutrients are supplied and waste products can be taken away from the joint space by diffusion. When a joint is injured or diseased, the volume of synovial fluid increases in the joint and is related to reports of pain.[23] The synovial fluid, like all viscous substances, resists shear loads.[16]

Changes in the synovial layers of the joint capsule are seen in inflammatory arthritic condition, such as rheumatoid arthritis, and are associated with articular cartilage degeneration. Increased levels of inflammatory cells hinder the lubricating features of synovial fluid. The synovial layer of the capsule hypertrophies and develops *pannus,* invasive granulation tissue that further destroys the joint articular cartilage. Rheumatic diseases are systemic and will affect more than one joint in the body. As a result they ultimately lead to decreased mobility and functional capacity.[24]

HYALINE CARTILAGE

Hyaline cartilage, from the Greek word *hyalos,* meaning “glass,” forms a relatively thin (1 to 7 mm) covering on the ends of the bones in synovial joints. It provides a smooth,

resilient, low-friction surface and is important for bearing and distributing forces over a person's lifetime. Detailed discussion of its composition and function will be discussed later in the section on Specific Connective Tissue Composition in this chapter. Hyaline cartilage is commonly referred to as articular cartilage.

Patient Application 2–2

Enid Brown is a 50-year-old female who was recently diagnosed with rheumatoid arthritis. She had been experiencing episodes of stiffness and soreness in her hands, right knee, and neck over the last year. Recently, she realized that the joints in her fingers were red and swollen in both hands. She had pain and crepitus when she tied to grab anything, especially when she tried to twist the lids off jars. Her primary care physician referred her to a rheumatologist who diagnosed rheumatoid arthritis. Rheumatoid arthritis is an autoimmune disease that is characterized by inflammation, swelling, and hypertrophy of the synovial lining of synovial joints, which leads to the symptoms described by Enid. The disease is progressive and the inflammatory response will ultimately affect the health of the articular cartilage and surrounding connective tissues, such as joint capsules and ligaments.

Foundational Concepts 2–3

Synovial Joint Capsule

The synovial joint capsule:

- Provides an enclosure for the joint cavity
- Contributes to passive joint constraints and stability via the fibrous layer
- Provides lubrication and nutrition to intra-articular joint structures via the synovial layer
- Has sensory receptors in both the fibrous and synovial layers that provide information regarding joint position, movement, and pain

SYNOVIAL JOINT ASSOCIATED STRUCTURES

Fibrocartilaginous menisci, labrums, plates, and discs help to increase joint congruence

("fit"), absorb compressive joint forces, increase stability, and facilitate motion. For example, the wedge-shaped fibrocartilage medial and lateral menisci of the knee deepen the relatively flat tibial plateau, providing increased congruence of the tibia with the large convex femoral condyles. That greater contact area not only increases stability of the joint but also allows for distribution of joint compressive forces across a larger surface area, thus decreasing joint stress. In fact, it has been estimated that the menisci of the knee bear 45% to 70% of the load across the knee depending on joint angle.[25] In another example, fibrocartilage labra deepen and extend the articular concave of the articular surface to which they attach. The acetabular labrum attaches around the entire periphery of the pelvic acetabulum, enhancing joint stability both by deepening the acetabulum and by creating negative pressure that holds the femoral head in the acetabulum.[26] A fibrocartilage articular disc may extend all the way across a joint and actually divide the joint into two separate cavities. The articular disc of the sternoclavicular joint transects the joint and is attached in such a way that it increases joint stability while serving as a pivot point for motions. In each instance the fibrocartilage accessory joint structure provides increased joint stability with little or no sacrifice of mobility.

Ligaments play an important role in keeping joint surfaces aligned and in both guiding and restraining motion. Excessive separation or translation of joint surfaces is limited by passive tension in ligaments, as well as by the fibrous joint capsule and muscle tendons that cross the joint. As described previously, ligaments may blend with the joint capsule and appear as thickenings in the capsule (e.g., the iliofemoral ligament, the glenohumeral ligament). Other ligaments are distinct from the capsule and may consist of dense white bands or cords of connective tissue (e.g., anterior cruciate ligament,

lateral collateral ligament of the knee). Ligaments are typically named descriptively according to their location, shape, bony attachments, and relationship to one another.[6] The anterior longitudinal ligament of the spine derives its name from both location (anterior) and shape (longitudinal). The medial and lateral collateral ligaments of the elbow and knee joints are examples of ligaments named for location. Ligaments such as the coracohumeral ligament that connects the coracoid process of the scapula to the humerus, and the radioulnar ligaments that connect the radius to the ulna are named for their bony attachments. The deltoid ligament at the ankle joint is named for its shape. Occasionally, ligaments are named for the individuals who first identified them (e.g., the Y-ligament of Bigelow—an alternate name for the iliofemoral ligament).

Although the primary task of a tendon is to transmit forces developed by muscles to their bony attachments to create or limit rotation of a joint, active and passive tendon forces may stabilize the joint by creating joint compression or by restricting excessive or unwanted translatory movement between bony segments. Shapes and configurations of tendons vary throughout the body.[27–29] They can be round and rope-like structures such as the biceps brachii tendon at the elbow or the tibialis posterior tendon as it wraps around the medial malleolus. Tendons can also be flat like the tendons of the supraspinatus, latissimus dorsi, and pectoralis major muscles. Very broad flattened tendons are often referred to as **aponeuroses.** Tendons can also lie *within* a muscle to allow short muscle fibers to connect to a central tendon as is seen in the soleus and deltoid muscles.[27]

Bursae are flat dense irregular connective tissue sacs lined with a synovial membrane in which the inner sides of the sacs are separated by a fluid film. Bursae are found where

structures in tight approximation to each other might move on each other, such as between tendon and bone, bone and skin, muscle and bone, or ligament and bone. Bursae located between the skin and bone (such as the prepatellar bursa at the knee) are called subcutaneous bursae.[6] Subtendinous bursae lie between tendon and bone, and submuscular bursae lie between muscle and bone.



Foundational Concepts 2–4

Synovial Joint Stability

In general, synovial joints offer the greatest amount of movement between two contiguous bones as compared with fibrous or cartilaginous joints. However, that increase in mobility can come at the expense of stability. There are several features that can be associated with synovial joints to enhance stability without seriously impacting mobility:

- Ligaments are passive stabilizers of the joint that can blend in with the capsule or appear as individual structures.
- Fibrocartilage menisci, labrums, plates, and discs provide passive joint stability by deepening articular surfaces and increasing joint congruence.

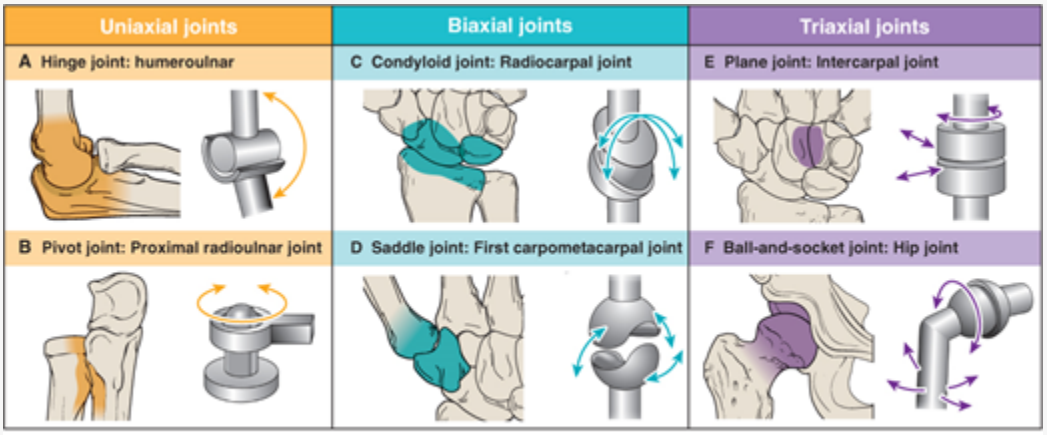
CLASSIFICATION OF SYNOVIAL JOINTS

Synovial joints can be divided further into subclassifications based on the shape and configuration of the ends of the bony components.[30] These classifications describe simple geometric surfaces and include **plane, ball-and-socket, condyloid, saddle, hinge,** and **pivot** (Fig. 2–9). It is important to keep in mind that joint surfaces and geometry are more complex and that these geometric classifications provide a simplified description of the orientation and function of the articulating surfaces.[31] Synovial joints within these classifications also can be categorized based on the number of axes about which "gross visible" rotary motion at the joint occurs: **uniaxial, biaxial,** and **triaxial.**[32–34] The actual direction and magnitudes of motion at a joint can be dictated not only by joint

geometry, but also by surrounding articular structures.

FIGURE 2-9

Synovial joints can be subclassified both by joint geometry and by the number of axes around which visible motion of the bony segments can take place at the joint. A sample of each type of joint is accompanied by a mechanical representation of its movement.



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Uniaxial (also known as **uniplanar**) joints are those with visible rotation of the bony components in one plane around a single axis (one degree of freedom). Hinge and pivot joints are uniaxial. A hinge joint mechanically resembles a door hinge where a cylindrical component fits into a concave component. The humeroulnar articulation of the elbow joint is an example of a hinge joint, formed by the trochlea on the distal end of the humerus and the trochlear notch on the proximal end of the ulna (see Fig. 2–9A). A pivot joint mechanically has a cylindrical component that rotates within a ring-shaped component. The proximal radioulnar joint is an example, with the annular ligament and radial notch on the ulna forming a ring within which the head of the

radius rotates (see Fig. 2–9B).

Biaxial (also known as **biplanar**) joints are those with visible rotation of the bony components in two planes around two axes (two degrees of freedom). Condyloid and saddle joints are biaxial. The term **ellipsoid** is often used interchangeably with condyloid. The radiocarpal joint is an example of a condyloid joint—formed by convexities of the scaphoid, lunate, and scapholunate ligament in both the sagittal and frontal planes with corresponding concavities on the distal radius (see Fig. 2–9C). A saddle joint has a complex configuration where both segments are convex in one plane and concave in a perpendicular plane. This shape is similar to that of a horse saddle where an anterior to posterior concavity runs perpendicular to a medial to lateral convexity. The carpometacarpal joint of the thumb is an example of a saddle joint, formed by the trapezium's articulation with the base of the first metacarpal (see Fig. 2–9D). The convexity of the base of the first metacarpal moves on the concavity of the trapezium in flexion/extension, whereas the concavity of the metacarpal moves on the convexity of the trapezium in abduction/adduction.

Triaxial or **multiaxial** joints are those in which the bony components are free to rotate in three planes around three axes (three degrees of freedom). The two types of joints in this category are plane joints and ball-and-socket joints. Plane joints are articulations between two relatively flat surfaces. The opposing surfaces can have a variety of configurations with slight but mostly inconsequential curvatures that permit both rotation and sliding between two or more bones. Examples of these joints can be seen in intercarpal articulations such as that between the capitate and hamate (see Fig. 2–9E). Ball-and-socket joints are formed by a ball-like convex surface that fits into a

concave socket. An example of a ball-and-socket joint is the hip (femoroacetabular) joint, formed by the concave head of the femur and convex socket (see Fig. 2–9F). The rotary motions of the flexion/extension occur in the sagittal plane around a coronal axis; abduction/adduction occurs in the frontal plane around an anteroposterior axis and medial/lateral rotation occurs in the transverse plane around a longitudinal axis.

Joint Motion

In the descriptions of the subclassifications of synovial joints, joints were categorized according to the "visible" motions around one (uniaxial), two (biaxial), or three (triaxial/multiaxial) axes of motion. The rotatory movement of the bony segments of a synovial joint are known as **osteokinematics.** During osteokinematic movement of the bony levers, the contiguous articular surfaces within a joint turn over their points of contact with each other; this shifting in points of articular contact is known as **arthrokinematics.**

Osteokinematics

Osteokinematics refers to the rotary movement of the bony levers in space during physiological joint motion.[32] These movements are typically described by the plane in which the motion occurs (sagittal, frontal, and transverse planes), the axis about which they occur (coronal, anteroposterior, and longitudinal, respectively), and by the direction of movement (flexion/extension, abduction/adduction, and medial/lateral rotation).

Arthrokinematics

Often during osteokinematics, one of the joint surfaces is relatively stable and serves as a base for the motion, whereas the other segment moves on that stable base. The terms **roll, slide,** and **spin** are used to describe the different types of changes in articular

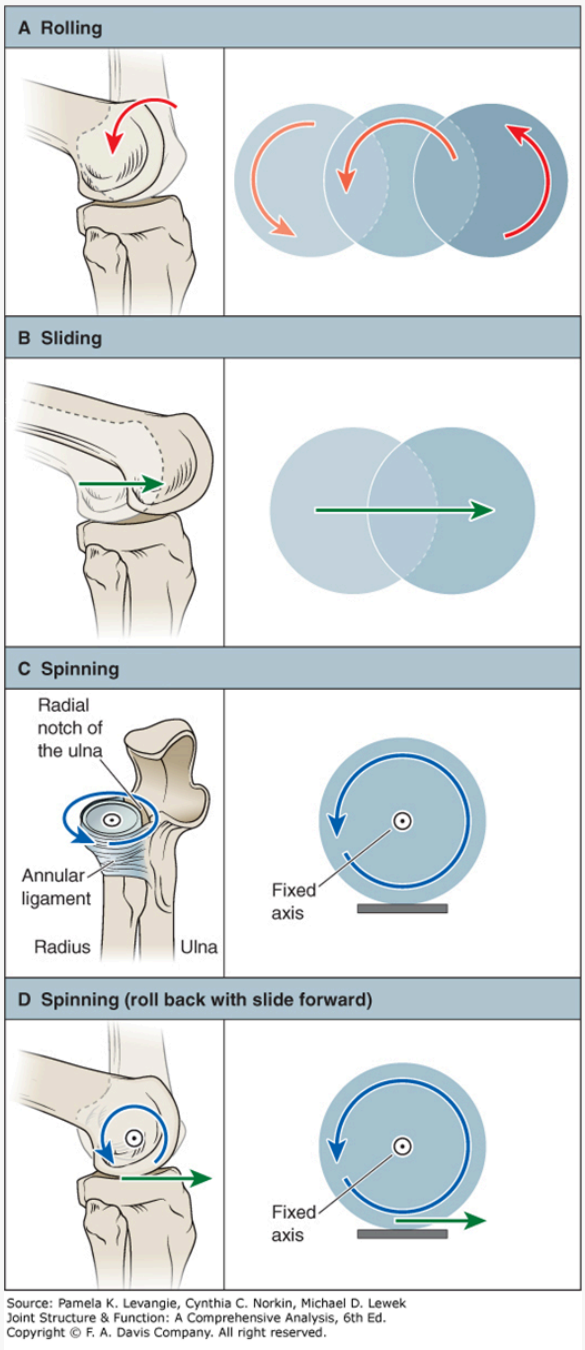
contact that the moving segment can make on the more stable segment.[17,32-34] These changes in articular contact are known as arthrokinematics, or accessory motions. Unlike osteokinematic motions, arthrokinematics cannot be volitionally isolated under normal conditions.[32,34]

A roll (Fig. 2–10A) refers to the rolling of one joint surface on another, as in a tire rolling on the road. During a pure rolling motion, a progression of points of contact between both the moving and the stable surfaces occurs. Sliding (Fig. 2–10B), also commonly known as gliding, refers to the linear translation of one component on another, as when a braked wheel skids. In pure sliding, the same point on the sliding surface moves over changing points of contact in the stable component. Combinations of rolling and sliding usually occur together during most physiological movements. Spin refers to rotation of the moving component, as when a top spins. In a pure spin around a fixed axis, the point of contact changes for the moving component but the point of contact on the stable component does not (Fig. 2–10C). A spin around a relatively fixed axis may also result when a roll and a slide occur in opposite directions and in approximately equal amounts (Fig. 2–10D). When rolling and sliding occur disproportionately, the resulting "spin" may take place about an axis that is not fixed but shifts as the motion continues.

FIGURE 2-10

Arthrokinematic joint motions include **A.** rolling (femur rolls posteriorly on the tibial plateau), **B.** sliding (femur slides anteriorly on the tibial plateau, and **C.** spinning (of the radial head within the annular ligament). **D.** When the femur simultaneously rolls posteriorly

and slides anteriorly on the tibial plateau, the effect appears to be a spin of the femoral condyles around a relatively fixed axis.



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Uniaxial, biaxial and triaxial joints are referred to as having one, two and three degrees of freedom, respectively. White and Panjabi[34] refer to joint motions and their associated

intra-articular (arthrokinematic) components as having the potential for six degrees of freedom: three degrees of rotation around three axes and three degrees of translation along those same three axes. The section on Description of Motions in Chapter 1 and other chapters in this text also refer to the potential for six degrees of freedom. The six degrees of freedom and three degrees of freedom models are not really in conflict because the White and Panjabi model is an elaboration of the simpler model. The White and Panjabi model is the foundation of at least some of the manual therapy approaches to joint interventions.

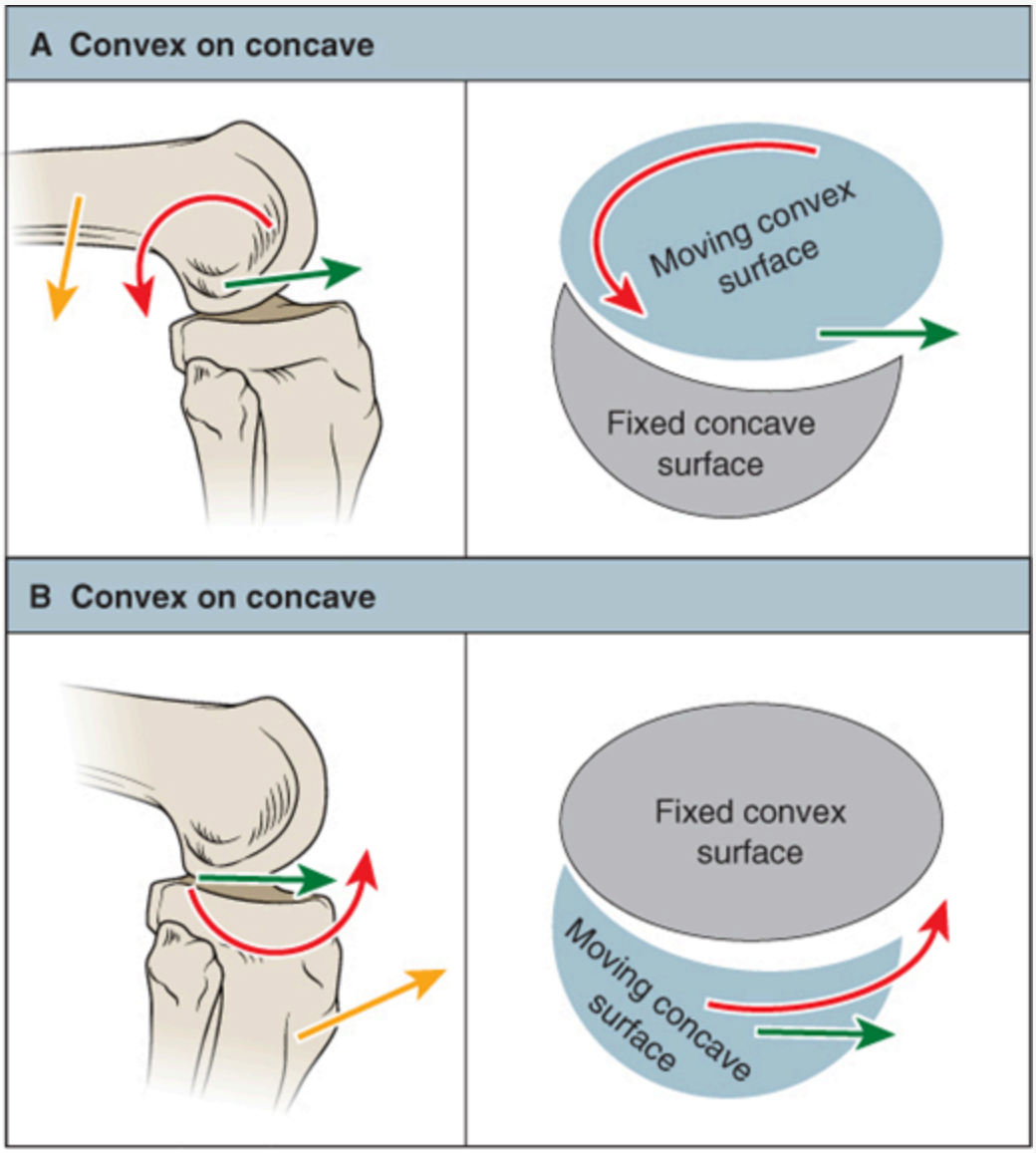
During human joint motion, rolling and sliding often occur together in differing proportions to maintain joint integrity. Whereas rolling of the articular surface occurs in the same direction as the osteokinematic motion of the moving lever (see Fig. 2–10A), the osteokinematic/arthrokinematic relationship can differ depending on the shape of the moving and stable articular surfaces. This shift in relationship is commonly referred to as the **convex-concave rule.**[35]

When a convex joint surface moves on a relatively stable concave surface, the convex surface rolls in the same direction as the osteokinematic movement of the bony lever as might be expected. However, sliding of the convex surface will occur in the *opposite direction* to the roll to maintain optimum contact. Elaborating on the example shown in Figure 2–10D, the initial stages of weightbearing knee flexion occurs with the convex femoral condyles rolling posteriorly on the fixed tibial plateau in the same direction as the posteriorly rotating (flexing) femur (**Fig. 2–11**). After this initial rolling, the femoral condyles must begin to slide *anteriorly* while continuing to roll *posteriorly* so that the femoral condyles do not roll off the posterior tibial plateau (**Fig. 2–11A**). This roll and

slide combination in the flexing knee approximates a pure spin of the femoral condyles on the posterior tibial plateau only in the final stages of knee flexion. This is in contrast to what is seen when a concave articulating surface is moving on a stable convex surface. Although rolling still occurs in the same direction as the osteokinematic movement of the segment, sliding typically occurs in the *same direction* as rolling of the articular surface. During extension of the tibia (as in a seated position), the concave tibial plateau rolls *anteriorly* in the same direction as the anteriorly rotating (extending) tibia. The tibial plateau must also slide *anteriorly* in the same direction as the roll to fully extend the tibia while maintaining contact of the tibial plateau with the femoral condyles (Fig. 2–11B). The ability of a joint to move through its full osteokinematic (physiological) range of motion (ROM) is dependent upon the ability of the contacting articulating segments to roll, slide, or spin based on the demands of the shape of the moving surface and appropriate constraints from the surrounding tissues.

FIGURE 2-11

Convex/concave rule. **A.** During flexion of the *femur,* the convex femoral condyles move on a fixed concave surface; the convex femoral condyles roll posteriorly (red arrow) in the same direction as the flexing femur (orange arrow), while sliding anteriorly (green arrow) in a direction opposite to the roll. **B.** During extension of the *tibia,* the concave tibia plateau rolls anteriorly (red arrow) in the same direction as the extending tibia (orange arrow) and slides anteriorly (green arrow) in the same direction as the roll.



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For articular surfaces to be free to move in the appropriate direction (arthrokinematics) as the bony lever rotates (osteokinematics), the joint must have a certain amount of **"joint play."** This freedom of movement of one articular surface on another can be tested by an examiner when the joint is in a **loose-packed** position, typically tested as the amount of available passive (manual) sliding of one surface upon the other. The

loose-packed position of a joint most often is described as the point in the joint's range where the capsule and ligaments provide the least restraint to passive sliding. If the supporting joint structures are excessively lax (e.g., stretched or torn), the joint may have too much play and become unstable. If the joint structures are excessively tight (e.g., adaptively shortened or hypertrophic), the joint will have too little movement between the articular surfaces; movement of the bony lever will be restricted because the appropriate intra-articular movement will not accompany the physiological movement.

For most synovial joints, there is a point in the ROM where minimal or no joint play between the articular surfaces is expected. This is referred to as the **close-packed** position for a joint and is the point in the ROM where the ligaments and capsule are maximally taut. The close-packed position is usually (but not always) at the end of a joint's ROM, is the position of greatest joint stability, and is most resistant to distraction forces that tend to cause separation of the joint surfaces; little or no joint play is possible. The position of full extension is the close-packed position for the knee and interphalangeal joints.[32] For many joints the close-packed position of a joint can also be referred to as the "locked" position as is true for the knee.



Patient Application 2–3

James Connelly, a 62-year-old male, fell off of his stepladder and fractured his tibial plateau. He was placed in a long leg cast from his thigh to his ankle that kept his knee in 20 degrees of flexion for 6 weeks. Upon removal of his cast, his ability to actively flex or fully extend his knee was very limited, although there is evidence that the tibial plateau fracture healed without any disruption to the tibial plateau. Although the knee joint musculature is understandably weak after prolonged casting, immobilization changes to the joint capsule and joint ligaments are a significant

factor in James' joint motion issues. If arthrokinematics (roll, slide, and spin) of the femoral condyles or tibial plateau are excessively constrained by tight capsuloligamentous structures, the osteokinematic motions of flexion and extension of the tibia (or of the femur) will be limited. Beginning James' treatment by facilitating the arthrokinematic motions typically expected of the tibia at the knee through manual joint mobilization techniques (increasing joint play) may be more effective and safer initially in restoring knee joint ROM than forcing the tibia into flexion or extension.

Range of Motion

The anatomic or physiologic ROM of a joint refers to the amount of *passive rotary* motion available to a joint within the anatomic limits of the joint structure around one axis and in a particular plane.[32] The extent of the anatomic range (osteokinematic motion) is determined by a number of factors, including the shape of the joint surfaces, the joint capsule, ligaments, muscle bulk, contiguous bony structures, and surrounding musculotendinous tissues, any of which can affect arthrokinematics and osteokinematics of the joint. In some joints there are no bony limitations to motion and the ROM is limited only by soft tissue structures. Other joints have definite bony restrictions to motion in addition to soft tissue limitations. The humeroulnar joint at the elbow is limited in extension by bony contact of the olecranon of the ulna with the olecranon fossa of the humerus, whereas flexion is limited most often by approximation of the forearm and biceps brachii muscle bellies. The sensation experienced by the examiner performing passive movements to the end of physiologic ROM at each joint is referred to as the end-feel. Using terminology first promoted by Kaltenborn[35], a motion that is limited by approximation of soft tissues (as is typical of elbow flexion) is considered to have a **soft end-feel**; a motion limited by capsuloligamentous restraints (as is typical of knee extension) is considered to have a **firm end-feel**; and a motion with

a bony limitation (as is typical of elbow extension) is considered to have a **hard end-feel.**

A joint ROM is considered to be atypical when motion either exceeds or fails to reach the typically expected anatomic (osteokinematic) limits of motion. When a passive ROM exceeds the expected limits, the joint can be labeled as **hypermobile.** When the passive ROM is less than what would typically be permitted at the joint, the joint can be labeled as **hypomobile.** Either hypermobility or hypomobility of a joint may have negative consequences not only at the affected joint but also on adjacent joint structures. Increased joint stresses may result in further changes to joint structures and can lead to activity and participation limitations.

Hypermobility may be caused by a failure of bony elements or soft tissues, including weak musculature, to restrain the motion. Inadequate joint restraints can lead to excessive joint play and instability. For example, the anterior cruciate ligament is a major stabilizing ligament of the knee, preventing, among other movements, excessive anterior sliding of the tibial plateau on the femoral condyles. Rupture of this ligament can lead to knee joint instability if the surrounding muscles (e.g., hamstrings) and remaining articular structures (capsule, ligaments, and menisci) cannot adequately substitute for the torn ligament. The result may be avoidance of or stopping activities that stress the now unstable knee joint, as well as damage to other joint structures through excessive stress.

Hypomobility of a joint may be caused by bony or cartilaginous blocks to motion or by the inability of the capsule, ligaments, or muscles to elongate sufficiently to allow a typical ROM. For example, the hip typically extends during the terminal stance phase of gait as the body passes over the supporting limb. With hip osteoarthritis, hip extension

can be limited both by the resulting overgrowth or unevenness of the articular surfaces and by excessive tension in the hip flexors that is a response to hip pain. Step length will be shortened because of the lack of hip extension. The person may compensate by increasing lumbar extension in terminal stance to increase step length and facilitate forward progression. This adaptive change, over time, can have negative consequences to the low back. A **contracture** is a shortening of soft tissue structures around a joint with accompanying fibrotic changes. A contracture can be another cause of hypomobility or may be a consequence of sustained hypomobility over time.



Foundational Concepts 2–5

Joint Motion

- Osteokinematics: rotary movement of the bony lever(s) of a joint in one or more planes and around one or more axes.
- Arthrokinematics: rolling, sliding, or spinning of one contiguous joint surface on another.
- Convex-concave rule: a convex joint surface that moves on a stable concave surface generally rolls in one direction and slides in the *opposite* direction; a concave joint surface that moves on a stable convex joint surface generally rolls and slides in the *same* direction.
- Physiologic ROM: the typical amount of expected passive joint rotation in one plane within the anatomic limits of the joint structure as a function of both osteokinematic and arthrokinematic factors.
- End-feel: the soft, firm, or hard sensation experienced by the examiner at the end of the available passive ROM at each joint, depending on structure(s) that check further motion.

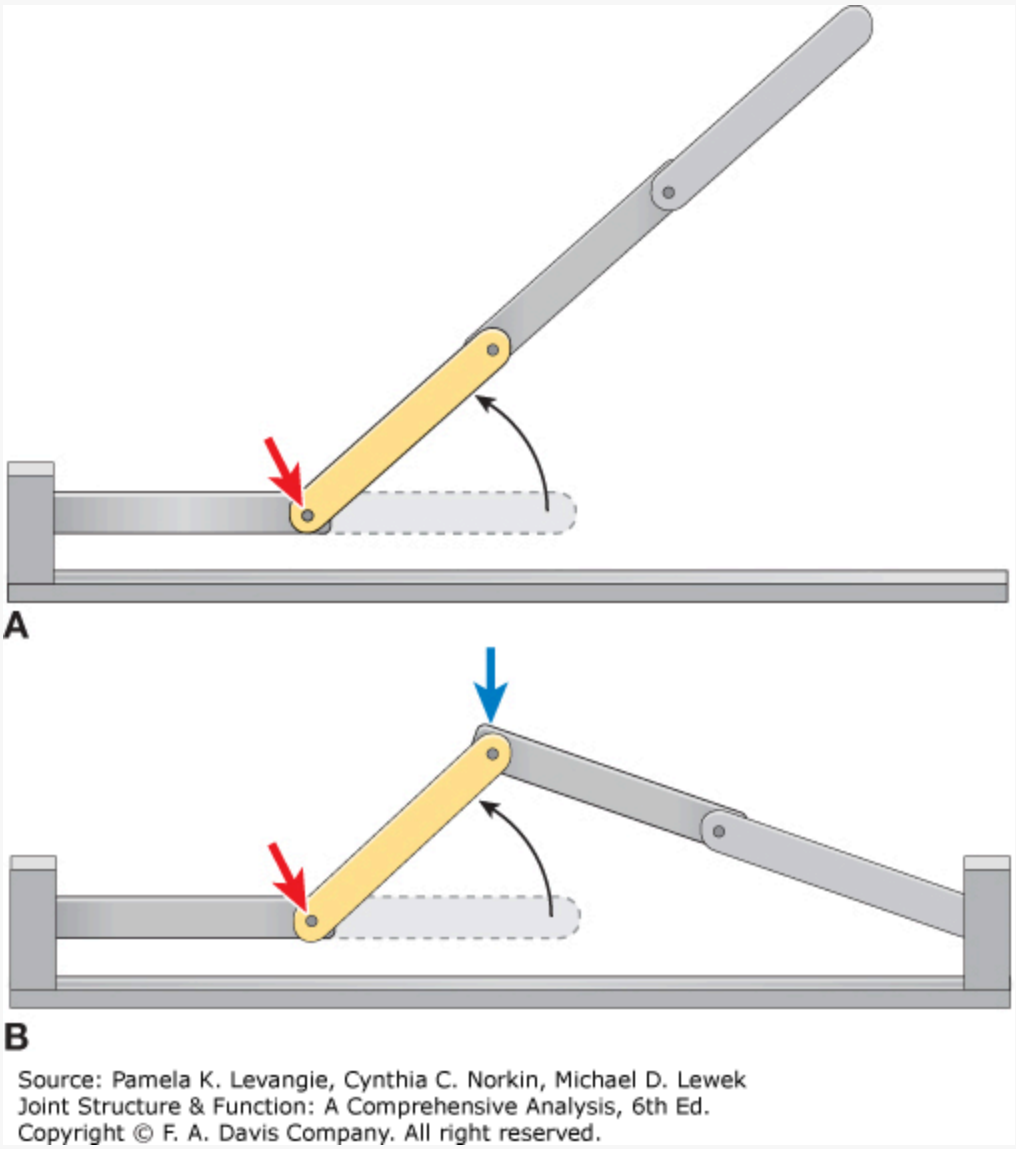
Kinematic Chains

The concepts of open and closed chains were introduced in Chapter 1 in the section on Multisegment (Closed Chain) Force Analysis to identify how forces applied to one segment can affect another segment. The concepts are also relevant to understanding

the interdependence of how segments move through space. Engineers originally described a so-called “kinematic” chain as a series of rigid segments linked together by a series of pin-centered joints (as, literally, in a length of large-linked chain). In an **open chain,** one end of the series of segments is free to move while the other is fixed; movement can take place at one link (joint) without causing motion at other links (joints) in the chain, although the segments will move in space (Fig. 2–12A). In a **closed chain,** both ends of the chain are fixed; movement at one link (joint) not only moves one or more other segments in space but will also create movement in one or more other links (joints) in the chain[32] (Fig. 2–12B).

FIGURE 2-12

Mechanical open and closed chains. **A.** In an open 4-segment chain (fixed at only one end), rotation of one segment can occur around a single axis (red arrow) without causing rotation at another axis. **B.** In a closed 4-segment chain (fixed at both ends), rotation of the same segment causes rotation not only around the same axis (red arrow) but around at least one other axis (blue arrow) as well.



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The term *closed chain* has often been applied to human movements that take place under weightbearing conditions, where the distal segment of a limb is not free to move (see Expanded Concepts 1–10 in Chapter 1). Under weightbearing conditions, motion at one joint generally is accompanied by motion at one or more joints for the body to remain stable. For instance, under open-chain (non-weightbearing) conditions, the right foot is not fixed on the ground; right knee flexion can occur independent of motion at the contiguous hip and ankle joints (Fig. 2–13A). When both feet are weightbearing

and fixed on the ground, knee flexion will occur bilaterally and result in simultaneous hip flexion and ankle dorsiflexion if the head is to remain upright and the line of gravity is to remain within the base of support (Fig. 2–13B). In this instance (and as is common in bilateral weightbearing), the closed chain includes all joints between the right foot, up the right lower extremity, across the pelvis, and down the left extremity to the left foot. The closed chain may also be thought of as extending from the weightbearing feet to the head that is relatively fixed in space (eyes oriented vertically and over the base of support). Although weightbearing and closed chain are sometimes used synonymously, the person in Figures 2–13A and B is weightbearing in both instances. Weightbearing and closed chain are synonymous only when both ends of the chain are fixed, whether by bearing weight or through some other constraint or adaptive mechanism.

FIGURE 2-13

Open and closed chains in human motion **A.** Knee flexion in an open chain may occur without motion at the contiguous hip and ankle. **B.** In a closed chain, knee flexion must be accompanied by hip flexion and ankle dorsiflexion to keep the head upright and the line of gravity within the base of support.



A B

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In human movement, body segments, especially the upper extremities, are frequently free to move distally (or at one end) in an open chain. Motion of bony segments in an open chain may function either independently of each other or in a myriad of combinations limited only by available degrees of freedom and ranges of motion of each joint in the chain. If one joint in an open chain is immobilized by an atypical

structural constraint, the lost motion may be made up by motions at more proximal or distal joints in the chain. In Figure 2–12A, immobilization of the joint at the red arrow would not prevent full ROMs from occurring at the more distal joints. Motions of joints in a closed chain are more predictable because the interdependence of the contiguous segments reduces the available options. If one joint in a closed-chain is immobilized, motion at contiguous joints may also be lost. In Figure 2–12B, immobilization of the joint at the red arrow would prevent any motion at the next joint (blue arrow) and probably at the next joint axis as well. The concept of open and closed chains is valuable for understanding both kinematic and kinetic concepts of human motion, effects of therapeutic exercise, and consequences of injury and disease on the joints of the body. Therefore, the concepts will be referenced throughout this text to understand the extent of functional loss that might occur with joint injury or disease.



Patient Application 2–4

George Chen is a 40-year-old electrician and business owner who caught his skate on another player's stick when playing ice hockey. George ended up with a bimalleolar (tibial and fibular malleolar) fracture that was treated with open reduction and internal fixation. A plate and screws were placed along the distal fibula; additional screws were placed to secure the medial malleolus and to stabilize the distal tibiofibular joint. He was non-weightbearing on crutches for a period of time, followed by a walking boot. When the walking boot was discontinued, George had only 5° dorsiflexion and 20° plantarflexion, as well as restricted subtalar motion. George's current foot/ankle limitations will affect the entire lower extremity chain when he is weightbearing. With his ankle ROM limitations and without mobility in the subtalar joint to accommodate, George's knee and hip flexion will be limited sufficiently to affect weightbearing activities like walking (especially on sloped surfaces), stair climbing, sitting in low chairs, and squatting to help his daughter tie her shoes. With manual joint mobilization, the subtalar mobility (largely impaired by the immobilization rather than by injury) can be restored but the hardware in the

tibia and fibula may limit restoration of tibiofibular, and therefore ankle joint, function.

Materials Found in Human Joints

Having described the structures that make up fibrous, cartilaginous, and synovial joints and the factors that influence movements available to those joints, this section focuses on the tissues that make up those joint structures: connective tissue. Human joints are comprised of living tissues that change their structure in response to changing environmental or functional demands. Tissues require nourishment and some amount of stress to survive; they can adapt to imposed stresses or demands but may also become injured if the tissues' adaptation fails. The ability of the tissues to adapt can be challenged by a variety of factors including injury, disease, and aging. An understanding of the composition of tissues and how they respond to stresses and strains is necessary to understand how tissues adapt or fail to adapt to imposed stresses.

Composition of Connective Tissue

The joint structures that were discussed thus far in this chapter are all composed of connective tissues, including bones, bursae, capsules, cartilage, discs, labrums, menisci, plates, ligaments, and tendons. These joint structures fall into three primary categories of connective tissue, including bone, cartilage, and **dense connective tissue.** Although there are other classifications of connective tissues, this chapter will detail primarily these three. The gross anatomic structure and microarchitecture, composition, and biomechanical behavior of connective tissues is under continual investigation as changes in technology improve our ability to understand these tissues. Despite the variations among connective tissue types, there are elements that are

common among them. Connective tissues are characterized by widely dispersed cells and a large volume of **extracellular matrix.** At the microscopic level, the extracellular matrix has both **interfibrillar** (also known as the **ground substance**) and **fibrillar** (fibrous) **components.** Whereas tissues such as nerve and muscle depend predominantly on cell structure to determine function, connective tissue function is strongly influenced by its extracellular components.

Cells

The cells of all connective tissues function primarily (although not exclusively) to synthesize the extracellular matrix of connective tissue. The **fibroblast** is the basic cell of most connective tissues. Depending on its mechanical and physiological environment, fibroblasts may specialize to become **chondroblasts** (cartilage), **tenoblasts** (tendon), or **osteoblasts** (bone); these cells are called **fibrocytes, chondrocytes, tenocytes,** and **osteocytes** when they mature and become less metabolically active. The distinction between a "blast" and a "cyte" is based primarily on appearance, which reflects cell synthetic activity. Generally, "blasts" are more metabolically active in secreting both fibrillar and interfibrillar components of the extracellular matrix, whereas "cytes" are more mature cells that maintain the interfibrillar component of the extracellular matrix. Given the appropriate environment or stimuli, differentiated connective tissue cells can "de-differentiate" and change the type of extracellular matrix they produce. For example, tendon cells can produce a cartilage-like (rather than tendon-like) tissue when subjected to prolonged compressive forces.[36–38] Such findings suggest that connective tissue structure can be modified by changes in loading conditions; therefore, it might be possible to manipulate the mechanical environment to cause connective tissues to synthesize somewhat different materials that might enhance their function.

Extracellular Matrix

The extracellular matrix is the part of connective tissues outside the cells. It comprises almost the entire volume of connective tissue and determines the tissue's function. The extracellular matrix primarily contains proteins and water and is organized into its fibrillar and surrounding interfibrillar components.

FIBRILLAR COMPONENT

The fibrillar, or fibrous, component of the extracellular matrix contains two major classes of structural proteins: collagen and elastin.[11] **Collagen,** the main substance of most connective tissues, is found in all multicellular organisms. The most abundant protein in the human body, it accounts for 25% to 30% of all protein in mammals.[39] Collagen has a tensile strength similar to steel and is responsible for the functional integrity of connective tissue structures and the resistance of connective tissue to tensile forces.[39–47]

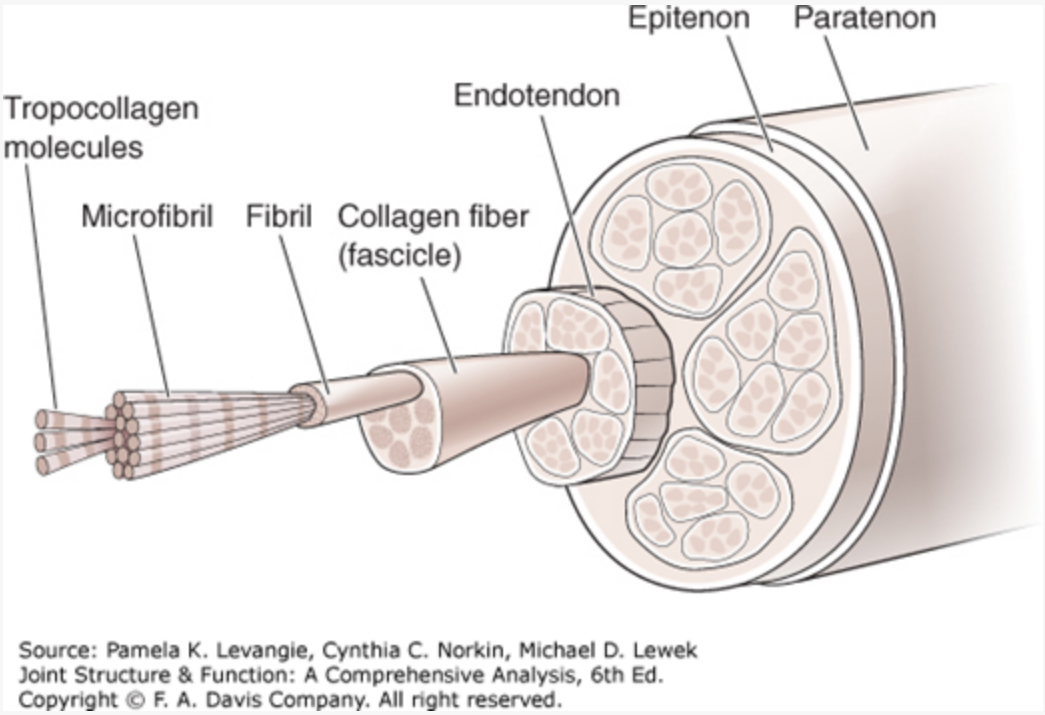
Many types of collagen have been identified, but the functions of many are not yet well understood.[39–43] The fibril-forming collagens (types I, II, and III) are the most common. Type I collagen accounts for 90% of the total collagen in the body and is found in most connective tissues, including tendons, ligaments, menisci, fibrocartilage, joint capsules, synovium, bones, labrums, and skin.[39–45] Type I collagen appears to be responsible for the tensile strength of tissues. Type II collagen is found mainly in cartilage and intervertebral discs where compressive forces are common.[40,41] Type III collagen is found in skin, joint capsules, muscle and tendon sheaths, and in healing tissues where, like type I, tensile stresses are common.[41,47]

The basic building block of collagen is a triple helix of three polypeptide chains called

the **tropocollagen molecule.** The tropocollagen molecules aggregate in groups to form **microfibrils**; the microfibrils in turn combine to **fibrils** (fibers) of varying sizes[44,47] (**Fig. 2–14**). Cross-links stabilize and strengthen the fibrils, with the cross-links being either intramolecular (between peptide chains within a collagen molecule) or intermolecular (between molecules of adjacent fibrils).[48] Fibrils enlarge as more collagen is added to them and as more cross-linking occurs among neighboring molecules; thus, older fibrils are larger and contain more cross-links, making them stronger. Generally, the organization of type I collagen in both tendons and ligaments continues to follow a hierarchical structure where fibrils collect to form successively larger aggregations or bundles. Specific descriptions and nomenclature for successive bundles vary across resources but all appear to agree on a hierarchical pattern.[27,44] As seen in Figure 2–14, collagen fibrils bundle together to form the **collagen fiber.** Cross-links formed between the collagen fibers may be arranged in many different ways and vary in size and length. In most relaxed tissues, the collagen fibers have a wavy configuration called a crimp. When collagen fibers are stretched, the crimp disappears. Collagen fibers bundle to create successively larger bundles that ultimately constitute the tendon or ligament.

FIGURE 2-14

Dense connective tissues, using the tendon as an example here, have a hierarchical structure beginning with the tropocollagen molecule (left) and building to the composed tissue (right), with successive bundles enclosed in an endotendon sheath and, ultimately, the epitenon or paratenon.



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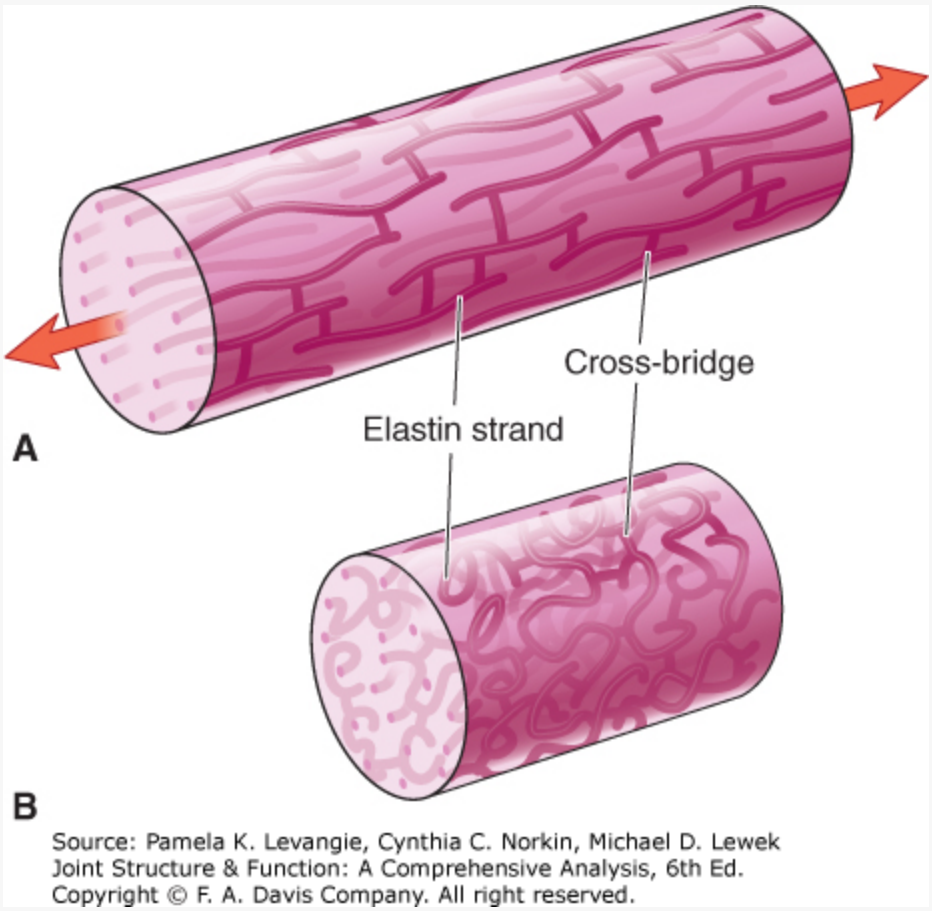
Bundles of collagen fibers are enclosed in loose connective tissue sheaths, called **endotenons,** in a tendon (see Fig. 2–14). These sheaths enclose the nerves, lymphatic vessels, and blood vessels supplying the tendon and include cross-links to adjacent fibers.[44] The **epitenon** encircles the tendon as a whole and is connected to the endotenon on its inferior surface. Many tendons are also covered by a **paratenon** on its outer layer. Tendons that wrap around bones or tendons do not have a paratenon but are covered by tendon sheaths with synovial linings that interface with the tendon to reduce friction.[27]

Elastin also is found in many connective tissues but, unlike collagen, the molecule consists of single alpha-like strands without a triple helix.[11] The alpha-like strands are cross-linked to each other to form rubber-like elastic fibers. Each elastin molecule uncoils into a more extended formation when the fiber is stretched (Fig. 2–15A), then

recoils spontaneously when the stretching force is removed (Figure 2–15B). Elastin fibers branch freely and are found in all joint structures including the skin, the tracheobronchial tree, and the walls of arteries. Elastin makes up a much smaller portion of the fibrous component in the extracellular matrix than does collagen. As one might expect, tissues that require “give” contain more elastin. The aorta contains approximately 30% elastin and 20% collagen (percentage of the tissue dry weight), the ligamentum nuchae of the cervical spine has 75% elastin and 15% collagen, whereas the Achilles tendon contains only 4.4% elastin and 86% collagen.

FIGURE 2-15

A. When an elastin fiber is under tension (stretched), the strands straighten out and the fiber lengthens, with the length limited in part by cross-linking of the strands. **B.** When tension on the elastin fiber is released, the strands recoil and the elastin fiber shortens.



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INTERFIBRILLAR COMPONENT

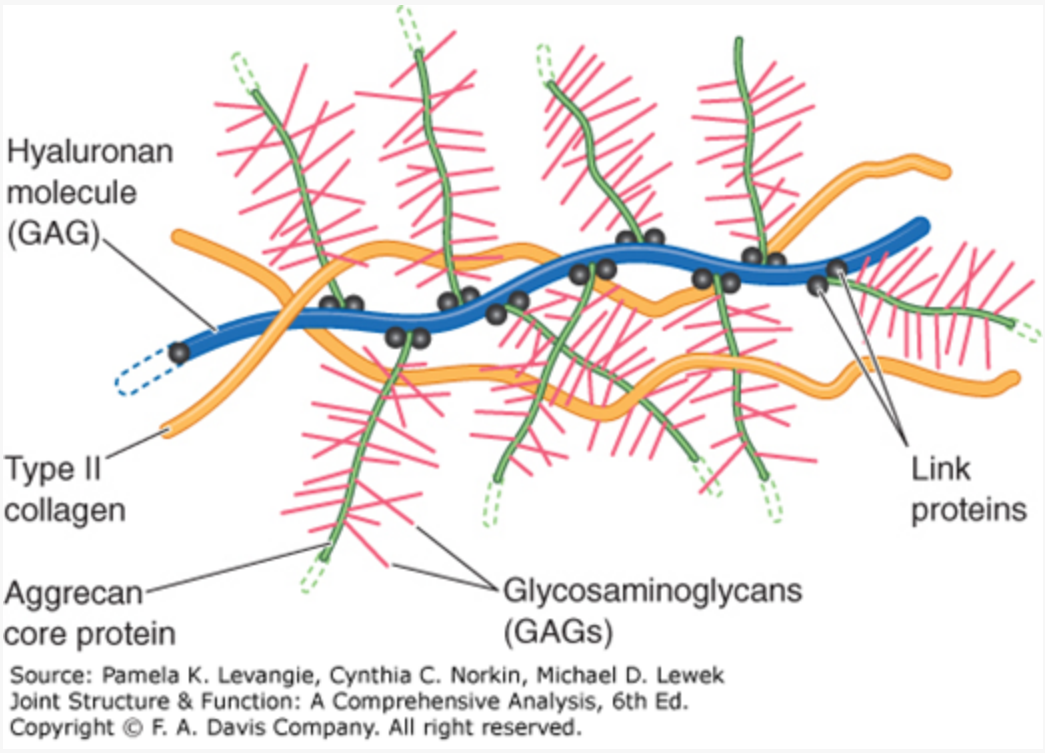
The interfibrillar component of connective tissue, often referred to as the ground substance, contains primarily water and proteins called **proteoglycans (PGs)** with attached **glycosaminoglycans (GAGs).**[49–51] A PG can contain up to approximately 100 GAGs, which stick out from its protein core to form a shape like a bottle brush.[52,53] Hyaluronan is a GAG that does not attach to the protein core but exists as either a free GAG chain of variable length (e.g., in a tendon or ligament) or a core molecule to which large numbers of PGs are attached (e.g., in cartilage).

One large cartilage PG is called **aggrecan.** The protein core of aggrecan has GAGs (such as chondroitin sulfate and keratin sulfate) bristling out from its length; the protein core

attaches to a hyaluronan chain via separate link proteins[52] (Fig. 2–16). These large aggregates of PGs with their attached GAGs are largely responsible for the water-binding that characterizes the extracellular matrix of cartilage and gives articular cartilage the ability to withstand compression. Some of the GAGs associated with articular cartilage (hyaluronan, chondroitin sulfate, and glucosamine) are used as injectables or supplements to treat osteoarthritis, although their efficacy remains unproven.[54]

FIGURE 2-16

The extracellular matrix (ECM) of articular cartilage includes the large aggrecan proteoglycan (PG) with glycosaminoglycans (GAGs) attached to its protein core. The protein core is stabilized on the hyaluronan filament by link proteins. These interfibrillar components will lie between, in this example, type II collagen fibers.



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The PGs in the extracellular matrix of a connective tissue (bone, cartilage, tendon, or ligament) affect its hydration through their attached GAGs.[53] The GAG chains attract water into the interfibrillar matrix, creating a tensile stress on the surrounding fibrillar collagen network. The collagen fibers resist and contain the swelling, thus increasing the rigidity of the extracellular matrix. This increased rigidity allows the tissue to resist compressive forces. The PGs also form a reservoir for nutrients and growth factors that may play a role in directing or limiting the size of collagen fibers. The amount and type of PGs in a tissue is another example of the form-function interaction of connective tissues because tissues that are subjected to high compressive forces (like cartilage) have more PGs and different GAGs than tissues that resist tensile forces.[37,38] Table 2–2 provides a synopsis of the variations in extracellular composition found in connective tissues associated with synovial joints.

Table 2-2

Composition of Extracellular Matrix for Selected Connective Tissues

Foundational Concepts 2–6

Connective Tissue Composition

Whether a connective tissue is bone, cartilage, or dense connective tissue (tendon or ligament) is determined by its cellular and extracellular composition. Cellular composition may shift depending on the demands placed on the tissue (form/function interaction):

- Cells
  - Fibroblasts/cytes: the basic cell forms that can differentiate into other cell types and are the predominant cell in ligaments; fibroblasts produce the extracellular matrix
  - Tenoblasts: found in tendons
  - Chondroblasts/cytes: found in cartilage
  - Osteoblasts/cytes: found in bone
- Extracellular Matrix
  - Interfibrillar components: water, PGs, and GAGs
  - Fibrillar components: collagen (mainly type I or II) and elastin

Specific Connective Tissue Composition

The following sections provide brief overviews of the more distinguishing elements in the composition of connective tissue structures that are associated with synovial joints.

Ligaments

Ligaments are heterogeneous structures containing only a small amount of cells (about

10% to 20% of the tissue volume, primarily consisting of fibroblasts) and a large amount of extracellular matrix (about 80% to 90% of tissue volume). The fibrillar component of the extracellular matrix in most ligaments is composed of mainly type I collagen with varying amounts of elastin. One notable exception is the ligamentum flavum of the spine, which has a distinctly yellowish color and contains a large amount of elastin (75% of the tissue dry weight).[11] The type I collagen fibrils in ligaments are densely packed with a relatively low concentration of interfibrillar proteoglycans. Most ligaments have a similar hierarchical structure to that of tendons[55] (see Fig. 2–14), with fiber bundles arranged in line with the applied tensile forces. The arrangement of the collagen fibers and the collagen/elastin fiber ratio varies among ligaments, depending on the relative abilities of these structures to provide stability and allow mobility for a particular joint.

Ligaments are subjected to varying directions of tensile forces depending on the joint angle, so the collagen fibers in ligaments run in many directions—enabling the ligament to resist forces in more than one direction. For example, the posterior fibers of the medial collateral ligament of the knee may be stressed by extension, while the middle fibers are under tension when a varus stress is applied to the knee joint.

The appearance and composition of ligaments change near their **entheses**, the attachments to bone. Ligaments may insert directly into bone via fibrocartilage or indirectly via fibrous attachments.[56] With fibrous attachments, the collagen fibers blend into the **periosteum** of the bone, which is in turn attached to the underlying cortical bone via **Sharpey's fibers**. These fibrous attachments have been likened to trees that are rooted in the ground, with Sharpey's fibers serving as the "roots" for the ligament

(and similarly for a tendon). The stiffening of the ligament-bone interface at direct fibrocartilage insertions decreases the likelihood that the ligament will give way at the enthesis; however, it is a common site for degenerative change, usually in the underlying bone.[56–59]

Tendons

Tendons and ligaments have a similar composition and basic structure although the cells in tendons are predominantly tenoblasts and tenocytes and the proportions and organization of the components of their extracellular matrix differ slightly.[51] Tendons contain mostly type I collagen (more than 95%), with type III in the tendon sheaths.[43,59] Tendons contain slightly more type I collagen and slightly less type III collagen than ligaments (see Table 2–2). The increased type I collagen in tendons (compared with ligaments) is thought to be an adaptation to larger tensile forces because type I collagen is considered stronger than type III collagen.[43]

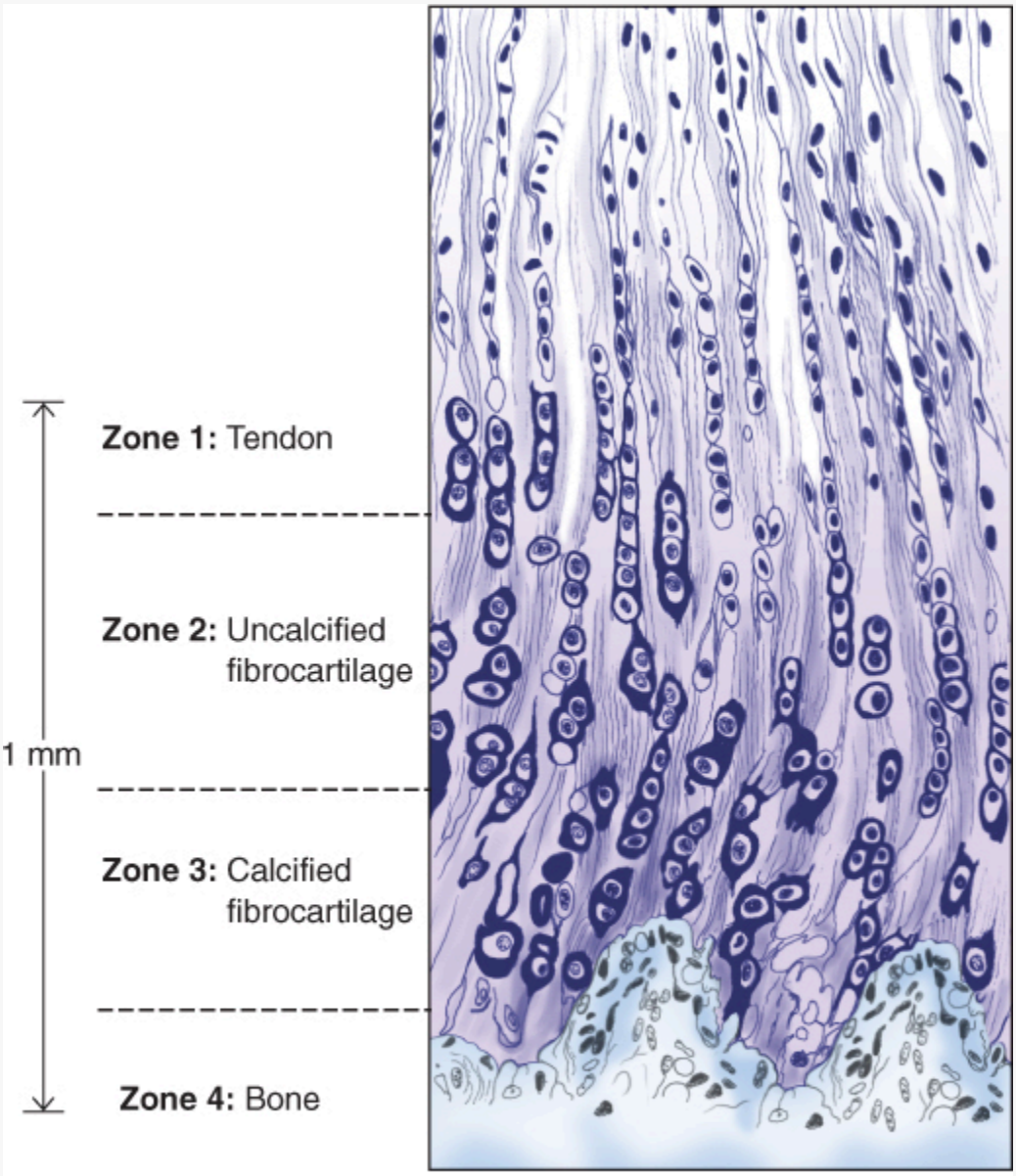
The collagen fibrils of the tendon form successively larger bundles surrounded by sheaths as described in Figure 2–14.[46] The loose connective tissue sheath of the endotenon contains mostly type III collagen fibrils and encloses the nerves, lymphatic vessels, and blood vessels that supply the tendon.[43] Each fascicle of the tendon is associated with a discrete group of muscle fibers or motor units at the muscle-tendon junction.[60,61] Several fascicles may form a larger bundle that also is enclosed in the endotenon. The epitenon sheath encloses the entire tendon. The paratenon is a double-layered sheath of areolar tissue that is loosely attached to the outer surface of the epitenon. The epitenon and paratenon together are sometimes called the **peritendon.** The peritendon may become a synovium-lined sheath called the **tenosynovium** (or

tendon sheath) in tendons that are subjected to high levels of friction such as where tendons pass over bony prominences or through fibrous tunnels. The paratenon protects the tendon by allowing it to freely slide past adjacent structures, and provides a source of replacement cells if the tendon is injured. The tensile forces on tendons are created by a muscle's contractile force. In contrast to ligaments, forces in tendons tend to be applied in primarily one direction, so the collagen fibers in tendons are straighter and more parallel. The tendon attaches to bone at one end and to the muscle at the other, each with unique structures.

Tendons, like ligaments, can attach to bone in one of two ways: either a fibrocartilaginous or a fibrous enthesis.[56] The fibrocartilaginous attachment of tendon to bone involves gradual changes in the tendon structure over a length of about 1 mm, divided into four zones based on histological observation (Fig. 2–17).[56] The first zone of the enthesis consists of tendon proper that is continuous with the midsubstance of the tendon. The second zone contains uncalcified fibrocartilage and marks the beginning of the transition from tendon to bone. The third zone contains calcified fibrocartilage, and the fourth zone is bone. The changes in tissue are gradual and continuous, which is presumed to aid in effective load transfer between the very different tendon and bone materials. A **tidemark** frequently appears between the calcified and uncalcified parts of the enthesis, representing the boundary between hard and soft tissues; however, the tidemark is not an absolute line of demarcation. Dramatic changes in connective tissue composition occur in the different zones, suggesting that the tendon insertion is subjected to both compressive and tensile forces.

FIGURE 2-17

The bone-tendon (or ligament) fibrocartilaginous junction. There are four zones, from pure tendon (zone 1) to bone (zone 4). In between zones 1 and 3, the material gradually transitions from fibrocartilage (zone 2) to mineralized fibrocartilage (zone 3).



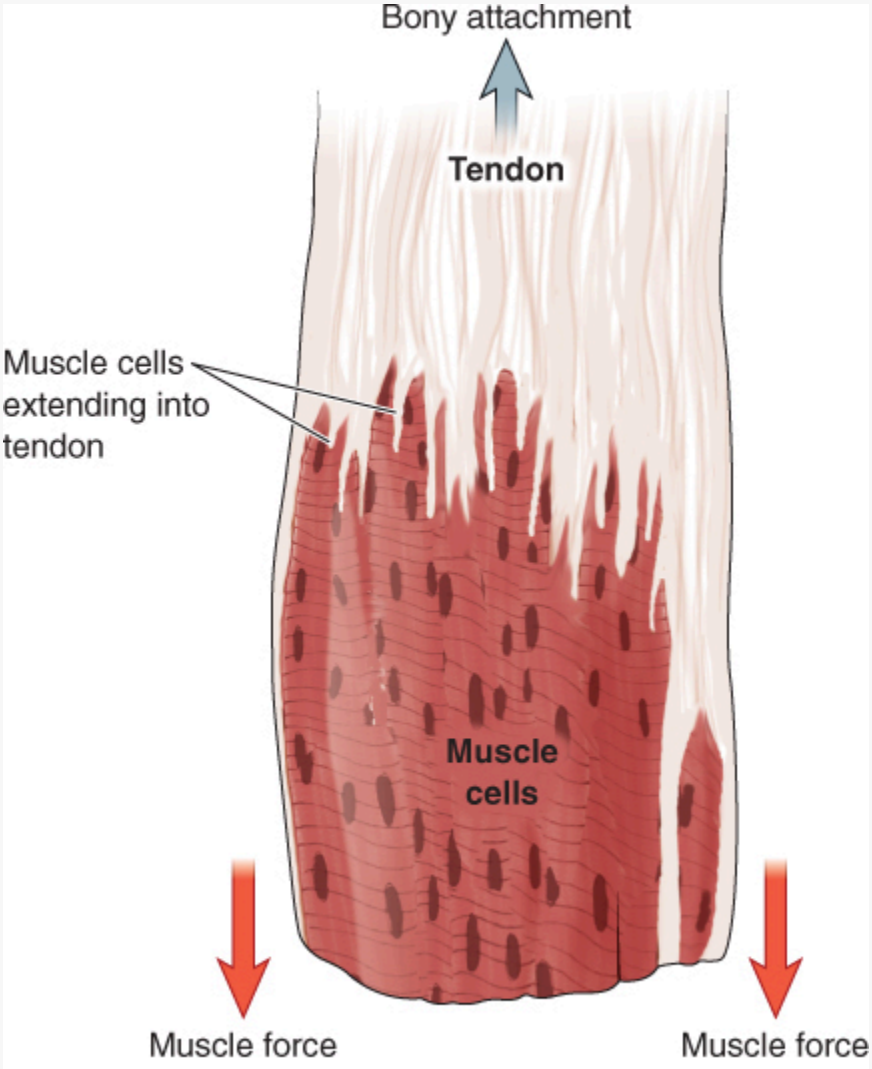
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The fibrous entheses may be divided into two categories: periosteal and bony.[56,58] At the periosteal enthesis, the tendon fibers attach to the periosteum, which provides an indirect attachment of tendon to bone. With bony entheses, the tendon attaches directly to the bone. There can be a mixture of the two types of attachment, and periosteal attachments sometimes convert to bony attachments with age. Examples of these mixed attachments include the attachments of the deltoid muscle to the humerus, scapula, and clavicle. The new enthesis formed after the surgical reattachment of a tendon to bone is initially fibrous, although a fibrocartilage attachment may form with time.[56]

Tendons attach to muscle at the **myotendinous junction (MTJ)** through highly folded membranes where the muscle fibers interdigitate with the tendon[60,61] (Fig. 2–18). There are direct connections between the muscle fiber cell membrane and the fibroblasts, PGs, and collagen of the tendon. The endotenon and epitenon of the tendon blend into the endomysium and epimysium of the muscle, respectively, forming a meshwork of connective tissue around the muscle fibers. The interdigitating configuration of the musculotendinous junction is essential for normal function of the musculotendinous unit, which is very sensitive to mechanical conditions and tends to become flatter and less infolded when loads are decreased. This change weakens the junction and may make it more susceptible to injury. Thus, when muscle and tendon loading begins after a period of immobilization, loads should begin at a lower level and progress gradually to reduce risk of injury.

FIGURE 2-18

The musculotendinous junction. The muscle cells interdigitate with the tendon. The endotendon blends into the endomysium, and the epitenon blends into the epimysium, which forms a meshwork of connective tissue around the muscle fibers.



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Foundational Concepts 2–7

Tendon and Ligament Connective Tissues

- Contain mostly type I collagen, which has the greatest tensile strength of any collagen type.
- Reflect the stresses placed on them through alignment of collagen, with tendons having a more parallel alignment from linear muscle pulls than ligaments that may be required to resist stresses in multiple directions at the joint.
- Have collagen that is organized in a hierarchical structure that aligns with the applied forces to accommodate the high tensile forces experienced by these tissues.
- Attach to bone through a gradual transition in tissue composition that "diffuses" the load at the tissue-bone interface, perhaps helping prevent injury.[56]
- Have attachment sites that are highly sensitive to changes in loading, especially declines in loading.

Cartilage

Cartilage is usually divided into the following types: fibrocartilage, hyaline (articular) cartilage, and elastic cartilage.[62] Hyaline cartilage lines the articulating ends of bones and is one of the distinguishing features of a synovial joint. Fibrocartilage forms the bonding material in many cartilaginous joints, such as at the interbody articulations in the spine and the costosternal joints. Fibrocartilage may also be present in some synovial joints in forms such as discs or plates. Elastic cartilage is found in the ears and epiglottis and has more elastin than fibrocartilage has.[62] Elastic cartilage will not be discussed further because it does not occur in or around joints.

HYALINE CARTILAGE

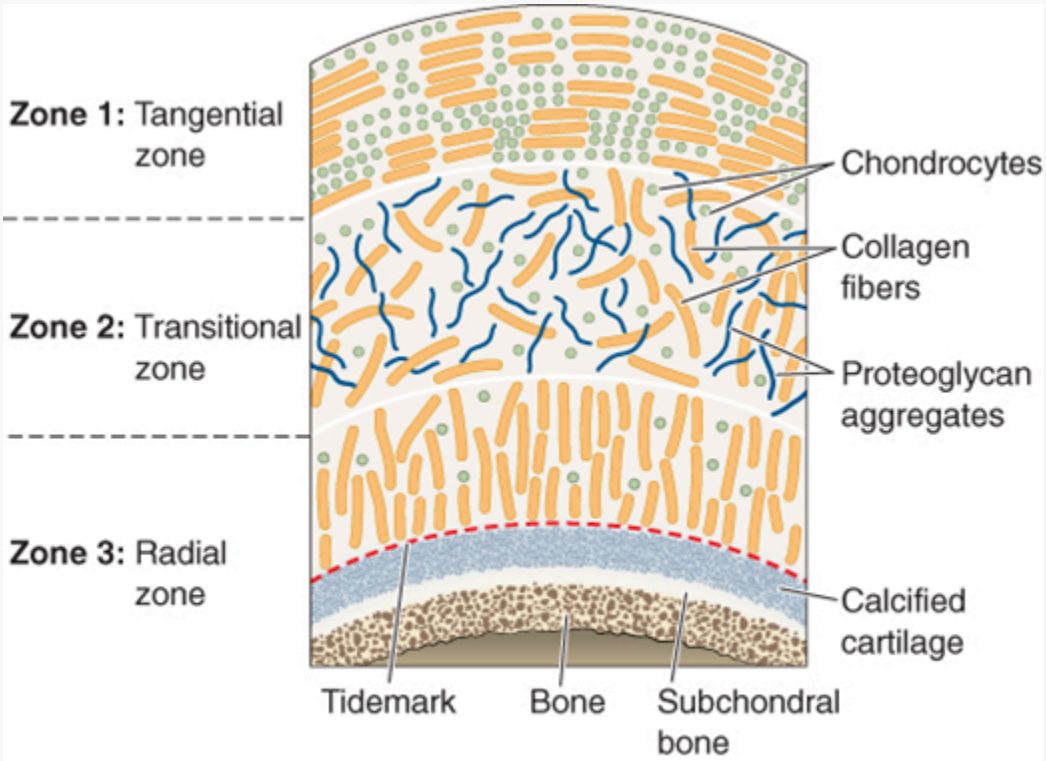
Hyaline (articular) cartilage includes chondroblast and the more mature chondrocyte cells and has an extracellular matrix that contains much more interfibrillar material than that of tendons and ligaments.[63] The chondroblasts produce collagen, PGs (mainly aggrecan), and GAGs that are all extruded into the extracellular matrix. The fibrillar

component of the extracellular matrix includes some elastin and other types of collagen, but type II collagen accounts for about 90% to 95% of the collagen content.[63] Collagen is dispersed throughout the interfibrillar component of the extracellular matrix,[49] forming a meshwork that surrounds and compresses the PGs and their attached water molecules.[49,63] Articular cartilage contains much more PG content than do other joint structures.

Three distinct layers (zones) of articular cartilage are found on the ends of the bony components of synovial joints[64] (**Fig. 2–19**). In the outermost layer (zone 1: tangential zone), type II collagen fibers are arranged parallel (tangential) to the joint surface. This layer faces the joint cavity that lies within the joint capsule. This smooth outer layer helps reduce friction between opposing joint surfaces and distributes forces over the joint surfaces. In the next layer (zone 2: middle or transitional zone), type II collagen fibers form an open meshwork. The collagen network keeps the interfibrillar PG and water contained, giving the cartilage its ability to absorb compressive forces. In the third layer (zone 3: radial zone), perpendicular (radial) collagen fibers cross the interface between uncalcified and calcified cartilage to find a secure hold in the calcified cartilage.[6,49] The calcified layer of cartilage, sometimes referred to as the fourth zone, lies adjacent to subchondral bone and anchors the cartilage securely to the bone.[63] This calcified layer is impervious, limiting the ability of nutrients to cross into the cartilage from the vascular bone underlying it.[65] The interface between the calcified and uncalcified cartilage is the tidemark[62,63]—similar to that found between the calcified and uncalcified areas in the fibrocartilaginous connection of tendon to bone.

FIGURE 2-19

Structure of hyaline cartilage with the “zones” named by their largely distinctive organization of collagen fibers.



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Hyaline cartilage is thought to have virtually no cellular turnover, based on the facts that the tissue: (1) is hypocellular and avascular; (2) relies on diffusion for its nutrient supply; and (3) contains only terminally differentiated cells. Normally, replacement of the calcified layer of articular cartilage with bone occurs by **endochondral ossification.** The calcification front advances toward the noncalcified area of cartilage slowly, in equilibrium with the rates of absorption of calcified cartilage and, perhaps, new extracellular matrix formation by cells.[66,67] In aging, the balance changes as deeper

layers of cartilage (e.g., zone 3) are gradually replaced with bone, resulting in cartilage thinning more superficially. In injuries that involve microfractures to the subchondral bone, a secondary ossification center in the bone may be activated to produce new bone growth, which expands into the calcified layer of cartilage, advancing the tidemark and thinning the noncalcified layer.[68]

The design of articular cartilage is a remarkable example of the interaction between form and function. Aggregating PGs attract a large volume of water, creating an osmotic swelling pressure in the cartilage.[69] With compression of the articular cartilage (e.g., in weightbearing), swelling pressure creates tension in the hoops of the collagen network, which in turn creates an opposing force that keeps the PGs and water contained; when the swelling pressure and load on the joint reach equilibrium, the articular cartilage deformation stops.[70] Cartilage resistance to compressive force thus depends on two features: (1) a large volume of aggregating PGs and (2) an intact collagen network. During joint motion, or when the cartilage is compressed, some of the fluid content of the cartilage exudes into the joint space through pores in the outermost collagen layer (zone 1).[69–71] The fluid then flows back into the cartilage after the load is removed, carrying nutrients from synovial fluid in the joint space that supply the chondrocytes. Because hyaline cartilage is devoid of blood vessels, its nourishment is derived solely from this synovial flow of fluid; thus, the free flow of fluid is essential for the survival of hyaline cartilage.

Fluid flow is affected by the magnitude and duration of the applied force.[71] If the force is either excessive or sustained over a long period, the lack of compression and release reduces fluid flow.[69] Diminished flow also occurs with decreased loading. Hyaline

cartilage can thus undergo degenerative changes after either prolonged loading or unloading due to inadequate nutrition as fluid flow is diminished.[72] Cartilage health thus appears to depend on alternating cycles of moderate compressive forces.[73]

Damage to the superficial collagen layer through excessive frictional forces or trauma removes the meshwork that resists the swelling pressure of the PGs. Initially, the articular cartilage swells and becomes thicker as the PGs attract more water without the opposing force of the now-absent superficial collagen network.[74] Eventually, fluid movement slows, reducing cell nutrition and the ability to synthesize new extracellular matrix. Without the containment of the superficial collagen meshwork, PGs begin to escape into the synovial fluid, gradually eroding and thinning the cartilage. This is the sequence that occurs during the development of osteoarthritis.[74]

FIBROCARTILAGE

Fibrocartilage composition is different from that of hyaline cartilage. The cellular composition includes fibroblasts, fibrocytes, and, on occasion, fibrochondrocytes. When compared with hyaline cartilage, fibrocartilage has a smaller percentage of water and proteoglycans, with a greater percentage of collagen. Although type II collagen is present in fibrocartilage, type I collagen is more prevalent. The density of these collagen fibers helps trap and retain water. Fibrocartilage primarily uses the structure of the collagen to keep water in the tissue, whereas hyaline cartilage utilizes structure and chemical attraction of the PGs.[75]

Collagen fiber organization has been described as varying across regions in fibrocartilage and is thought to be related to the function of the region. For example, collagen fibers are randomly aligned in the superficial layer of the meniscus of the knee

and circumferentially arranged in the deep zone; radial fibers are observed throughout the deep zone and on the periphery.[75] Tension in the circumferential fibers resists the deformation caused by compression of the cartilage, and radial fibers are thought to prevent longitudinal splitting of the circumferential fibers.[76]

Whereas fibrocartilage has some blood supply (unlike hyaline cartilage), vascularity is still limited. Blood supply to fibrocartilage is typically found only in more peripheral regions that lie adjacent to the attachments of the vascularized joint capsule.[26,75] Therefore, diffusion through loading, as occurs in hyaline cartilage, is still necessary for exchange of nutrients and waste products for much of the fibrocartilaginous structure.



Patient Application 2–5

Tendon and Ligament Connective Tissues

Katie is a 17-year-old soccer player who tore the medial meniscus in her knee. After working in physical therapy for 5 weeks, she continues to have pain, swelling, and some catching as she flexes her knee. Discussing surgical options, her doctor tells her that she would like to repair the meniscus if possible but will only be able to do this if the tear is in the periphery of the meniscus. The central portion of the meniscus has no blood supply, which is needed for the meniscus to heal. If the tear is into the center portion of the meniscus where it cannot be repaired, the surgeon would have to remove a portion of the torn meniscus. The meniscus normally dissipates some of the weightbearing load on the articular cartilage of the knee (especially the flatter tibial plateau). Consequently, removal of a portion of the meniscus could increase the load (and deformation) of the articular cartilage, reducing fluid flow through the avascular hyaline cartilage and putting Katie's knee at risk for osteoarthritic changes over time. If the meniscal tear is isolated to the outer portion of the meniscus, a repair and healing of this slightly vascularized portion may be feasible.



Foundational Concepts 2–8

Cartilage

- Hyaline cartilage contains mostly type II collagen which, combined with a relatively high proportion of PGs and water in the extracellular matrix, will resist compressive forces.
- Fibrocartilage typically contains both type I and II collagen, indicating that the tissue is undergoing both compressive and tensile stresses.
- Hyaline cartilage is completely avascular; fibrocartilage has a limited blood supply that is restricted to areas that are adjacent to the synovial capsule.
- Avascular hyaline cartilage and the portion of fibrocartilage with limited or no vascularity depend on synovial fluid to supply nutrients and remove waste through application and removal of compression.

Bone

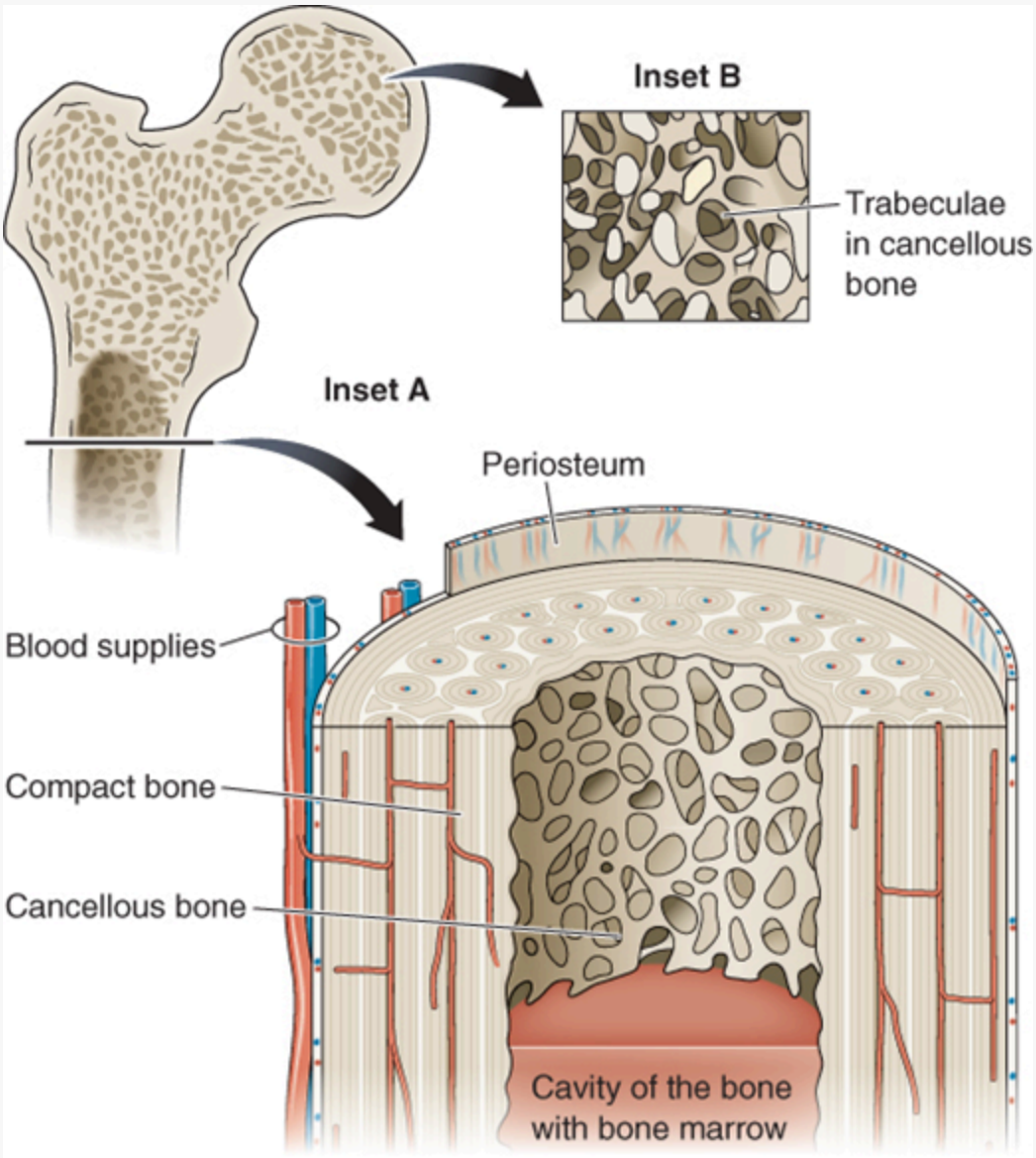
Bone is the hardest of all the connective tissues. The extracellular matrix is composed of organic fibers, mainly type I collagen, and inorganic mineral crystals, primarily **hydroxyapatite,** which fill in around the fibers.[6,11] The organic material gives bone its flexibility and tensile strength, whereas the inorganic material gives bone its compressive strength.[77] The cellular composition of bone includes fibroblasts, osteoblasts, osteocytes, osteoclasts, and progenitor cells that can differentiate into osteoblasts. The fibroblasts produce type I collagen and other extracellular matrix components. The osteoblasts are the primary bone-forming cells that are responsible not only for the synthesis of bone but also for its deposition of inorganic materials and mineralization. Osteoblasts also secrete **procollagen** (the precursor of type I collagen) into the surrounding matrix. Osteoclasts, large monocyte-derived polymorphous cells, are responsible for bone resorption. Homeostasis between absorption and deposition is fine-tuned by nutrition, hormonal status, and mechanical loading.[77]

Bone has two layers, a dense outer layer and a spongier inner layer (Fig. 2–20).[6] The inner layer is called **cancellous** (also **trabecular** or **spongy) bone**; the outer layer is

called **compact** (also **cortical) bone.** In cancellous bone, the calcified tissue forms thin plates called **trabeculae** that are laid down in line with stresses placed on the bone. It has been suggested that the loading history of the trabeculae, including loading from multiple directions, influences the distribution of bone density and trabecular orientation.[78] Increases in bone density in some areas and decreases in density in other areas occur in response to the loads placed on the bone. The cancellous bone is covered by a layer of dense, compact cortical bone, which is laid down in concentric layers.

FIGURE 2-20

Using the femur to represent a typical long bone, a cross section of the shaft of the bone (inset A) shows the dense outer layer of bone is the compact (cortical) bone, whereas the inside is cancellous (spongy or trabecular) bone. The cancellous bone shows the thin trabecular plates (inset B) that line up along stress lines. The entire outer surface of the bone (exclusive of articular surfaces) is surrounded by a thin fibrous periosteum that carries the blood vessels to and from the bone.



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Expanded Concepts 2–1

Strength of Bone

The compressive strength of compact bone is about 250 megapascals (MPa), greater than that of concrete (approximately 4 MPa) or wood (100 MPa), but less than that of steel (400 to 1500 MPa). Bone is about as strong as cast iron (140 to 300 MPa) but weighs much less. Bone is not as strong in tension as it is in compression, with its

strength dependent on density (mineralization) as well as the orientation of the trabeculae in relation to the load. If the load is applied along the long axis of the bone, the bone is much stronger.

The **periosteum** is a fibrous layer that covers the entire surface of each bone except the articular surface. Collagen fibers from ligaments and tendons blend into the periosteum, and Sharpey's fibers from attaching tendons and ligaments pass from the periosteum to deeper layers of bone. The periosteum houses cells that are precursors to osteoblasts and osteoclasts and includes many capillaries that provide nourishment. The periosteum thus acts as a reservoir for cells that are needed for growth and repair. If the periosteum and underlying bone are damaged as a result of trauma or surgery, the healing capacity of the bone will be decreased.

At the microscopic level, both cortical and cancellous bone show two distinct types of bone architecture: **woven bone** and **lamellar bone.** In woven (primary) bone, collagen fibers are irregularly arranged to form a pattern of alternating coarse and fine fibers that resembles woven material. Woven bone is young bone, formed rapidly without an underlying framework. It is often found in newborns, fracture callus, and metaphyseal regions of long bones. Lamellar bone requires an extracellular matrix framework to form, is organized into parallel layers, and is the older bone that constitutes most of the adult skeleton.

Bone is dynamic and remodels throughout life as it responds to changes in forces, such as an increased pull of tendons with strengthening, changes in weight, or stresses from new activities.[78] The change in bone shape (form) to match function, as noted earlier in the chapter, is known as Wolff's law. The application of new or increased forces causes

osteoblast activity to increase, and as a result, bone mass increases. With reduction of usual forces, osteoclast activity predominates and bone mass decreases. Internal influences, such as aging, nutritional status, metabolic processes, and disease, among others, also may affect bone remodeling because bone is interdependent with both physiological and mechanical functions.[77,79]

An imbalance between bone synthesis and resorption, in which osteoclasts break down or absorb the bone at a faster rate than the osteoblasts can remodel or rebuild the bone, results in a condition called **osteoporosis.**[80] In osteoporosis, bones have decreased mineral density (mass per unit volume) compared with normal bone and thus are weaker and more susceptible to fracture. Bone mineralization may also be decreased while cells continue to synthesize other elements of the extracellular matrix; this is called **osteopenia.** Bones that have been immobilized are frequently osteopenic.



Patient Application 2–6

June Sabato was diagnosed with multiple sclerosis at age 56. During a recent exacerbation of her symptoms, her physician placed her temporarily on glucocorticoids, a type of steroid, to decrease her symptoms. June's symptoms subsided with the steroids and June wondered why the doctor didn't give her a prescription for regular use. Her doctor explained that steroids affect the cells responsible for bone health and could make her osteoporotic. If a drug impairs osteoblast function and increases osteoclast formation, as a steroid can do, the balance between bone formation and degradation results in greater bone degradation and weaker bones that are at risk for fracture.[79]



Expanded Concepts 2–2

In Vitro Tissue Growth

Connective tissues are now being grown under laboratory conditions for later

implantation to replace damaged tissues. For example, new chondrocytes can be grown and later injected into areas of cartilage degeneration (autologous cartilage implantation).[81] The starting point is fibroblasts (often taken from damaged cartilage that can be regrown *in vitro*), usually seeded in some sort of extracellular mesh or scaffold.

Behavioral Properties of Connective Tissue

Homogeneous materials like steel display the same mechanical behavior no matter the direction in which forces are applied and are called **isotropic** materials. In contrast, heterogeneous materials, like connective tissues, behave differently depending on the size and direction of applied forces and are called **anisotropic.** Connective tissues are called *heterogeneous* because they are composed of a mixture of solid and semisolid components. The function of the tissue material as a whole depends on a combination of the properties of the different components, the varying proportions of each component in the structure, and the interactions among these components.[32,33,82]

Structural and Material Properties

Human joint components experience changing forces during ordinary daily activities and must have the ability to withstand these forces to provide support and protection. The mechanical behavior of these components dictates how different structures and tissues provide support. Mechanical behavior is described with respect to both structural and material properties. Structural properties take into consideration such characteristics as the cross-sectional area and length. Material properties provide information about the component properties of the tissue itself.

Load, Force, and Elongation

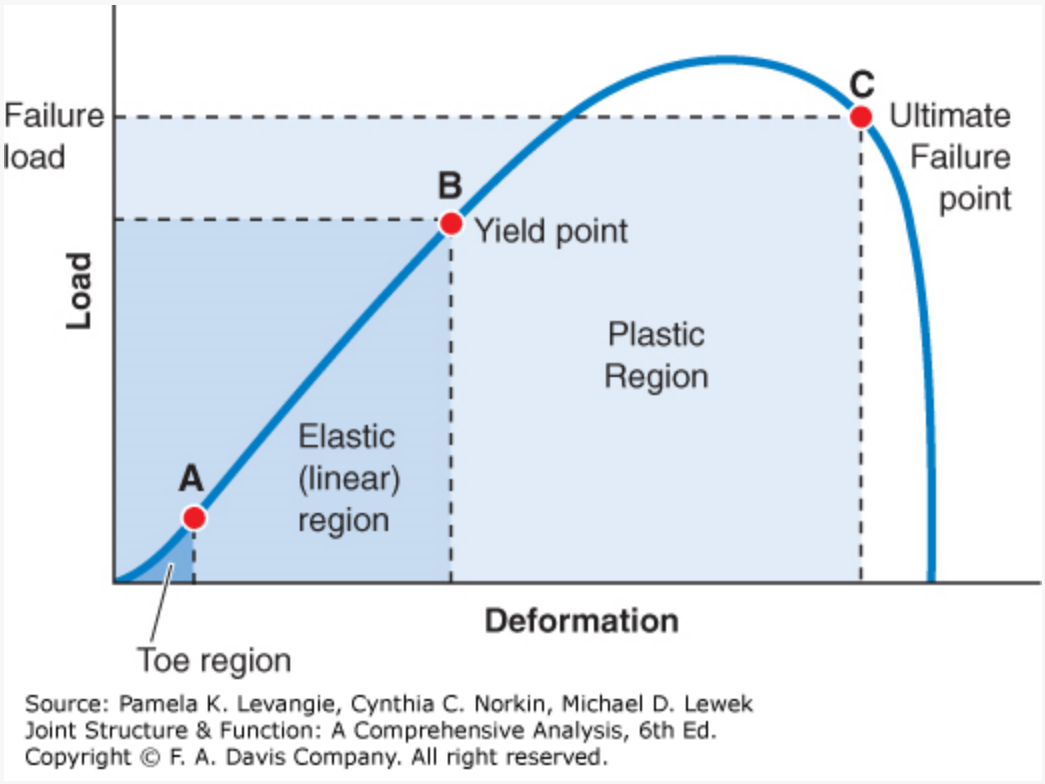
The term **load** refers to a force or forces applied to a structure. Many examples are discussed in Chapter 1, including the forces exerted by the ankle weight, the leg-press footplate, and gravity on a leg-foot segment. The magnitude, direction, and rate of force application, as well as the size and composition of the tissue, will all affect the tissue's response to load.

When a force acts on an object, it produces a **deformation** (a change in shape). A tensile load produces elongation; a compressive force produces compression. The **load-deformation curve** (Fig. 2–21) is the result of plotting the applied load (force) against the deformation, providing information about the strength properties of a particular material or structure.[83–85] The load-deformation curve of connective tissue reflects the **toe, elasticity, plasticity, ultimate strength,** and **stiffness** of the material, as well as the amount of energy that the material can absorb before it fails (given by the area under the load-deformation curve). When a load (for example, a tensile force) is first applied to the tissue, it takes very little force for the crimp (waviness or laxity) in the tissue to cause the fibers to straighten out. This is referred to as the **toe region** of the curve. After the fibers straighten out, there is an area where the relationship of the load and deformation is linear; an increase in load will produce a proportional (but not necessarily equal) increase in deformation. This is known as the **elastic region.** The slope of this linear region represents the stiffness of the material (where stiffness is the change in length over change in force). The inverse of stiffness is **compliance.** If the slope of the load-deformation curve is steep (a large increase in load results in a proportionally small increase in deformation), the material exhibits high stiffness and low compliance. If the slope of the curve is gradual (a relatively small load results in a

proportionally large increase in deformation), the material exhibits low stiffness and high compliance. If loading is confined to the elastic region, the deformation of the material will not be permanent; the structure will return to its original dimensions after the load is removed. At the end of the elastic region **(yield point),** the material will no longer return to its original state when the load is removed. Although the structure will appear to be intact after the load is removed, the material will not recover its original length or shape; the deformation is permanent. The portion of the load-deformation curve between the yield point and overt tissue failure is the **plastic region.** Any recovery of the original structure in a material loaded into the plastic range probably could only occur through the synthesis and reorganization of new tissue components as occurs in some ligament sprains. If loading continues to deform the tissue through the plastic region, the material will reach the **ultimate failure point.** The load being applied when this point is reached is the **failure load.**

FIGURE 2-21

Load-deformation curve for a connective tissue tested in tension. Initially, the crimp straightens with application of very little force (toe region). Actual stretching of collagen fibers takes place in the elastic region that is characterized by its linearity (A to B). After the elastic region ends at the yield point (B), further force application causes deformation that results in a residual change in tissue structure (plastic region). Continuation of or increase in load beyond the plastic region may cause the tissue to rupture at its ultimate failure point (C), at which point the material can no longer sustain the load.



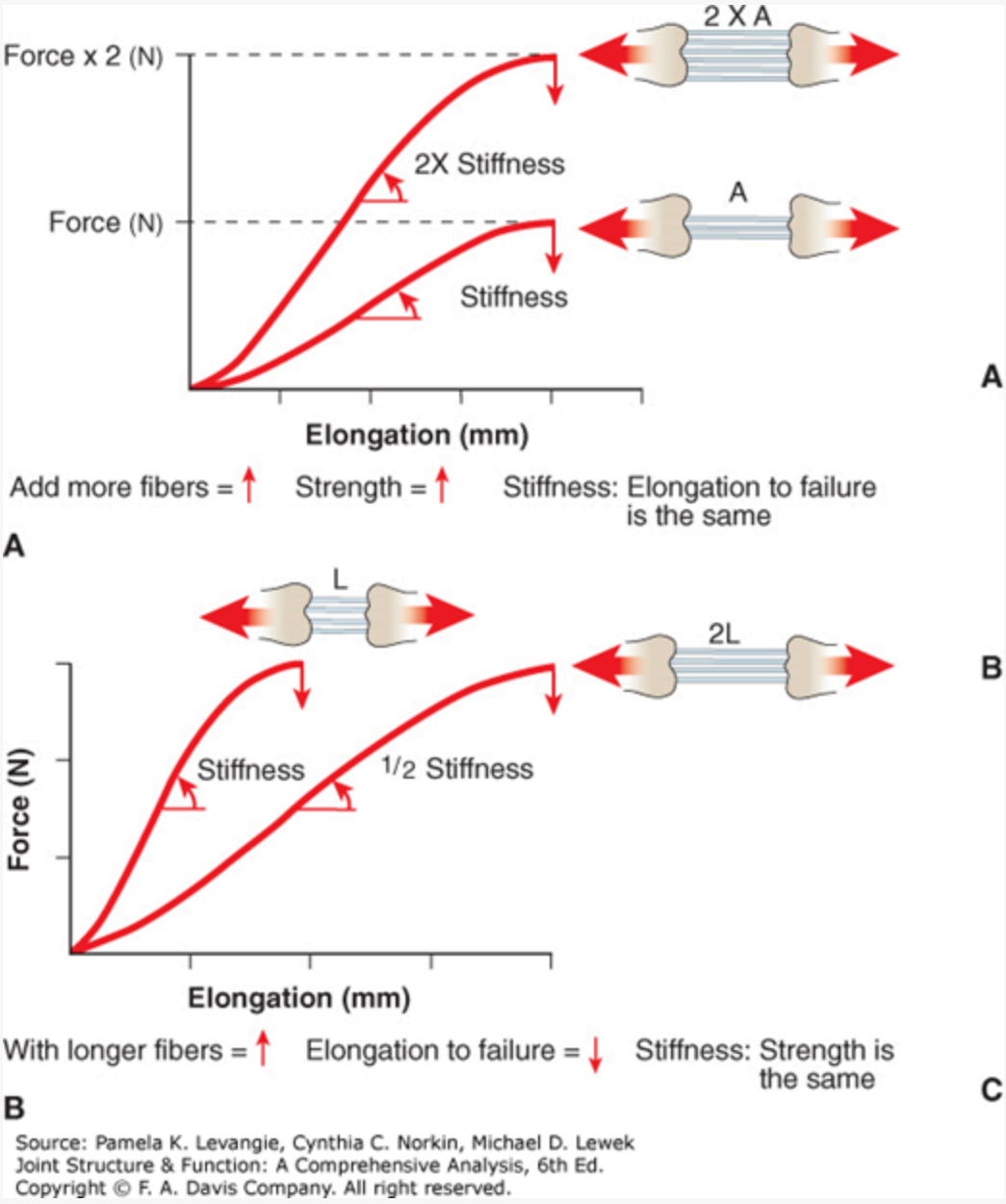
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Force values on the load-deformation curve depend on both the size of the structure and its composition. A structure with a greater cross-sectional area can withstand more force with less deformation than a structure of the same length with less cross-sectional area (Fig. 2–22A). A longer structure deforms more when a force is applied than does a shorter structure of similar cross section (Fig. 2–22B). If two tissues are composed of the same material, the tissue with the greater cross-sectional area will have more tensile strength (stiffness), and the longer tissue will be less stiff. Because the forces and deformations measured during testing are so dependent on the structural features of the tissue (cross-sectional area and length), the load-deformation curve is said to reflect the “structural properties” of the structure being tested. Tensile force is measured in newtons (N), compressive force (pressure) is measured in pascals (Pa), and elongation or compression is measured in units of length such as millimeters (mm) or

micromillimeters (μm).

FIGURE 2-22

Changes to cross-sectional area and length of the same tissue will affect its overall response to load (force). **A.** Increasing cross-sectional area means the tissues can withstand more force at any given length before failure (it is stiffer). **B.** Increasing tissue length means it can elongate farther under the same loading conditions (it is less stiff). Stiffness is reflected by the slope of the linear portion of the curves. *(Adapted from Butler DL, Grood ES, Noyes FR, et al: Biomechanics of ligaments and tendons. Exerc Sport Sci Rev 6:144, 1978; with permission from Lippincott Williams & Wilkins.)*



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Stress and Strain

When loads (forces) are applied to a structure or material, there are resulting forces generated within the material that oppose the applied forces. These forces within the material depend on the composition of the material. If we account for the mechanical effect of the tissue structure (cross-sectional area and length), testing can tell us

something about the tissue's material properties. We can control for tissue length or cross-sectional area by converting the magnitude of the applied force into a quantity that is independent of tissue size: **stress.** Stress is the force applied to a cross-sectional unit of material, and can be expressed mathematically with the following formula, where F = applied force and A = area:

$$\text{Stress} = F/A$$

Stress is expressed in units of pascals ($P = N/m^2$) or megapascals ($MPa = N/mm^2$). These are the same units used to measure pressure, which is also a force per unit area. However, pressure is produced by a compressive force applied perpendicular to the surface of the material. The percentage change in the length (or cross section) of a structure or material is called **strain.**[86,87] Like stress, strain cannot be measured directly but is calculated mathematically with the following formula, where L1 = original length and L2 = final length:

$$\text{Strain} = (L2 - L1) \div L1$$

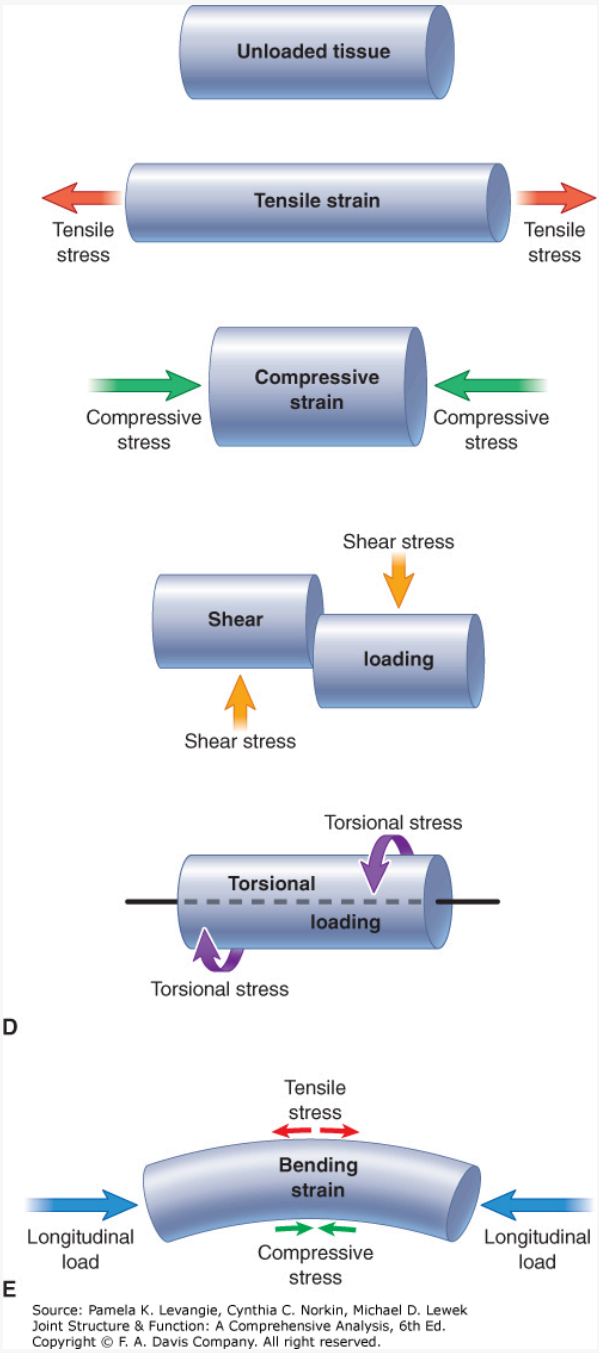
Strain is expressed as a percentage and thus has no units.

The type of strain that occurs in human tissue depends on how the load is applied to the tissue. If two applied forces act on a material along the same line but in opposite directions, they create a distractive or tensile load (tensile stress) and cause a tensile strain within the structure or material (Fig. 2–23A).[33,34] If two applied forces act in a line toward each other, they constitute compressive loading (compressive stress) and cause a compressive strain in the structure (Fig. 2–23B). If two applied forces are parallel and

are applied in opposite directions but are not in-line with one another, they constitute shear loading (shear stress; Fig. 2–23C). Forces applied perpendicular to the long axis of a structure constitute torsional loading (torsional stress; Fig. 2–23D). When longitudinal forces are applied to a curved structure such as the femur (slightly curved side-to-side and front-to-back), the bending loads create tensile stress on the convex side and compressive stress on the concave side (Fig. 2–23E).[88]

FIGURE 2-23

The various types of stresses (loads) to which connective tissues can be subjected with resulting deformation (strain). **A.** Tensile stress. **B.** Compressive stress. **C.** Shear loading. **D.** Torsional stress. **E.** Bending stress.



The types of stress and strain that develop in human tissues depend on the material, the type of load applied, the point at which the load is applied, the direction and magnitude of the load, and the rate and duration of loading.[88,89] Because stress and strain are independent of length or cross section of the material based on their

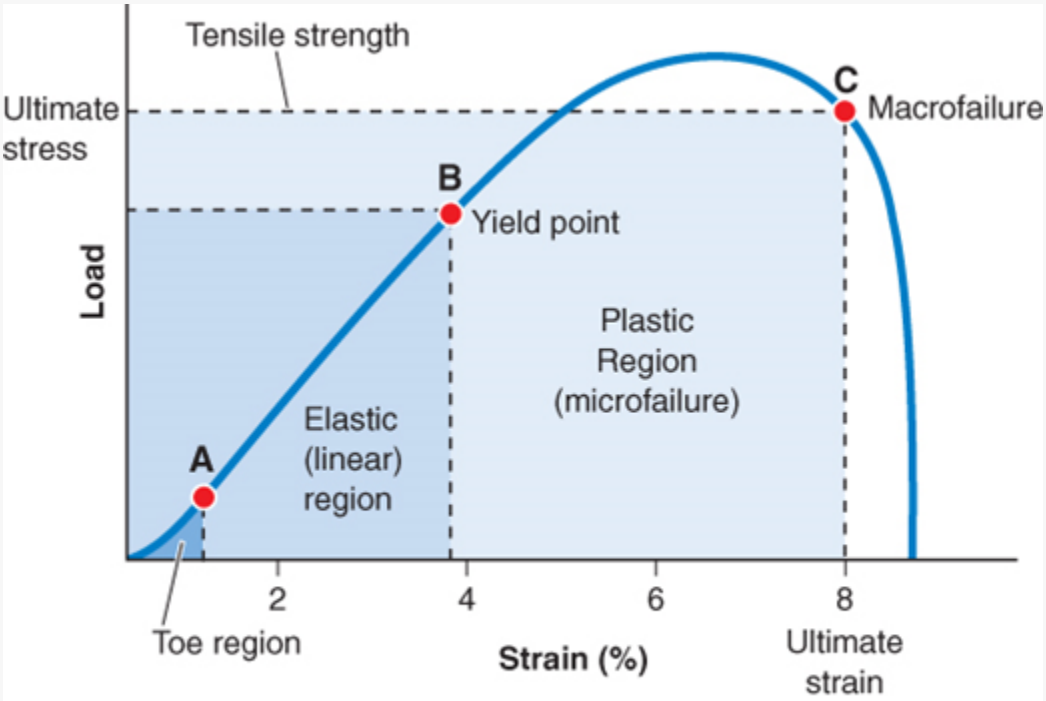
formulas, the stress-strain curve is said to reflect the **material properties** of the tissue in contrast to the load-deformation curve that reflects the structural properties of the tissue.

Stress-strain curves (Fig. 2–24) can be used to compare the strength properties of one material with that of another material or to compare the same tissue under different conditions (e.g., ligaments before and after immobilization). The stress-strain curve contains the same defining points as the load-deformation curve, but the shape of the curve and the amount of stress and strain will vary with the composition of the material. The stiffness of a material under compressive or tensile loading is represented by the slope of the linear portion of the stress-strain curve (elastic region). Stiffness (also known as **Young's modulus** or the **modulus of elasticity**) is found by dividing the change (Δ) in stress by the change (Δ) in strain for any two consecutive sets of points in the elastic range (linear portion) of the curve.

FIGURE 2-24

An example of a stress-strain curve for collagenous materials. The results are independent of tissue dimensions and thus reflect the material of which the tissue is made. The toe region is the area from initiation of the force to the beginning of fibril stretch, up to about 1% strain (A); the elastic region (A-B) is where there is a proportional (linear) increase in both stress and strain with typical activities causing about 4% strain; and B-C is the plastic region where there is microdamage to (microfailure of) the tissue structure. At the point of

ultimate stress and strain, the tissue fails completely (macrofailure) and the stress in the tissue drops to zero.



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Each material has its own unique stress-strain curve. The stress-strain information for a ligament can be used as an example to better understand the information available in a stress-strain curve (see Fig. 2–24). A minimal amount of force applied to the ligament produces deformation of the ligament in the toe region; stress is low and the strain (change in length) is typically in the 1% to 2% range. Laboratory tests determine the toe region in a ligament by measuring the force and lengthening of the ligament needed to take up the slack in the ligament's fibrils before the force is transmitted to a bone. In the elastic (linear region) of a stress-strain curve, collagen fibrils are now actually being stretched and are resisting the applied force. The slope of the linear region of the stress-

strain curve reflects the type of collagen, the fibril size, and the cross-linking among collagen molecules. When the stress is removed, the ligament will return to its pre-stressed dimensions, although this return may take some time. Ligamentous stresses that occur with normal activities typically produce up to about 4% strain.[90,91] In the plastic region of a ligament, the failure of collagen fibers (**microfailure**) begins, after which the ligament is no longer capable of returning to its original length when the force is removed. Clinical examples of microfailure include Grade I and II ligament sprains. Recovery after this level of loading involves aspects of healing such as synthesis of new tissue and cross-linking of collagen molecules to repair the damage. If force continues to be applied beyond the plastic region, the remaining collagen fibrils experience increased stress and rapidly rupture sequentially, creating overt failure (**macrofailure**) of the tissue. The terms **ultimate stress** and **ultimate strain** describe the stress and strain, respectively, just before the tissue fails. In the case of a ligament or tendon, failure in the middle of the structure through a disruption of the connective tissue fibers is called a **rupture.** If the failure occurs at the bony attachment of the ligament or tendon, it is called an **avulsion.** When failure occurs in a bone, it is called a **fracture.** Slow loading rates tend to create avulsions or fractures, whereas fast loading rates create mid-substance tears.

Each type of connective tissue is able to withstand a different percentage of strain before failure. In general, ligaments and tendons are able to deform more than cartilage, and cartilage is able to deform more than bone. However, the total deformation also depends on the size (length, width, or depth) of the structure.



Expanded Concepts 2–3

Cortical bone has high stiffness (a high modulus of elasticity) and low compliance, whereas subcutaneous fat has low stiffness (a low modulus of elasticity) and high compliance. The stiffness of cortical bone is between 17 and 25 gigaPascals (GPa), similar to that of wood. As points of comparison, metal (much stiffer than bone) has a steep elastic region indicating that large forces result in relatively little deformation; metal also has a long, flat plastic region indicating that the material deforms extensively at peak loads before failing. In contrast, glass also has a steep elastic region (although not quite as steep as metal) but has almost no plastic region because glass abruptly fails once its elastic limits are reached.[88]



Foundational Concepts 2–9

Ligament Sprains

When a substantial force (stress) is applied to a ligament, the following sequence of events will occur if the force is of sufficient magnitude:

1. The ligament appears to lengthen slightly but there will be no resistance to the increase in length within the tissue (the collagen fibrils are simply straightening out). This is the toe response.
2. The ligament will actually lengthen (collagen fibrils will stretch) as increased stress produces a proportionally greater strain of the ligament. This is an elastic response.
3. Some of the fibers within the ligament will begin to tear, disrupting (among other things) collagen crosslinking. This degree of plastic response is considered to be a Grade I sprain of the ligament.
4. Further tearing of ligamentous fibers will continue while some continue to remain intact. This response is further in the plastic range, closer to macrofailure, and is considered to be a Grade II sprain of the ligament.
5. The ligament will tear completely (rupture). Macrofailure is considered to be a Grade III sprain of the ligament.

Viscoelasticity

All connective tissues are **viscoelastic** materials: they combine the properties of

elasticity and **viscosity,** making their behavior dependent on time, rate, and history.[88,89] Elasticity (e.g., elastic response) refers to the material's ability to return to its original length or shape after the removal of a deforming load. Elasticity implies that deformation of the tissue is directly proportional to the applied forces or loads. The elastic qualities in connective tissues primarily depend on collagen and elastin content and organization.

When a material is stretched, positive work is done (work = force × distance) and energy in the stretched material increases. An elastic material stores this energy and readily returns it as negative work so that the stretched elastic material can recoil immediately to its original dimensions. For example, during many functional activities (e.g., walking, running, jumping), a lengthening (eccentric) muscle contraction stretches the attached tendon, and this elastic energy is returned during the subsequent shortening (concentric) contraction of the muscle-tendon unit and contributes to the muscle's ability to generate tension.[90–92]

Viscosity refers to a material's resistance to flow. It is a fluid property, and depends on the PG and water composition of the tissue. A tissue with high viscosity will exhibit high resistance to deformation, whereas a less viscous fluid will deform more readily. When forces are applied to viscous materials, the tissues exhibit time-dependent and rate-dependent properties. Viscosity diminishes as temperature rises or loads are slowly applied and increases as pressure increases or loads are rapidly applied.



Expanded Concepts 2–4

Viscosity

Motor oils with different viscosities are used when more or less resistance to deformation is required. When temperatures are high, the oil's resistance will be lower so a more viscous type of oil (10W-30) may be used to maintain lubrication. During the cold of winter, a less viscous oil (5W-30) will still allow the oil to deform (flow in this case) more readily and coat the engine surfaces.

Time-Dependent and Rate-Dependent Properties

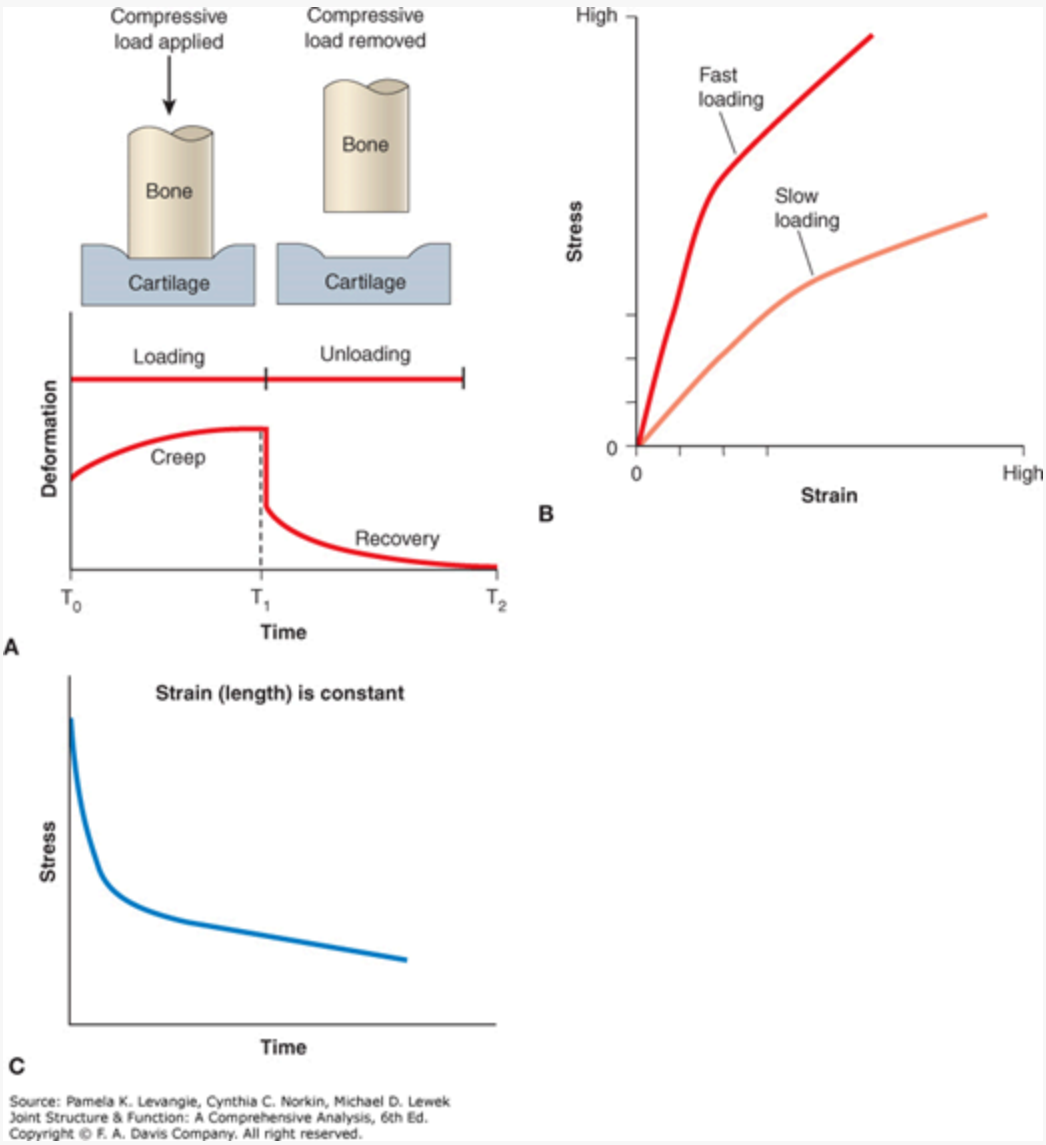
Viscoelastic materials are capable of undergoing a change in shape with application of deforming forces and returning to their original state after removal of the force. However, their viscous qualities make the deformation and return to the original shape of the material time-dependent. A viscoelastic material possesses characteristics of **creep, stress-relaxation,** and **strain-rate sensitivity.**[89]

Creep

If a force is applied to a viscoelastic tissue, there will be an immediate elastic response; if that same force is maintained after the immediate change in shape, the deformation of the material will gradually continue. This tissue response is known as creep. For example, if you hang a weight on the end of an elastic band, you will get an immediate elastic deformation. However, it will also gradually elongate further over time as the weight hangs. Connective tissues also will gradually creep (continue elongation) after an initial elastic response to a constant (e.g., tensile) load and then gradually return to the tissues' original length (recovery) after the load is removed. In a clinical setting, this might apply to stretching a shortened tissue: the clinician maintains a constant force while the tissue gradually elongates. For cartilage and bone, compressive loading is used to test creep; the depth of indentation in the material over time is measured as well as the time to recovery once the load is removed (Fig. 2–25A).

FIGURE 2-25

Time- and rate-dependent properties of dense connective tissues. **A.** Creep: when the tissue (e.g., cartilage) is loaded with a fixed force (e.g., by bone compression), the original indentation in the cartilage increases with time (T_0 to T_1); when the load is removed (T_1), the tissue gradually recovers its original shape in a nonlinear manner (T_1 to T_2). **B.** Stress-relaxation: if the tissue is stretched to a fixed length and held there (constant strain), the stress needed to maintain this length will decrease with time. **C.** If the tissue is loaded rapidly (fast loading), more force (stress) is required to deform (strain) the tissue than is needed when there is slow loading of the same tissue.



Stress-Relaxation

If a viscoelastic tissue is stretched to a fixed length (constant strain), the force (stress) required to maintain that length will decrease over time (Fig. 2–25B). This is called **stress-relaxation.** In a clinical setting, a therapist may perceive this as a reduced resistance to stretch (less force is required to maintain the same tissue length).

Strain-Rate Sensitivity

Most viscoelastic tissues behave differently if loaded rapidly versus slowly, which is called **strain-rate sensitivity.**[93] When a load is applied rapidly, the tissue is stiffer and a larger force is needed to deform the tissue than if the load was applied slowly (Fig. 2–25C). Creep and stress-relaxation also will be greater under conditions of rapid loading.

Patient Application 2–7

Angie Bagoda is a 21-year-old female who underwent surgical repair of a torn ACL. Atypically, she still had not regained full knee extension one month after surgery. A course of more aggressive stretching of the knee joint capsuloligamentous structures was recommended. Using an understanding of tissue creep, the therapist applies a manual force to Angie's knee that is just short of producing pain. That same manual force is then sustained at that same level over a period of up to 2 minutes to induce creep deformation (elongation) of the capsuloligamentous tissues. After a brief rest, the procedure is repeated with a gradual lengthening of the tissues occurring. The intervention is followed by a cold pack to minimize any inflammation that might result from breaking of cross-links within the collagen fibers of the tissue.

If Angie isn't able to tolerate a sustained force sufficient to produce tissue creep, an alternative strategy can be tried. The therapist can take the joint to the point of pain and hold the joint at that same point without attempting to stretch the tissues any further. That position of the knee is maintained until the manual force needed to hold that position diminishes and Angie feels the tension in the capsuloligamentous tissue relax (stress-relaxation). After a brief rest, Angie's knee is once again taken to the point of pain to repeat the procedure. It is likely that the capsuloligamentous tissues, after stress-relaxation, will allow slightly greater lengthening before the point of discomfort is again reached. As with the first technique, an application of ice afterwards is a reasonable precaution.

Whether using tissue creep or stress-relaxation to lengthen Angie's shortened capsuloligamentous structure, the total length change should probably be no more than 2% to 6% strain to avoid injury (microfailure) in the tight tissues.

Properties of Specific Tissues

Connective tissues change their structure or composition (and thus their function) in response to applied forces; that is, they adapt to changes in forces over time. In a tendon, changes in applied compression forces can result in changes in both the amount and type of GAGs and PGs the tissue contains;[37,38] increases in tensile forces can cause increases in type I collagen. This adaptive behavior illustrates the dynamic nature of connective tissue and the strong relationships among structure, composition, and function. The remarkable ability of connective tissues to respond to load alterations is often referred to as the **SAID principle (*s*pecific *a*daptation to *i*mposed *d*emand).**[94] The principles of tissue adaptation have been used to create a "physical stress theory" that can be used to guide one's approach to intervention during rehabilitation.[95] Although there are commonalities to adaptive responses, we will also look at the properties of specific types of connective tissues to better understand their responses to stress and strains.

Bone

Cortical bone is stiffer than cancellous (trabecular) bone; that is, cortical bone can withstand greater magnitudes of stress with less deformation (strain) than cancellous bone. However, strain in cortical bone that exceeds 2% will result in tissue failure (fracture), whereas cancellous bone can sustain strains of 75% (but at lower stresses) before failing in vivo.[88] The type of stress and how it is applied to a given tissue also makes a difference. When cortical bone is loaded in compression, longitudinal sections of the bone show the greatest strength (ability to withstand high stress before failure). In tensile testing of the femur, longitudinal sections display twice the stiffness of transverse sections. In general, cortical bone can withstand much higher compressive

stresses before failure as compared with tensile stresses, with the same being true of cancellous bone. In other words, bone can withstand greater stress (with less strain) in compression than in tension.[88]

As is true of other tissues, large loads applied to bone over short periods of time will produce high stresses and low strains (strain-rate sensitivity), whereas loads applied over longer periods of time will produce lower stresses and higher strains (creep and stress-relaxation). The physiological response of cancellous bone to regular increases in loading is hypertrophy. If loading is decreased or absent over time, the trabeculae in cancellous bone become smaller and weaker. The rate, frequency, duration, magnitude, and type of loading all affect bony adaptation. Frequent loads of low magnitude can result in a stress fracture, whereas a single application of a high load can cause failure. Bone loses stiffness and strength with repetitive loading as a result of **creep strain.** Creep strain occurs when a tissue is loaded again (and again) before the tissue has had time to return to its original shape. With each new imposition of the force, the tissue (still in a deformed state) gets pushed farther and farther toward or into the plastic region.

Tendons

Tendons exhibit creep when subject to sustained tensile loading, most often when stress is created via muscle contraction. With increases in force that bring the tendon into its elastic region, continued deformation may occur through slippage between collagen molecules within fibrils, then interfibrillar slippage between fibrils, and, finally, gross disruption of collagen fibers (plasticity or microfailure). The fibers in the tendon, however, are not perfectly parallel. The fibers that become straightened (reach the end

of toe) first may be the first to fail under increased or sustained forces—or the smaller, weaker fibrils may rupture before larger, stronger fibrils. Most normal activities load tendons in the toe region and in the first part of the linear region.

The physiological response of tendons to intermittent tension is a moderate increase in thickness and strength.[96] Differences in stress-strain curves among different tendons reflect differences in the proportion of type I and type III collagen, differences in crosslinking, maturity of collagen fibers, organization of fibrils, variations in ground substance concentration, and level of hydration. The cross-sectional area, the material, and the length of the tendon determine the amount of force that a tendon can resist and the amount of elongation that it can undergo.[41,97] Under normal conditions, tendons with greater cross-sectional areas should be able to withstand larger forces than tendons with smaller cross-sectional areas, unless the tendon with the greater cross-sectional area is composed of weaker material; thus, it is reasonable to assume that the “thick” Achilles tendon is stronger than the “thin” palmaris longus tendon.[96] Under unusual conditions, this relationship may not be true. For example, an injured tendon may be much larger in diameter than an uninjured tendon, but the injured tendon contains less collagen, smaller fibrils, and fewer crosslinks, rendering it weaker than its smaller counterpart.[97] Tendinosis in the Achilles tendon, a degenerative disorder of the tendon, is characterized by a localized thickening and collagen fiber disorganization that lowers the tendon stiffness, rendering it more susceptible to failure.[98]

Under normal conditions, the tendon is most vulnerable at either end rather than in its midsubstance. Because of the change in composition as the tendon inserts into the

bone (with resulting uneven distribution of forces within the tissue), the enthesis is a common site of degenerative changes and injury.[56,58] The myotendinous junction (MTJ) appears to be stronger than the enthesis in spite of the MTJ being a common site for muscle strains and pulls.[99] However, the MTJ depends on the interdigitation of muscle and tendon tissues for its strength so that any injury that distorts the form of the MTJ may decrease its tensile strength and predispose it to further injury.

Healthy tendons rarely rupture under typical conditions and are able to withstand large tensile forces without injury.[100] However, weakened tendons are more likely to be injured. Tendons subject to immobilization show atrophy at the MTJ, with a loss of infolding and a decrease in collagen concentration and crosslinking.[99–101] Exposure to corticosteroids, nutritional deficiencies, hormonal imbalance, dialysis, chronic loading into the high linear region of the stress-strain curve with inadequate time for recovery, and sudden large loads may predispose the tendon to injury even at physiological levels of loading (levels typically experienced by the tissue); in other words, the same load now produces more deformation in the tendon.

Tendons adapt readily to changes in the magnitude and direction of loading. Tendons subjected to continual compressive forces will alter their composition to what can resemble cartilage, with a resulting decline in tensile strength.[102] Conversely, tendons subjected to tensile loads, especially physiological loads over long periods of time, will increase in size, collagen concentration, and collagen crosslinking.[103–105] Progressive loading programs are successfully used to treat tendon dysfunction, presumably through inducing changes in tendon composition.[97,106-109]

Ligaments

As noted previously, tendons and ligaments are very similar mechanically. The physiological response of ligaments to intermittent tension, like that of tendons, is an increase in thickness and strength.[83,84] Ligaments must withstand forces in all directions, so they have a more varied orientation of collagen fibrils that makes them slightly less resistant to tensile stress but better able to function within a range of load directions without being damaged. This is another example of form (in this case, collagen fibril orientation) following function.

Cartilage

Three forces interact in cartilage to resist applied load: (1) stress developed in the fibrillar portion of extracellular matrix (type II collagen), (2) swelling pressures developed in the interstitial fluid (PGs and water), and (3) frictional drag resulting from fluid flow through the extracellular matrix.[49] Compression of cartilage reduces the volume of the cartilage and increases the pressure, causing outward flow of interstitial fluid. Fluid flow through the extracellular matrix creates frictional resistance to the flow within the tissues (frictional drag). Exudation of fluid occurs rapidly at first, causing a concomitant rapid rate of deformation. Subsequently, fluid flow and deformation gradually diminish and cease when compressive stress in cartilage balances the applied load.[68,69,93] Magnetic resonance imaging (MRI) has made it possible to study changes in cartilage volume and thickness in the joints of living subjects. In an MRI study of the knee joints of eight volunteers, Eckstein and colleagues[110] found that up to 13% of the fluid was displaced from the patellar cartilage 3 to 7 minutes after exercise (50 knee bends).

The tensile behavior of cartilage collagen is similar to that of ligaments and tendons but is caused in different ways. Tensile stresses within cartilage are created in the superficial collagen network as the compressed PGs and water push against the collagen fibers.[63] The nonlinear behavior in the toe region of the stress-strain curve for cartilage is thought to be caused by the drag force between the collagen meshwork and the PGs rather than the straightening of collagen fibers seen in tendon and ligament. In the elastic region, cartilage collagen fibers become taut and are stretched just as in ligament and tendon fibers. Cartilage specimens from the different zones of cartilage (see Fig. 2–19) show differences in tensile behavior, presumably due to differences in the orientation of the collagen fibers among the zones.[63] Cartilage resistance to shear depends on the amount of collagen present because PGs provide little resistance to shear. Shear stresses are apt to develop at the interface between the calcified cartilage layer and the subchondral bone.



Foundational Concepts 2–10

Effects of Altered Loading on Connective Tissue

- Connective tissues become weaker and lose their typical structure if they are not at least periodically loaded (stressed).
- Changes with decreases in typical loading patterns occur rapidly.
- Recovery of normal structure and function requires gradual and progressive loading.
- Loads should be tailored to the type of target connective tissue.

General Changes With Disease, Injury, Immobilization, Exercise, and Overuse

Each part of a joint has one or more specific functions that are essential for the overall

performance of the joint. Therefore, anything that affects one part of a joint will disrupt the overall function of the joint. Likewise, anything that affects joint motion or joint stresses will affect all the structures that constitute that joint. This relationship between form and function is essential for therapists to remember during rehabilitation after injury. For example, when a bone is broken, the fracture may be the primary injury that dictates subsequent treatment, but lack of motion and decreased loading needed for bone healing also will have secondary effects on cartilage, ligaments, joint capsule, tendons, and muscles. The ideal rehabilitation protocol considers the behavior of all the affected structures and includes interventions tailored to induce positive adaptations in each structure. This means understanding the time course and nature of the adaptation of each tissue to altered loading conditions.

Complex joints are more likely to be affected by injury, disease, or aging than are simple joints. Complex joints have more parts and are subject to more wear and tear because the complexity is typically an attempt to maximize gain in ROM while minimizing the loss of stability. The function of the complex joints depends on a number of interrelated factors. For example, the capsule must produce synovial fluid, which must be of the appropriate composition and of sufficient quantity to lubricate and nourish the joint. The hyaline cartilage must be smooth enough that the joint surfaces can move easily, yet permeable enough to receive nourishment from the joint fluid. The cartilage must undergo periodic compressive loading and unloading to facilitate movement of the fluid, and the collagen network must be intact to contain the fluid attracted to the PGs. The ligaments and capsules must be strong enough to provide sufficient support for stabilization and yet be flexible enough to permit normal joint motion without excessive constraints. Tendons must be able to withstand the forces generated by

muscles as they produce movement.

Disease

The general effects of disease, injury, immobilization, and overuse may be illustrated by using the normal function of a joint structure as a basis for analysis. For example, when the synovial membrane of a joint is affected by a disease like rheumatoid arthritis, the production and composition of the synovial fluid may change, thus affecting joint lubrication. The disease process and the changes in joint structure that occur in rheumatoid arthritis involve far more than just synovial fluid alteration. Osteoarthritis, which may be genetic or mechanical in origin, is a different disease process where the cartilage is the primary target. With the erosion and splitting of the cartilage that occurs, friction is increased between the joint surfaces, thus further increasing the erosion process.

Injury

Joint support is likely to decrease after injury to one or more of the joint components. If a collapsible stepladder (see Fig. 2–1) has an unstable brace between the two sides of the ladder, damage and disruption of function may occur as a result of instability. If a heavy load is placed on the damaged ladder, the two sides will separate excessively under the compressive load and the ladder may collapse, further damaging other components of the ladder, not to mention the person on the ladder.

The effects of decreased support in a human joint are similar to those in a collapsible stepladder. If a joint ligament is torn, separation of the bony surfaces can occur and may result in wobbling or a deviation from the normal alignment of the bony components. Changes in alignment create abnormal joint opening on the side where

the ligament is torn. Other ligaments, tendons, and the joint capsule may be subjected to increased loading, leading them to become excessively stretched or compressed and unable to provide protection. The intact side of the joint may be subjected to abnormal compression during motion or weightbearing. In canine experiments in which an unstable knee joint is produced by transection of the anterior cruciate ligament, morphologic, biochemical, biomechanical, and metabolic changes occur in the articular cartilage shortly after transection.[111] Later, the articular cartilage becomes thicker, shows fibrillation, and develops osteophytes. The cartilage shows much higher water content than in the opposite knee, and the synovial fluid content of the knee is increased. In addition, a sharp increase in bone turnover occurs, as does a thickening of the subchondral bone.[111] According to Van Osch and colleagues, joint instability is a well-known cause of secondary osteoarthritis involving the knee joint.[112] The recognition that joint injuries, especially ligament injuries, lead to osteoarthritis suggests greater efforts are required to prevent and treat sport injuries in young people.

Immobilization (Stress Deprivation)

Any process or event that disturbs the normal function of a specific joint structure will start a chain of events that eventually affects every part of a joint and its surrounding structures. Immobilization is particularly detrimental to joint structure and function and may be externally imposed by a cast, reduced weightbearing (e.g., bedrest), or denervation.

Immobilization also may be self-imposed as a reaction to pain and inflammation. A joint injury that results in inflammation and swelling will assume a loose-packed position (where the capsuloligamentous structures are under least tension) to

accommodate the increased volume of fluid within the joint space. This position may be referred to as the **position of comfort** because pain is decreased in this position. Each joint has a position of minimum pressure and maximum capsular volume. For the knee and hip joints, the position of comfort is between 30° and 45° of flexion; for the ankle joint, the position is at 15° of plantar flexion.[32] If the joint is immobilized for a few weeks in the position of comfort, the joint capsule will adapt (shorten and lengthen), and contractures[113] can develop in the surrounding soft tissues. Consequently, resumption of a normal range of joint motion upon remobilization will be difficult.

Effects on Ligament and Tendon

Ligaments and tendons adapt to decreased load with reduced collagen content and reduced crosslinking among collagen molecules, although the overall collagen fiber size may remain the same; the tissue thus weakens. Resumption of previously normal loading may cause damaging stress and strain.[83,84] The musculotendinous junction of a tendon loses its interdigitating structure when not loaded, which makes it weaker.[99] The time course of these adaptations is fairly rapid. Ligaments and tendons show a 50% decrease in tensile strength and stiffness after 8 weeks of immobilization.[83,84] It is assumed that ligaments and tendons eventually recover their mechanical properties, but the time course of this recovery appears to be slow, with the total time for recovery unknown. In general, the time course for the loss of mechanical properties occurs over weeks, whereas recovery can take 12 to 18 months or more.[85] Gradual reloading is necessary to restore tendon and ligament strength, with sufficient time between loading episodes to allow time for the tissue to return to its original shape through both elastic recoil and creep recovery.

Effects on Articular Surfaces and Bone

The effects of immobilization are not confined to the surrounding soft tissues but may also affect the articular surfaces of the joint and the underlying bone. Biochemical and morphological changes may include: proliferation of fibrofatty connective tissue within the joint space, adhesions between the folds of the synovium, atrophy of cartilage, regional osteoporosis, weakening of ligaments at their insertion sites as a result of osteoclastic resorption of bone and Sharpey's fibers, a decrease in the PG content, and an increase in the water content of articular cartilage.[49,114,115] Thinning and softening of the articular cartilage occurs, and deformation under compressive test load increases up to 42%. As a result of changes in joint structures brought about by immobilization, decreases may be evident in the ROM available to the joint, the time between loading and failure, and the energy-absorbing capacity of the bone-ligament complex. Swelling or immobilization of a joint also inhibits and weakens the muscles surrounding the joint.[116–119] Therefore, the joint is unable to function normally and is at high risk of additional injury.[120] A summary of the possible effects of prolonged immobilization is presented in Table 2–3.

Table 2-3

Effects of Increased and Decreased Load on Connective Tissues



Foundational Concepts 2–11

Deleterious Effects of Immobilization

Joint structures will undergo potentially substantive changes during immobilization:

- Bone: weakened, decreased collagen and mineral content (osteopenia)
- Capsule: shrinking, increased resistance to movement (stiffer)
- Ligament: decreased crosslinks, decreased tensile or directional strength (weaker)
- Tendon: decreased crosslinks, disorganization of collagen, decreased tensile strength (weaker)
- Muscle: loss of sarcomeres in series, decreased contractile proteins (actin/myosin) resulting in reduced force production
- Cartilage: swelling, decreased PG concentration (softer, weaker)

These changes occur within 8 weeks, but recovery may take 18 months or longer.



Patient Application 2–8

Jamelle Watkins fractured his right femur in a car accident. Once the fracture was stabilized surgically with internal fixation, he was given crutches and told not to bear weight on his leg. Although fracture healing must take place, this type of immobilization is concerning for the health of the joints in his injured leg. Without proper cartilage loading, hyaline cartilage will swell, which will strain the superficial collagen network, reduce nutrient diffusion into the cartilage, and make the superficial collagen layer more susceptible to damage. Jamelle can be instructed in activities that will allow for some cartilage loading across the joints via muscle contraction (causing compression across joint surfaces) as well as joint ROM. Joint motion alternately compresses and unloads the joint surfaces, maintaining cartilage nutrition and helping to maintain capsule mobility. Once Jamelle is able to put weight on his leg, the probable changes in hyaline cartilage (as well as other structures) should be taken into consideration and weightbearing should be resumed gradually. Jamelle was lucky that he did not require full immobilization of his leg. In cases when joint motion and weightbearing need to be restricted for healing, it is preferable that a removable brace be used rather than a cast so that the patient can remove it for ROM exercise (presuming patient compliance with restrictions can be assumed). When non-weightbearing and joint immobilization is necessary for bone healing, a postoperative cast is often changed for a cast-brace at approximately 3 to 4 weeks once some new bone has started to form.

Recognition of the adverse effects of immobilization has led to the development of several strategies to help minimize the consequences. These include: (1) use of continuous passive motion (CPM) devices after joint surgery, (2) reduction in the duration of casting periods after fractures and sprains, (3) development of dynamic splinting devices to allow joint motion while preventing unwanted motion that may damage healing structures, (4) use of graded loading after immobilization, and (5) extension of the recovery period to months rather than days or weeks. A continuous passive motion device can move joints passively and repeatedly through a specified portion of the physiological ROM. The speed and range of the movement can be controlled. Continuous passive motion devices produce joint motion under low loading conditions, which in turn produces medium-frequency alternating compression that may stimulate cartilage formation. It is easier to control loading with these devices than with active movements and, therefore, easier to avoid the potentially deleterious compressive-tensile stresses and strains produced by active muscle contractions where these are not desirable. Continuous passive motion was shown to prevent some of the tendon weakening that occurs during immobilization, though not enough to maintain normal tissue strength.[121]

Exercise

All tissues appear to respond favorably to gradual progressive loading by adapting to meet increased mechanical demands. Exercise influences cell shape and physiological functions and can have a direct effect on matrix alignment. The response to exercise varies among tissues and depends on the nature of the stimulus, including the amount, type, and frequency of loading. The mechanism of connective tissue response to exercise appears to involve cells' detecting tissue strain and then modifying the type

and amount of tissue the cells produce. The amount, type, and frequency of deformation are important. Low-frequency compressive loading will increase cartilage formation, whereas higher frequencies can enhance bone synthesis. Higher magnitude or sustained compressive loading will induce fibrocartilage formation, whereas tensile loads induce tissue formation resembling that found in a tendon or ligament. According to Mueller and Maluf's Physical Stress Theory,[95] maintaining the normal mechanical state of connective tissues appears to require repetitive loading beyond a certain (albeit often unknown) threshold level. Below this threshold, the immobilization changes previously described occur rapidly.

Bone Response to Exercise

Bone deposition is increased with weightbearing exercise and in areas of bone subjected to increased muscle force.[122,123] This response of bone has been known for over 100 years, and exercise is now used as a therapeutic intervention to prevent bone loss.[124] A systematic review showed that in 11 of 16 studies, postmenopausal women showed improvements in bone density with either exercise or exercise plus calcium or estrogen.[123] Bone formation appears very sensitive to strains as well as (or perhaps rather than) the magnitude of the applied load. Very low magnitude high-frequency vibration has been shown to increase trabecular bone formation by 34%.[125] This suggests that even very low loads, well below the threshold for physical damage, may increase bone density. Rubin et al. proposed that these far smaller high-frequency (10 to 30 Hz) mechanical signals that continually barrage the skeleton during longer term activities such as standing and walking are what regulate skeletal architecture.[125–127] Even short durations of loading are effective; just 10 minutes of low-load, high-frequency stimulation has been shown to prevent bone loss induced by disuse.[125]

Lanyon suggested that the asymmetrical strains during normal load-bearing activities create an ever-changing strain distribution, with that variety inducing bone adaptation; novel strain distributions in particular result in an osteogenic response.[128] He further suggested that exercise regimens designed to control bone architecture can usefully capitalize on this feature of the adaptive (re-)modeling response, with each exercise session including as many novel strain distributions as possible—preferably involving high peak strains and strain rates that are repeated daily (or on alternate days).

Cartilage Response to Exercise

The response of cartilage to immobilization has been described, but its response to increased physiological loading is largely unknown. The health of hyaline cartilage depends on the application and removal of compressive loads. Chondrocytes are directly connected to their microenvironment through attachments between cell membrane proteins and collagen fibrils, and mechanical forces are transduced into intracellular synthetic activity. The mechanisms by which this transduction occurs and the magnitude and frequency of the loading that will optimize cartilage structure are not yet known. This is an area of active research as cartilage injuries heal very poorly; use of transplanted material to repair cartilage defects is also under active investigation. Since Salter's work,[129] it is well known that motion enhances tissue formation in cartilage defects, but the tissue formed is fibrocartilage rather than hyaline cartilage. Unlike fibroblasts (in bone, ligament, and tendon), chondrocytes do not readily migrate to and repair areas that have been injured. Defects that extend to subchondral bone are thought to have better healing potential because mesenchymal cells (from bone marrow) can differentiate into chondroblasts under the right loading conditions, hence the use of drilling (creating an artificial defect in the subchondral

bone) to surgically treat osteochondral defects.[130] There are no quantitative data available about changes in human articular cartilage after immobilization or exercise, although MRI shows promise in this regard. It appears that cyclic low-magnitude, low-frequency (less than 1 Hz) compressive loads may be best for inducing or maintaining cartilage structure.

Tendon Response to Exercise

Tendons respond to increased tensile loads by increasing collagen concentration, collagen crosslinking, tensile strength, and stiffness. Woo et al. showed that after 12 months of physical extensor training, the extensor digitorum tendons of swine increased their weight, strength, collagen content, and stiffness to match the normally stronger flexor digitorum longus, which did not change in response to the same program.[105] Biochemical changes occurred in chicken Achilles tendons after strenuous intermittent running, with increased collagen synthesis, crosslinking, tensile strength, and stiffness, although tendon size and weight did not change.[131,132] Chronic increased loading causes tendon hypertrophy and increased crosslinking.[132–134] In other words, both structural and material changes can take place. Interestingly, exercise also appears to offset some of the changes that occur in connective tissue with age.[135] Progressive tensile loading has been used successfully to treat chronic tendon disorders (thought at one time to be counterproductive) under the assumption that the tendon adapts to the increased loads.[106-109,136]

Ligament Response to Exercise

The effects of exercise in preventing negative and promoting positive changes in ligaments undergoing repair have been well demonstrated, but the effects of exercise on normal ligaments are less clear.[137–139] Recovery of normal ligament structure and

mechanics after immobilization is a slow process that can take months. It appears that exercise may speed this process, but the volume of loading and the time course of the adaptations are not known.

Expanded Concepts 2–5

Exercise can help facilitate recovery of tissue after immobilization. The types of exercise that may facilitate tissue recovery in the lower extremity include:

- Bone: walking, standing with weight shifts, bouncing, on/off pressure
- Cartilage: moderate loads through available ROM, walking, isometrics
- Ligament: gentle, progressive tensile stress, changes in stress directions
- Tendon: progressive tensile loading, strength training
- Muscle: progressive tensile loading at various speeds (recruit different motor units) and lower load exercise to fatigue (induce metabolic adaptation)
- Capsule: repeated exercise throughout physiological ROM

Although isometric muscle exercises (muscle contraction without joint motion) can be used even during periods of immobilization, it is recommended that large loads be avoided during that period.

Foundational Concepts 2–12

Connective Tissue Adaptation

- All connective tissues will adapt to increased load through changes in structural or material properties (form follows function).
- Newly imposed loads must be gradual and progressive; as the tissue adapts to the new loading conditions, the load must change to induce further adaptation.
- The type of connective tissue formed will match the type and volume of the load:
  - Compression: cartilage or bone
  - Tension: ligament or tendon
- Optimization of load volume and frequency, and the nature of human tissue adaptation, remain largely unknown as of yet.

Overuse

Although it is readily recognized that immobilization has negative consequences, both sustained and repetitive loading of joint structures also may have adverse effects. Damage can occur in one of two ways: (1) sudden application of large loads, and (2) repeated or sustained application of low loads. The former are non-physiological loads that create large stresses and strains, thus precipitating failure of the tissue on a microscopic or macroscopic scale (e.g., sprains, tendon ruptures, bone fractures). Physiological loads that are typical in magnitude but sustained or repeated while creep is occurring may also result in tissue plasticity or failure. Recovery of normal tissue shape after a load is removed takes an as yet unknown time. When a structure is subjected to imposition of a new load before it has recovered (returned to its original shape), the tissues may enter the plastic range and undergo microfailure. Ligaments, or capsules, subjected to constant tensile loading may undergo permanent changes in length. For example, after loss of the ACL, increased load on the posterior knee joint capsule may cause plastic deformation (permanent stretch), leading to increasing knee hyperextension. Cartilage subjected to constant compressive loading will creep and may show evidence of permanent deformation if the load continues long enough. Cell death may occur with rigid sustained pressure at focal points on the cartilage, with permeability and fluid flow further decreased.[62]

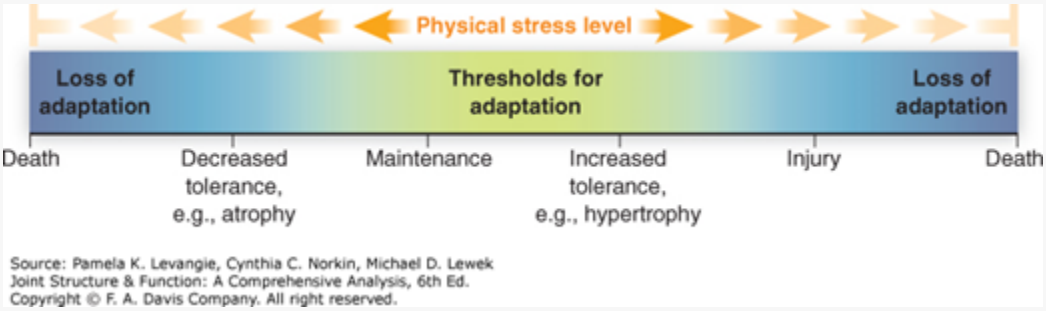
Joints and their supporting structures subjected to repetitive loading thus may be injured because they do not have enough time to recover before they are subjected to another loading cycle. An injury resulting from this type of repetitive strain loading may be referred to as an **overuse injury** or **syndrome, repetitive motion disorder,** or **repetitive strain injury.**[7] These disorders have been identified in athletes, dancers,

farmers, musicians, and factory and office workers. Such injuries appear to affect a greater proportion of women than men for unknown reasons.[140] Hart and associates hypothesized that intrinsic gender differences may exist in the regulation of connective tissue structures, perhaps related to hormone levels that fluctuate in women during pregnancy and across menstrual cycles.[140] Tendon biopsy material from human subjects undergoing surgery for repetitive motion disorders shows an inflammatory process in some tendons and a degenerative process in others.[141] In view of the findings to date, it appears that simple tissue fatigue is not a sufficient explanation for the cause of repetitive motion disorders and that additional research is needed to determine all of the factors involved in the cause, effect, prevention, and treatment of repetitive motion disorders.

Mueller and Maluf's physical stress theory[95] (Fig. 2–26) reflects the concept that physical stress is required for maintenance of tissue health and that tissues have at least limited ability to adapt to changes in stress (interaction of form and function). Tissues may effectively respond to increased or new stresses through adaptations such as hypertrophy, whereas decreased stresses will lead to decreased stress tolerance (e.g., atrophy). With sufficient exposure to excessive stress or to stress reduction, tissue failure (death) may occur. The limitation to this stress theory is that the margins between adaptive and non-adaptive stresses are unknown and, as we have already seen in this chapter, are dependent on innumerable interrelated factors across and within different types of connective tissue.

FIGURE 2-26

The responses to different levels of loading (physical stress level) on connective tissue structures based on Mueller and Maluf's physical stress theory.[95]



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
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Foundational Concepts 2–13

Chronic Overuse Injuries

- Chronic overuse injuries may involve repeated or sustained loads while the tissue is still in a deformed state.
- Recovery time, not load size, may be the critical variable in susceptibility of tissues to overuse injury.
- The roles of systemic influences (e.g., hormones, nutrition) and neurophysiological factors (e.g., referred pain, focal dystonia) in repetitive injuries remain to be explored.

Summary

- This chapter presents the elementary principles of joint design, a classification system for human joints, and an introduction to the materials found in human joints and their properties, as well as the effects of disease, immobilization, and overuse on joint structures.

- The health, strength, and function of joint structures depend on a threshold amount of stress and strain; this threshold can move up or down, depending on the state of the tissue.

- Nutrition and health of cartilage and bone depend on joint movement and muscle contraction. Cartilage nutrition depends on joint movement through a full ROM to ensure that the entire articular cartilage receives the nutrients necessary for survival.

- Stresses and strains are necessary to ligaments and tendons to maintain or increase their strength. Controlled loading and motion applied early in the rehabilitation process stimulate collagen synthesis and help align collagen fibrils. Bone density and strength increase following the stress and strain created by muscle activity and weightbearing. In contrast, bone density and strength decrease when stress and strain are absent.

- It appears that tissues have a threshold below which they atrophy and above which they become injured (see Fig. 2–26). This threshold can be modified through progressive load adjustment so that the tissue can withstand changes in forces accompanying new functional activities.

- The inadequacy of cartilage repair mechanisms and the slow recovery of bones, ligaments, and tendons suggest that the prevention of injury to recovering joint structures through the avoidance of excessive reloading is crucial. Gradual, progressive loading is the ideal. The therapist must skillfully load the tissues with the appropriate direction, magnitude, and frequency of forces to prevent

weakening or to induce adaptation.

In subsequent chapters, the specific structure and function of each of the major joints in the body will be explored. Knowledge of the basic elements of normal joint structure and function and understanding the changes that function can induce in structures (and vice versa) are necessary to appreciate the complex nature of human joints, as well as for identifying abnormal joint function, and analyzing the effects of injury, disease, or aging on joint structure and function.

Study Questions

1. Is a condyloid joint uniaxial, biaxial or triaxial? How many degrees of freedom does it have?
2. In the human body, under what conditions will joints form a closed chain? What are the implications of a closed chain on function?
3. What are the common elements of all synovial joints and what is the purpose of each?
4. What is the role of a meniscus or disk at a joint?
5. Describe the osteokinematics and arthrokinematics that take place when the convex end of the lever moves on a fixed concave surface.
6. Define the motions of spin, slide, and roll as they relate to intra-articular joint motions. As the femur flexes on a weightbearing tibia, which of these motions

take place?

7. Describe what it means for a joint to be close-packed and loose-packed. In which position is a joint more stable and why?

8. Define the terms hypomobility and hypermobility as each refers to human joint motion.

9. What are the common components of which all connective tissues are composed? In what way do these components differ among the primary connective tissues?

10. In what part of connective tissues do proteoglycans (PGs) belong? What is their function?

11. What factors affect the elasticity and point of failure of a tissue?

12. What are the ways by which capsule, ligament, and tendon attach to bone? Are these tissues more likely to fail at their attachments or at their midsubstance? Why?

13. Describe the differentiating components and arrangement of fibers in the layers (zones) of hyaline cartilage as well as the purpose of each in supporting the function of hyaline cartilage.

14. Which of the following are disruptive to proper nutrition for normal articular cartilage and why: prolonged loading, occasional loading, rare loading?

15. What differentiates bone from other connective tissues and cancellous bone from cortical bone?

16. Define stress, strain, stiffness, yield point, plastic region and ultimate failure in connective tissues.

17. What difference if any is there between a load-deformation curve and a stress-strain curve?

18. Differentiate between crimp, elasticity, and creep responses in viscoelastic materials.

19. What happens to viscoelastic tissue when the rate of loading is increased? What happens when the tissue is heated? How does a thicker tissue respond to stress in comparison to a thinner tissue of the same composition?

20. Describe what happens to each of the following tissues when they fail: Ligament, tendon, cartilage, bone.

21. What are the general effects of immobilization on connective tissues? What are the implications for these changes when remobilizing the tissues?

22. Describe how overuse may affect connective tissues.

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Chapter 3: Muscle Structure and Function

Gary Chleboun

Introduction

The skeletal muscles, like the joints, are designed to contribute to the body's need for both mobility and stability. Muscles serve a mobility function by producing or controlling the movement of a bony lever around a joint axis; they serve a stability function by constraining extraneous movement of joint surfaces and through approximation of joint segments. Without muscle function, the body is incapable of either supporting itself against gravity or producing motion.

Human movement is a complex interaction of muscle function and joint lever systems under the control of the nervous system. Clinicians routinely evaluate the muscle function of patients to determine its status and to formulate appropriate interventions to help the patient regain or enhance function. Understanding muscle function begins with a clear picture of the muscle's structure, from the contractile proteins within each muscle fiber to the organization of the fibers in the entire muscle. After examining muscle structure, we examine the basic mechanical properties of muscle fibers, the muscle in its entirety, and groups of muscles. To complete our understanding of muscle function, we will analyze the function of muscles working across joints to attempt to produce the intricate movements we use for daily activities, work, sport, and play. Patient applications will be used to apply principles related to basic muscle structure and function to selected common muscle injuries.

Elements of Muscle Structure

Skeletal muscles are composed of muscle tissue (contractile) and connective tissue (noncontractile). Muscle tissue has the ability to develop tension in response to chemical, electrical, or mechanical stimuli. Connective tissue elements, on the other hand, develop tension in response to passive loading.[1] Properties of the contractile and noncontractile tissues and the ways in which they are interrelated give muscles their unique characteristics.

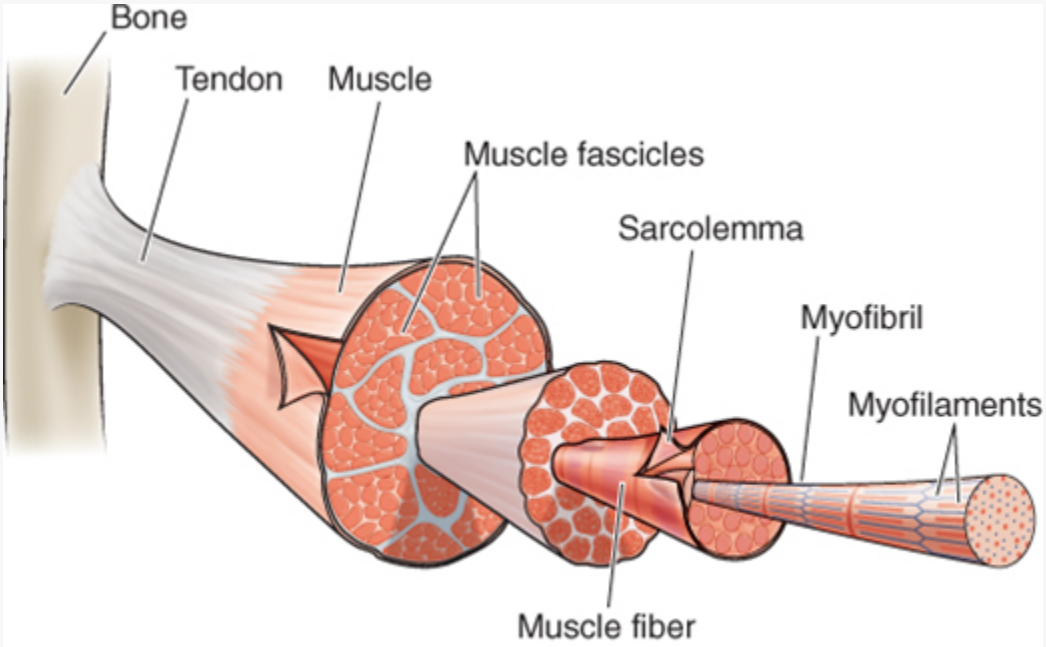
Composition of a Muscle Fiber

Contractile Proteins

A skeletal muscle is composed of many thousands of **muscle fibers** that are grouped into **fascicles.** Each muscle fiber in a fascicle is composed of numerous **myofibrils,** and each myofibril is composed of numerous stacked myofilaments (Fig. 3–1). The muscle also includes a number of different connective tissue elements that we will return to later. The arrangement, number, size, and type of the muscle fibers may vary from muscle to muscle, but every fiber is a single muscle cell that is enclosed in a cell membrane called the **sarcolemma.**[2–4] Like other cells in the body, the muscle fiber is composed of cytoplasm, which in a muscle is called **sarcoplasm.** The sarcoplasm of the cell (fiber) contains myofibrils that are the contractile structures of the muscle fiber as well as non-myofibrillar structures such as ribosomes, glycogen, and mitochondria that are required for cell metabolism.

FIGURE 3-1

Composition of a muscle fiber. Groups of muscle fibers form bundles called fascicles. The muscle fiber is enclosed in a cell membrane called the sarcolemma. The muscle fiber contains myofibrillar structures called myofibrils. The myofibril is composed of thick myosin and thin actin myofilaments.



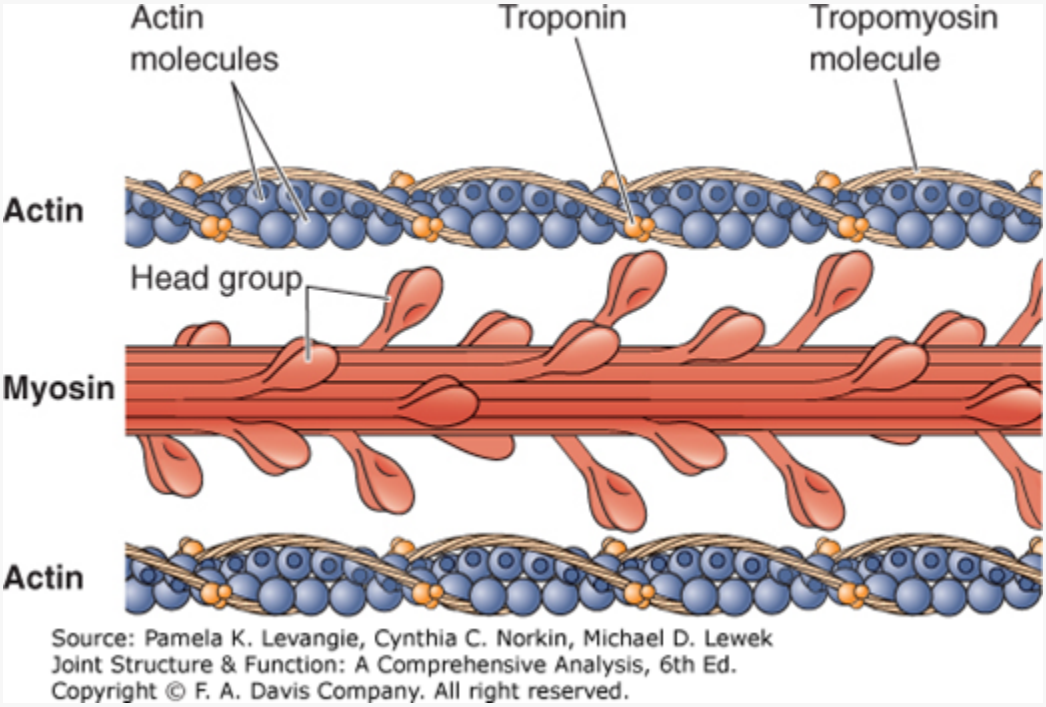
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A muscle fiber contains many myofibrils bundled together within the fiber. Each myofibril within the fiber is composed of thick (myosin) and thin (actin) **myofilaments** formed by protein molecules of **actin, myosin, troponin,** and **tropomysin** (Fig. 3–2). The interaction of actin and myosin myofilaments is essential for a muscle contraction to occur. Actin myofilaments are formed of two chainlike strings of actin molecules wound around each other. Molecules of the globular protein **troponin** are found in notches

between the two actin strings, and the protein **tropomyosin** is attached to each troponin molecule. The troponin and tropomyosin molecules control the binding of actin and myosin myofilaments.

FIGURE 3-2

Myofilaments. The thin (actin) filament is composed of globular actin molecules organized in two strands, with troponin molecules located in notches between the two strands. The tropomyosin molecules are thin and are shown lying along grooves in the actin strands. The thick (myosin) myofilament shows head groups that can attach to specific binding sites on the actin molecule.



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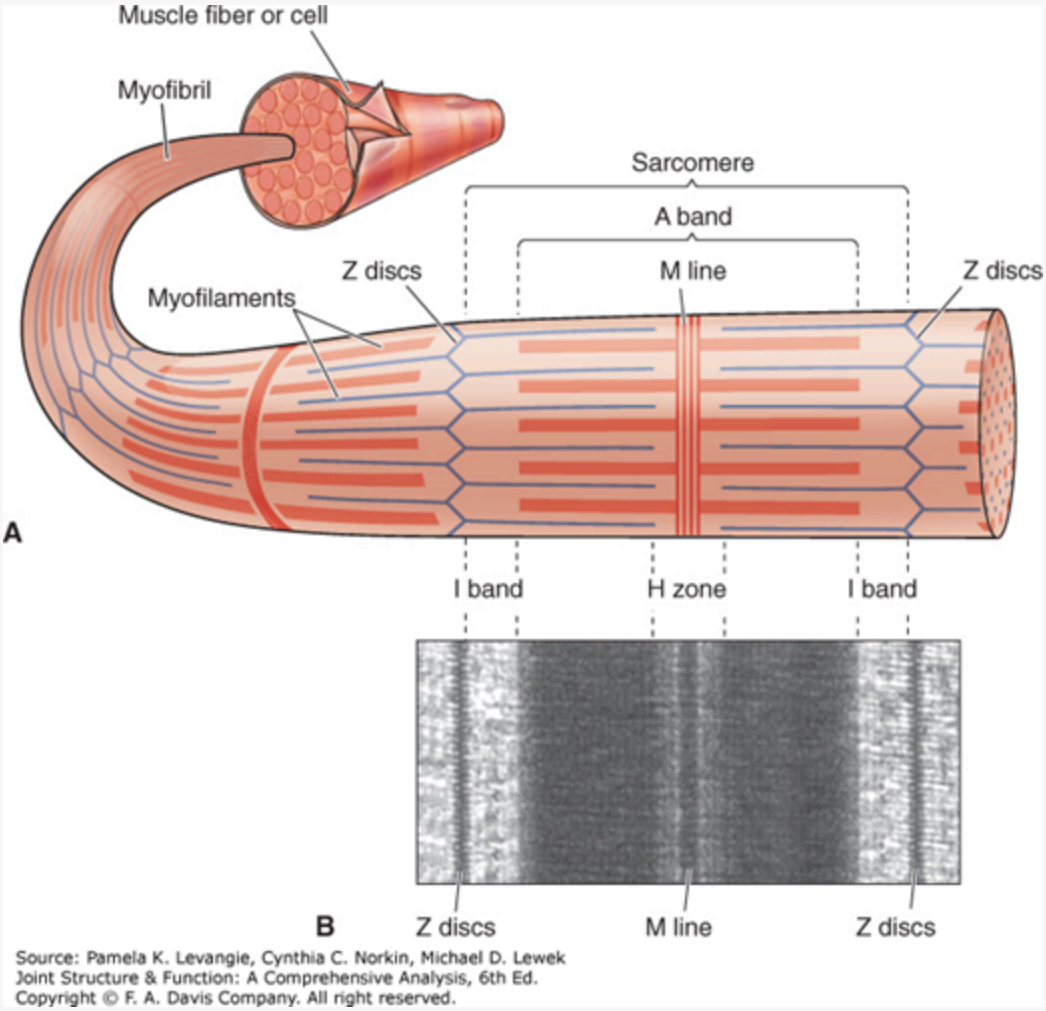
Each of the myosin molecules has globular enlargements called **head groups.**[5] The head groups, which are able to swivel, are the binding sites for attachment to the actin

and play a critical role in muscle contraction and relaxation.

When the entire myofibril is viewed through a microscope, the alternation of thick (myosin) and thin (actin) myofilaments forms a distinctive striped pattern (Fig. 3–3). Therefore, skeletal muscle is also called **striated muscle.**

FIGURE 3-3

Myofibrils in a muscle at rest as shown by **A.** a schematic representation and **B.** electron microscopy. The sarcomere is the portion of the myofibril that lies between the Z discs. The A band portion of the sarcomere contains an overlap of the myosin and actin filaments. The portion of the A band that contains only myosin filaments without overlap is called the H zone. The M line, located in the central portion of the H zone, contains transversely oriented myosin filaments that connect one myosin filament with another. The I band portion contains only actin fibers.



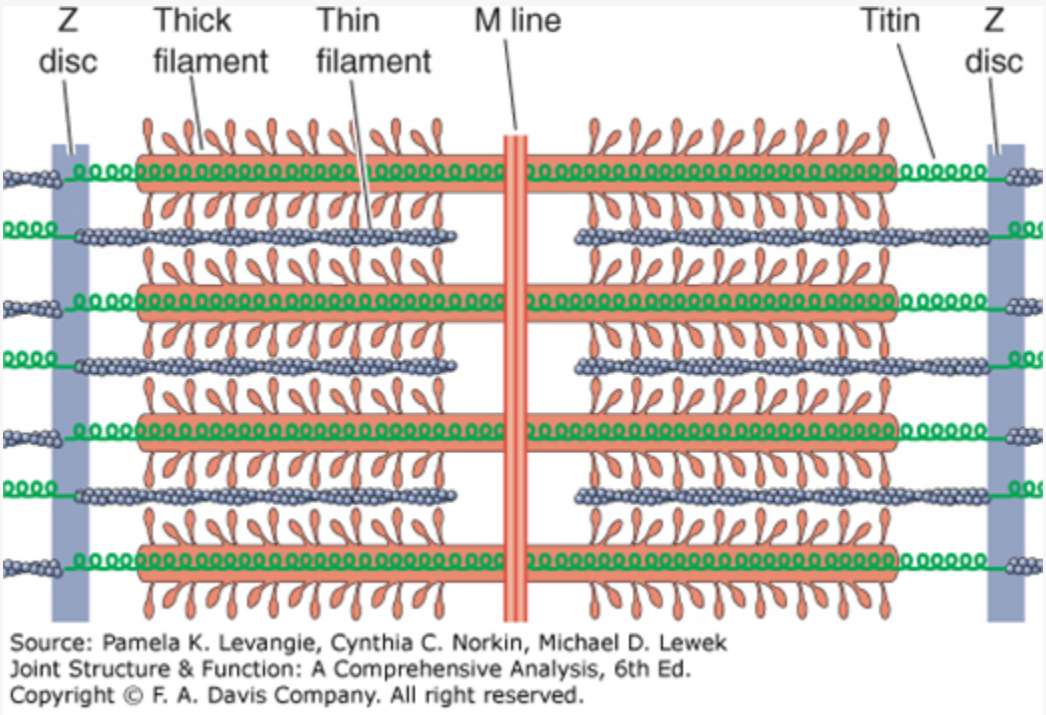
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Structural Proteins

The muscle fiber also consists of several structural proteins.[6,7] Some of these proteins (**intermediate filaments**) provide a structural scaffold for the muscle fiber, whereas others (e.g., **desmin**) may be involved in the transmission of force along the fiber and to adjoining fibers. One protein, **titin,** has a particularly important role in maintaining the position of the thick filament during a muscle contraction and in the development of passive tension.[8–11] Titin is a large protein that is attached along the thick filament and spans the gap from the thick filament to the Z discs (**Fig. 3–4**). Titin has been implicated as a contributor to a muscle's passive (as opposed to active) tension.

FIGURE 3-4

The titin molecule is attached along the thick filament and spans the region between the thick filament and the Z disc.



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The Contractile Unit

Organization of the Contractile Unit

The **sarcomere** is the contractile unit of the muscle and is distinguished by **bands** or **zones** that help to identify the arrangement of thick and thin filaments (see Fig. 3–3A). The portion of a myofibril that is located between any two Z discs along the myofibril is called the **sarcomere.** The Z discs are located at regular intervals along the myofibril, serving not only as boundaries for the serially linked sarcomeres but also to link together the thin filaments of each sarcomere. The portion of the sarcomere that extends over both the length of the thick filaments and a small portion of the thin filaments is called the **anisotropic** or, more commonly, the **A band.** Areas that include

only actin filaments are called **isotropic** or, more commonly, the **I bands.**[5] The terms "anisotropic" and "isotropic" refer to the behavior of these portions of the fibers when light shines on them. The **A band area,** the width of the myosin filaments, has a central area where there is no overlap with the thin filaments, called the **H zone.** The central portion of the H zone, which consists of the wide middle portion of the thick filament, is called the **M line.**

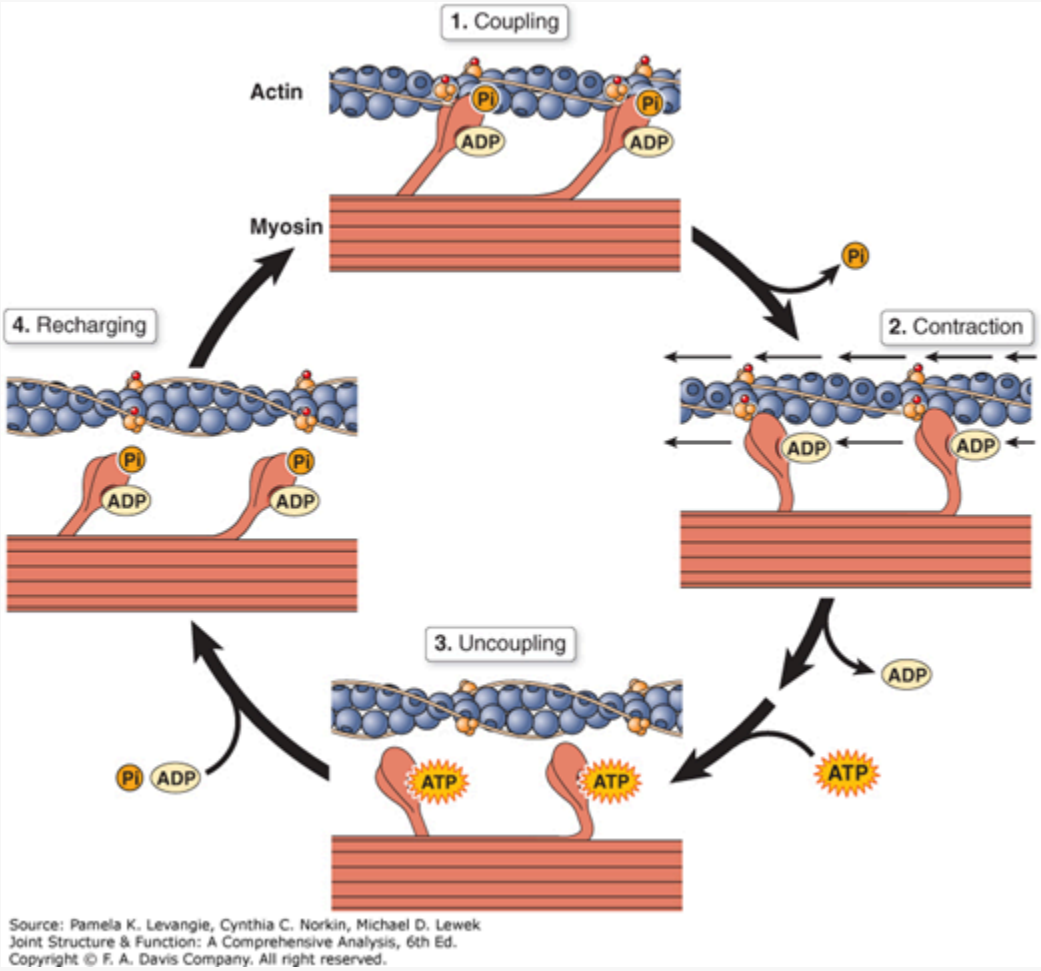
Cross-Bridge Interaction

A muscle contraction is initiated by the interaction of thick and thin filaments within each sarcomere. The interaction is initiated by the arrival of a nerve impulse at the motor endplate, which evokes an electric impulse, or **action potential**, that travels along the muscle fiber.[12] The action potential initiates the release of calcium ions,[13] and the calcium ions cause **troponin** to reposition the tropomyosin molecules so that receptor sites on the **actin** are free and the head groups of the **myosin** can bind with actin. This bonding of filaments is called a **cross-bridge**. Tension in the sarcomere is generated with the hydrolysis of **adenosine triphosphate (ATP)** and the release of **adenosine diphosphate (ADP)** from the myosin head[1,13–15] (Fig. 3–5).

FIGURE 3-5

Cross-bridge cycle. (1) Coupling: In the presence of calcium ions, the troponin-calcium complex moves to expose the active sites of actin, allowing the cross-bridge to form. (2) Contraction: Adenosine diphosphate (ADP) and inorganic phosphate (Pi) are released, which produces the power stroke of the myosin head, thus pulling the thin

filament in relation to the thick filament. (3) Uncoupling: ATP binds to the myosin head and the cross-bridge releases from the actin molecule active site. (4) Recharging. ATP hydrolysis occurs, producing ADP and Pi, which are bound to the myosin head, resulting in the cocking of the head to reattach to the actin binding site. The coupling, contraction, uncoupling, and recharging occurs rapidly to allow for a smooth contraction of the muscle.



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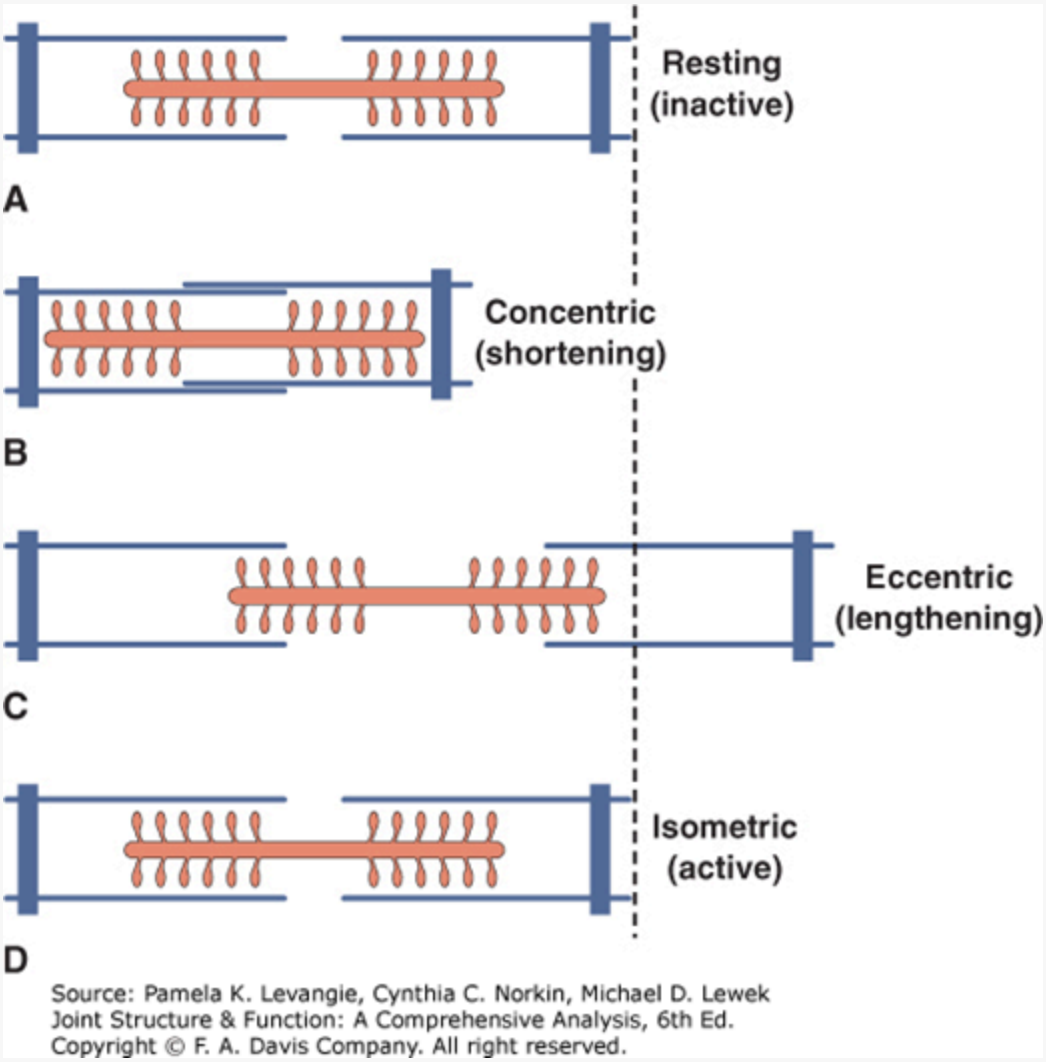
Types of Muscle Contraction

The sliding of the thin filaments toward and past the thick filaments, accompanied by

the formation and re-formation of cross-bridges in each sarcomere, results in the shortening of the muscle fiber and the generation of tension (Fig. 3–6). The muscle fiber shortens if a sufficient number of sarcomeres actively shorten and as long as one or both ends of the muscle fiber are free to move. The active shortening of a muscle is called a **concentric contraction,** or **shortening contraction,** whereby the sarcomere length (the distance between the Z discs) is shorter (Fig. 3–6B) than when the fiber is at rest (Fig. 3–6A). In contrast to a shortening contraction in which the thin filaments increasingly overlap the thick filaments, the muscle may undergo an **eccentric,** or **lengthening, contraction** whereby the sarcomere length is greater than in a resting fiber (Fig. 3–6C). In a lengthening contraction, the thin filaments are pulled away from the thick filaments by a force external to the muscle; cross-bridges are broken and re-form as the muscle lengthens. Tension is generated by the muscle as cross-bridges are re-formed. An active muscle fiber will not change length if the force created by the cross-bridge cycling is matched by the external force ($\Sigma F = 0$). The contraction of a muscle fiber without changing length is called an **isometric contraction** (Fig. 3–6D).

FIGURE 3-6

Types of muscle contraction from the perspective of change (or lack of change) in sarcomere length during the contraction. **A.** Resting length of the sarcomere. **B.** Concentric or shortening muscle contraction. **C.** Eccentric or lengthening muscle contraction. **D.** Isometric contraction with no change in length.



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Expanded Concepts 3–1

Terminology: Muscle Action Versus Muscle Contraction

A potentially more accurate way to describe the types of muscle "contraction" is to use the term *action*. Because the word *contract* means to draw together or shorten, it is an oxymoron to refer to an "eccentric contraction" or "lengthening contraction." However, during the eccentric contraction, the contractile units (cross-bridges) are attempting to contract and pull the thin filaments toward the thick filaments, but the forces external to the active muscle are greater than the forces within the muscle, which results in lengthening. Therefore, the term *contraction* implies the attempt to shorten and the term *eccentric* describes the lengthening. We have chosen to use the

more common term *contraction* in this text, but we realize that muscle *action* is a synonymous term.

Patient Application 3–1

Vik Patel, a 50-year-old male, was playing softball one summer evening. While trying to catch a fly ball, he stepped back with his right foot and slipped slightly. As his foot slipped, the ankle was forced into dorsiflexion, although the ankle plantar flexor muscles were contracting as Vik attempted to push off on the ball of his foot. At the moment he tried to push off, he felt a twinge of pain in his right calf muscle. Because the external moment (torque) created by the dorsiflexion force was greater than the plantarflexion moment, the plantarflexors contracted eccentrically (lengthened). This situation is a common mechanism of injury to the muscle or tendon that will result in pain localized to the muscle or tendon.

Foundational Concepts 3–1

Muscle Contraction Facts

The following is a summary of the important facts about muscle contraction at the sarcomere level:

- Tension is generated whenever cross-bridges are formed.
- Calcium influx initiates the muscle contraction.
- ATP hydrolysis fuels the cross-bridge cycle.
- In a concentric contraction, the thin myofilaments are pulled toward the thick myofilaments, and cross-bridges are formed, broken, and re-formed.
- In an eccentric contraction, the thin myofilaments are pulled away from the thick myofilaments, and cross-bridges are broken, re-formed, and broken again.
- In an isometric contraction, the length of the sarcomere is constant.

The Motor Unit

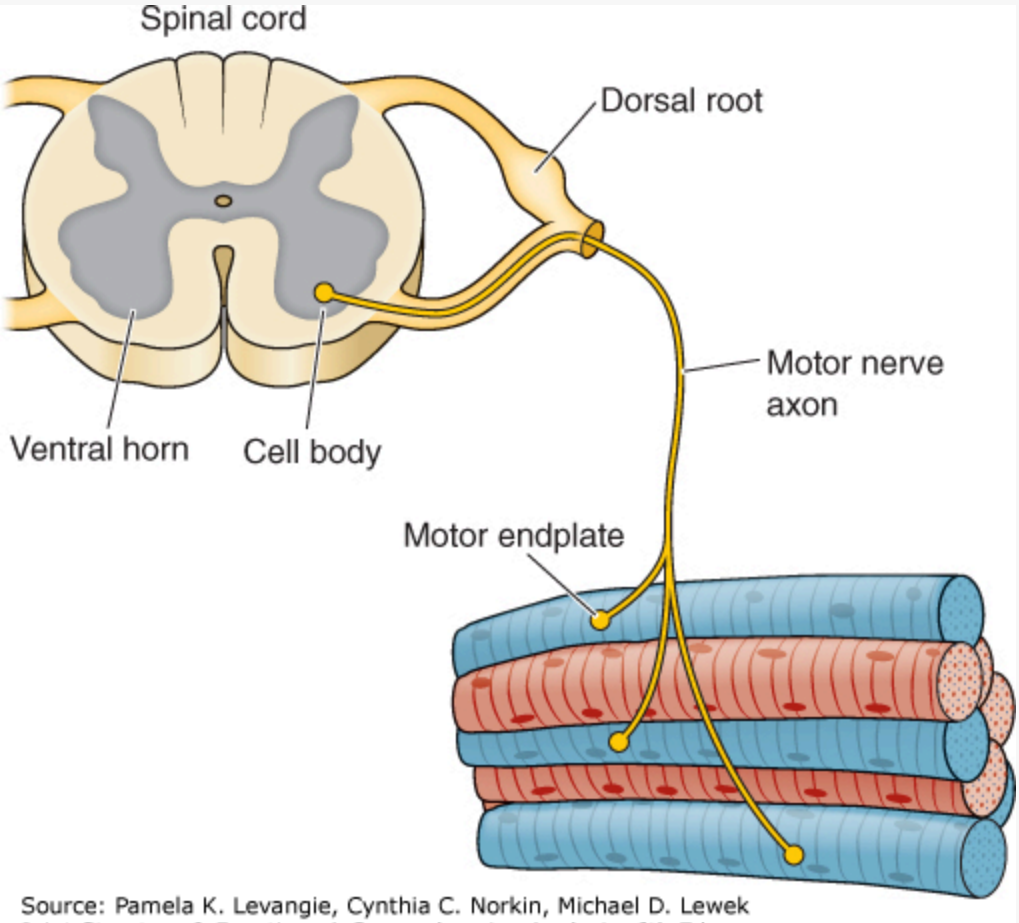
Organization of the Motor Unit

Although the sarcomere is the basic unit of the muscle that produces tension, it is

actually part of a larger complex called the **motor unit.** The motor unit consists of the **alpha motor neuron** and all of the muscle fibers it innervates. The contractile process is initiated when the muscle fibers of a motor unit receive a stimulus transmitted through an alpha motor neuron 3 (Fig. 3–7). The cell body of the neuron is located in the ventral horn of the spinal cord. The nerve cell **axon** extends from the cell body to the muscle, where it divides into a few or as many as thousands of smaller branches. Each of the smaller branches terminates in a motor endplate, which lies in close approximation to the sarcolemma of a single muscle fiber. All of the muscle fibers on which a branch of the axon terminates are part of one motor unit, along with the corresponding cell body and axon.

FIGURE 3-7

An alpha motor neuron. The cell body is in the ventral horn with the motor axon, leaving the ventral root of the spinal cord. Muscle fibers innervated by a single axon are not necessarily continuous from one to another.



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The contraction of the entire muscle is the result of many motor units firing *asynchronously* (not all motor units at the same time) and *repeatedly.*[1] The magnitude of the contraction of the entire muscle may be altered by changing the number of motor units that are activated or the frequency at which they are activated. The number of motor units in a muscle, as well as the structure of these units, varies from muscle to muscle.

Motor units vary according to the size of the neuron cell body, diameter of the axon, number of muscle fibers, and type of muscle fibers.[1] Each of these variations in

structure affects the function of the motor unit. Some motor units have small cell bodies, and others have large cell bodies. Units that have small cell bodies have small-diameter axons. Nerve impulses take longer to travel through small-diameter axons than they do through large-diameter axons. Therefore, in the small-diameter units, a stimulus will take longer to reach the muscle fibers than it will in a unit with a large-diameter axon.

The size of the motor unit is determined by the number of muscle fibers that it innervates and the size of the motor nerve axon. The number of fibers may vary from two or three to a few thousand. A muscle will have small motor units if the muscle either controls fine movements or is used to make small adjustments to the muscle force. Such motor units generally have small cell bodies and small-diameter axons. Muscles that are used to produce large forces and large movements usually have a predominance of large motor units, large cell bodies, and large-diameter axons. The motor units of the small muscles that control eye motions may contain as few as nine muscle fibers, whereas the gastrocnemius muscles have motor units that contain about 2,000 muscle fibers.[1] Muscles with a predominantly large number of fibers per motor unit usually have a relatively small total number of motor units than do muscles that have few fibers per motor unit. The platysma muscle in the neck has relatively small motor units consisting of approximately 25 muscle fibers each, but the muscle has a total of 1,000 of these motor units. The gastrocnemius, on the other hand, has relatively large motor units consisting of about 2,000 muscle fibers per unit, but the muscle has a relatively small number (600) of such units. In most instances, a muscle has at least some mix of small and large motor units.

Recruitment of Motor Units

Usually, when an isometric muscle action is desired, the motor units with small cell bodies and few motor fibers are recruited first by the nervous system and then, as force is increased, larger motor units are recruited.[16,17] This recruitment strategy is referred to as the **size principle of motor unit recruitment.**[16] Small motor units generate less tension than do large motor units and require less energy expenditure, and therefore this recruitment strategy is based ultimately on the force required for the motion. In this sense, it is thought to be energy conserving. If a few small motor units are capable of accomplishing the task, the recruitment of large motor units is unnecessary. If the task demands are such that the small motor units are unable to complete the task, larger motor units can then be recruited. However, the recruitment strategy may be based not only on energy conservation but also on the nature of the task (a multitask muscle such as the biceps brachii is recruited differently for flexion than it is for supination).[18] Additionally, the type of muscle action (concentric, eccentric, or isometric) may affect the order of motor unit recruitment.[17,19] The recruitment strategy may also vary among the individual muscles of a synergistic group, such as the four muscles of the quadriceps femoris, despite all having a common functional task.[20]

The frequency of a motor unit's firing also affects the force modulation. The relationship between frequency of firing and force is quite simple in that the greater the frequency of motor unit firing, the greater the muscle force. The contribution of recruitment or firing frequency to the development of muscle force may be different depending on the muscle. Small, distal muscles tend to rely more on the increased frequency of firing, and larger, proximal muscles may rely more on the recruitment of additional motor units.[21] Finally, a more rapid recruitment of motor units may occur during fatiguing

contractions in which a certain *position* is maintained than when a constant *force* is maintained.[22–24] For example, when performing a high-velocity eccentric contraction of the plantar flexor muscles, a patient might selectively recruit fast motor units rather than relying on the sequential recruitment of slow then fast motor units. This may contribute to high muscle forces and an increased chance of injury. It should be obvious from this brief discussion that motor unit recruitment is a complicated and multifaceted topic.



Foundational Concepts 3–2

Active Muscle Tension Summary

Active tension in a muscle may be affected by:

1. The number of muscle fibers in a motor unit (which affects the magnitude of the response to a stimulus).
2. The diameter of the axon innervating a motor unit (which determines the conduction velocity of the nerve impulse).
3. The number of motor units that are firing at any one time (which affects the total force response of the muscle).
4. The frequency of motor unit firing (which affects the total force response of the muscle).

Muscle Structure

Fiber Types

Three principal types of muscle fibers are found in varying proportions in human skeletal muscles. These fiber types may be distinguished from one another histochemically, metabolically, morphologically, and mechanically. Although there are different systems of nomenclature used in different texts, in this text the three primary

muscle fiber types will be referred to as **type I (slow), type IIA (intermediate),** and **type IIX (fast)** (Table 3–1).[1,4,5] In this classification system (the most common system for human skeletal muscle fiber typing), the myofibrillar ATPase activity under varying acidic and alkaline conditions is used to delineate fiber types. In fact, several intermediate fiber types have been identified through this scheme.

Table 3-1

Characteristics of Skeletal Muscle Fibers



Expanded Concepts 3–2

Another Muscle Fiber Classification Scheme

Another classification scheme uses the response of the muscle to metabolic enzymes as a way to differentiate fiber types. This scheme identifies three main fiber types: fast glycolytic (FG), fast oxidative glycolytic (FOG), and slow oxidative (SO).[4,25,26] This nomenclature is based on the combination of reactions of cellular enzymes with substrates to identify myofibrillar ATPase activity (fast versus slow), succinate dehydrogenase activity (oxidative potential), and α-glycerophosphate dehydrogenase activity (glycolytic potential). It is often assumed that the two schemes (FG/SO vs. type I/II) are interchangeable; however, this may not be the case. There appears to be much overlap of metabolic enzyme activity between type IIA and type IIX. The fact that metabolic enzyme activity levels depend on the degree of training of the muscle suggests that these two schemes may not be the same. Another scheme, using immunohistochemical analysis (identification of portions of the myosin molecule using antibodies), has found that types I, IIA, and IIX fibers correspond specifically to different types of myosin molecules (myosin heavy chain [MHC] I, MHC IIA, and MHC IIX).[25] The combination of this scheme and the myosin ATPase system provides an estimate of the contractile properties of the muscle.

Whichever classification scheme is used, it should be remembered that there is actually a continuum of fiber types without exact distinctions between types.

Each skeletal muscle in the body is composed of a combination of each of the three types of fibers, but variations exist among individuals in the percentage of each fiber type in similar muscles. The variations in fiber types among individuals are believed to be genetically determined. In post-mortem studies, the vastus lateralis, rectus femoris, and deltoid muscles contain about 50% type II and 50% type I fibers;[2] the gastrocnemius contains about 30% type II and 70% type I; and the hamstrings contain about 50% to 55% type II and 45% to 50% type I fibers.[27–29] In studies using muscle biopsy samples from younger subjects, the vastus lateralis tends to be about 54% type II fibers and 46% type I fibers.[30,31] Although the differences may be subtle, fiber types change with age so that there is a decrease in the number and size of type II fibers. This may account for the differences seen in many of the studies documenting fiber type percentages.

In contrast to the other muscles just identified, the soleus muscle contains up to 80% of type I fibers.[27,30,32] Muscles that have a relatively high proportion of type I fibers in relation to type II fibers, such as the soleus muscle, are able to carry on sustained activity because the type I fibers do not fatigue rapidly. These muscles are often called **stability** or **postural** muscles. The relatively small, slow motor units of the soleus muscle (with small cell bodies, small-diameter axons, and a small number of muscle fibers per motor unit) are almost continually active during erect standing to make the small adjustments in muscle tension that are required to maintain body balance and counteract the effects of gravity. Muscles that have a higher proportion of the type II

fibers, such as the hamstring and gastrocnemius muscles, are sometimes designated as **mobility** or **nonpostural** muscles. These muscles are involved in producing large ranges of motion (ROMs) of the bony components.[27–29] Type II fibers produce slightly more force per fiber than type I and are able to contract at a significantly higher velocity, thus producing a higher power output than type I fibers.[33] Although muscle fiber type is important in determining the function of a muscle, there are other aspects of muscle structure that also play an important role in determining muscle function.



Patient Application 3–2

Because fiber type is related to muscle function, in certain situations it is reasonable to make assumptions about which muscle may have been preferentially injured. Vik Patel's ankle plantarflexors were excessively loaded as Vik attempted to run. Because the soleus muscle is composed primarily of type I fibers (postural control) and the gastrocnemius is composed primarily of type II fibers (power and mobility), the gastrocnemius may be selectively recruited during eccentric contractions and, therefore, is more likely to be the affected muscle in Vik's case.

Muscle Architecture: Size, Arrangement, and Length

Many human muscles have an approximately equal proportion of fast and slow fiber types. Therefore, the determination of muscle function cannot be based solely on this single characteristic. In fact, the architecture of the whole muscle may be more important in determining muscle function than the fiber type.[34,35] The description of skeletal muscle architecture includes the arrangement of the fibers in relation to the axis of force (amount of **pennation**), **muscle fiber length, muscle length, muscle mass,** and the **physiological cross-sectional area (PCSA).** These structural variations affect not only the overall shape and size of the muscles but also the function of the skeletal muscles.

The two most important architectural characteristics that affect muscle function are the muscle fiber length and the PCSA. The fiber length at rest is determined by the number of sarcomeres along the fiber (most often referred to as the number of sarcomeres in series). The amount that the fiber can shorten or lengthen is dependent upon the number of sarcomeres in series; the more sarcomeres, the more a fiber can shorten because the contribution of each sarcomere to shortening is additive. Consequently, a long muscle fiber with more sarcomeres in series is capable of shortening more than a short muscle fiber. For example, if muscle fibers are capable of shortening to about 50% of resting length, a muscle fiber that is 6 cm long is able to shorten by 3 cm, whereas a fiber that is 4 cm long is able to shorten by only 2 cm. A muscle with long fibers and more shortening capability will be able to move the bony lever to which it is attached through a greater distance than a muscle with short fibers. However, the relationship between the muscle fiber length and the distance that it is able to move a bony lever is not always a direct relationship because, as we shall see, the arrangement of the muscle fibers also affects the length-shortening relationship.

The PCSA is a measure of the cross-sectional area of the muscle perpendicular to the orientation of the muscle fibers. The amount of force that a muscle produces is directly proportional to the number of sarcomeres in parallel (arranged side-by-side) within the muscle fiber. Therefore, if there are a large number of fibers packed into a muscle (as in a pennate muscle) or if the fiber increases in size (addition of myofibrils), the ability of the muscle to produce force will be increased.[34]

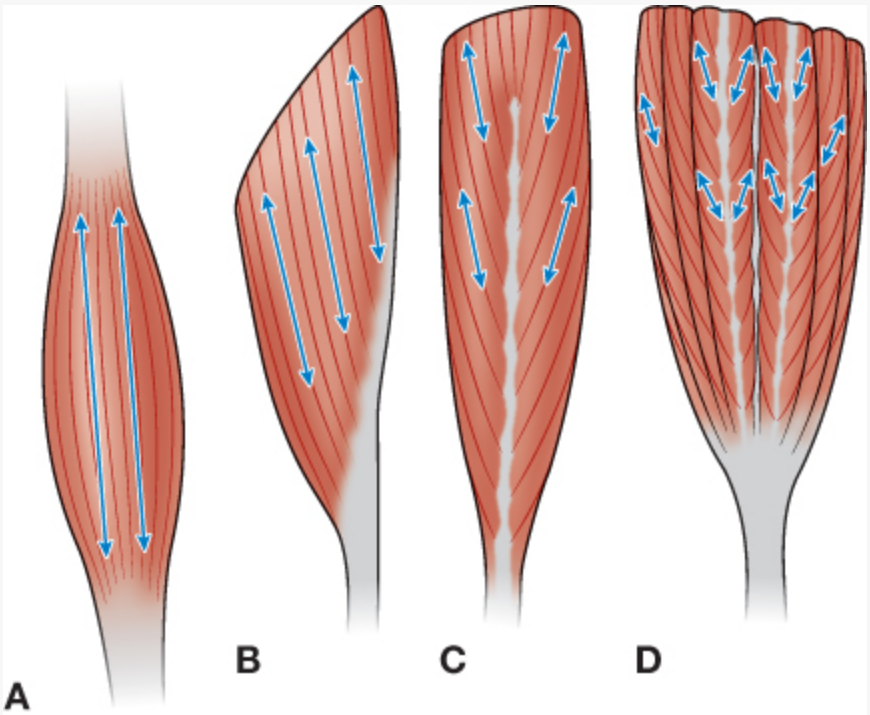
A good example of the relationship between muscle architecture and function is the comparison between the quadriceps and hamstring muscles. The quadriceps muscles

have a larger PCSA, and the hamstring muscles have longer fibers.[36–39] This architectural arrangement suggests that the quadriceps muscles are designed for force production and the hamstring muscles are designed for movements requiring a larger ROM. Because most of the hamstring muscles cross two joints (hip and knee), it would be expected that the muscle would need longer fibers for the greater excursion required during movements of both the hip and the knee.

Arrangement of fascicles (muscle fiber groups) varies among muscles (Fig. 3–8). The fasciculi may be parallel to the long axis of the muscle, as in fusiform-shaped muscles (Fig. 3–8A), or at an angle to the long axis in either a unipennate (Fig. 3–8B), bipennate (Fig. 3–8C), or multipennate (Fig. 3–8D) arrangement. Muscles that have a parallel fiber arrangement (parallel to the long axis of the muscle and to each other) are designated as **fusiform** or **strap** muscles. In fusiform muscles such as the sternocleidomastoid or sartorius muscle, the fascicles are long and extend throughout the entire length of the muscle. However, in the rectus abdominis, which is also considered a fusiform muscle, the fascicles are divided into short segments by fibrous intersections. In general, fusiform muscles with a parallel fiber arrangement have longer fibers and therefore produce a greater angle of motion of a bony lever than will muscles with a pennate fiber arrangement.

FIGURE 3-8

Arrangement of fasciculi in a muscle. **A.** Fusiform. **B.** Unipennate. **C.** Bipennate. **D.** Multipennate.



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Muscles that have a fiber arrangement oblique to the muscle's long axis are called **pennate muscles** because the fiber arrangement resembles that found in a feather. The fibers that make up the fascicles in pennate muscles are usually shorter and more numerous than the fibers in many of the fusiform muscles. In **unipennate** muscles, such as the flexor pollicis longus, the oblique fascicles fan out on only one side of a central muscle tendon. In a **bipennate** muscle, such as the biceps femoris and the tibialis anterior, the fascicles are obliquely set on both sides of a central tendon. In a **multipennate** muscle, such as the soleus or subscapularis, the oblique fascicles converge on several tendons.

The oblique angle of the muscle fibers in a pennate muscle disrupts the direct relationship between the length of the muscle fiber and the distance that the muscle

can move a bony lever, and decreases the amount of force that is directed along the long axis of the muscle. Only a portion of the force of the pennate muscles goes toward producing motion of the bony lever. In fact, the more obliquely a fiber lies to the long axis of the muscle, the less force the muscle can exert at the tendon. This decrease in muscle force is a function of the cosine of the pennation angle. Many human muscles have a pennation angle that is less than 30° at rest. Therefore, the muscle force at the tendon will be decreased by a maximum of 13% (cos 30° = 0.87).[4] When a muscle shortens during a contraction, the pennation angle becomes much more oblique, thus potentially affecting the tendon force even more.[13] This in theory decreases the force at the tendon. The loss of force on the tendon from the pennation angle, however, is offset because pennate muscles usually have a large number of muscle fibers as a result of increased fiber packing, thus increasing PCSA. Therefore, despite the loss of force as a result of pennation, a pennated muscle, such as the soleus, is still able to transmit a large amount of force to the tendon to which it attaches.

Muscular Connective Tissue

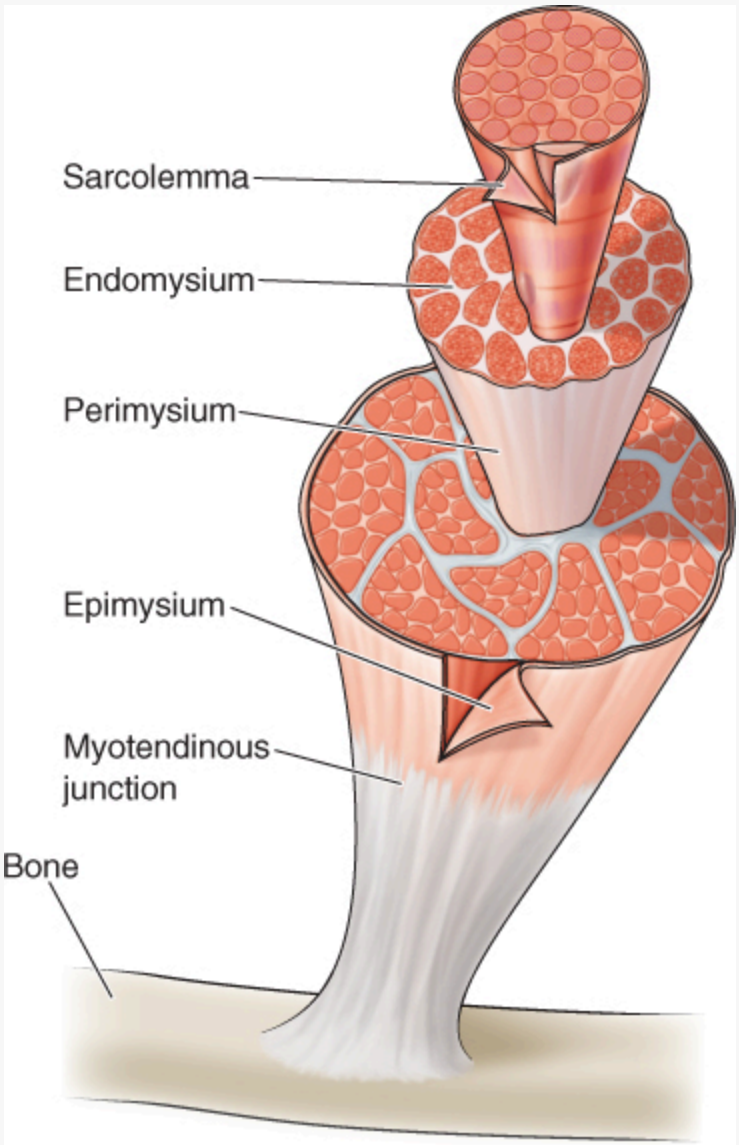
Organization of Connective Tissue in Muscle

Muscles and muscle fibers, like other soft tissues in the body, are surrounded and supported by connective tissue. The sarcolemma of individual muscle fibers is surrounded by connective tissue called the **endomysium.** Within the muscle fiber, the myofibril is connected to the endomysium via specialized proteins such as intermediate filaments and desmin, which were mentioned previously. Muscle fascicles (groups of muscle fibers) are covered by connective tissue called the **perimysium.** The endomysium and perimysium are continuous with the outer connective tissue sheath called the **epimysium,** which envelops the entire muscle (Fig. 3–9). The **myotendinous**

junction is an intricate connection between muscle fibers and the connective tissue of the tendon. Many special proteins are arranged to make this junction strong. Tendons are attached to bones by Sharpey fibers, which become continuous with the periosteum. Although the myotendinous junction is designed to be strong and is able to transmit the larger forces from the muscle to the tendon, the myotendinous junction is often the site of muscle strain injuries.

FIGURE 3-9

Muscular connective tissue. A schematic cross-sectional view of the connective tissue in a muscle shows the endomysium around individual fibers and their sarcolemmas, the perimysium surrounding groups of fibers (fascicle), and the epimysium as the outer layer of connective tissue.



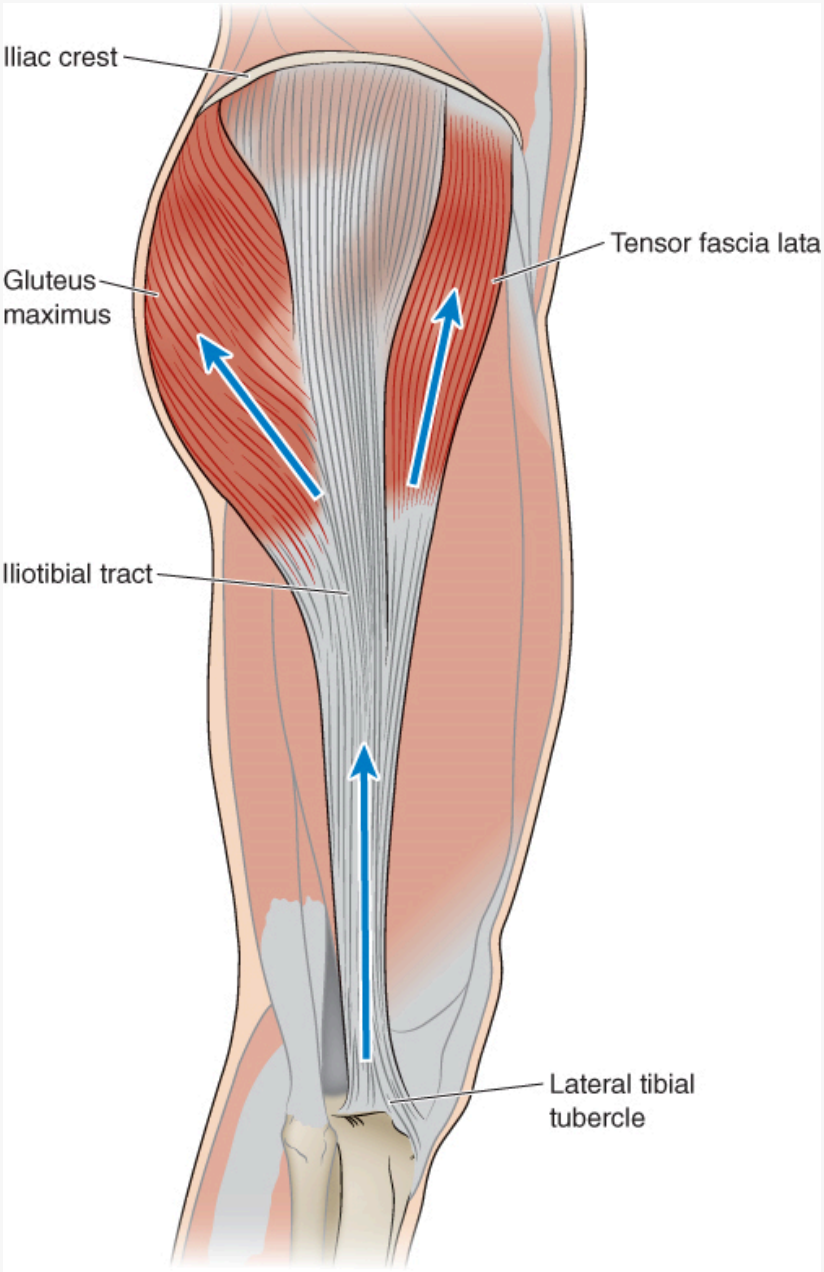
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Other connective tissues associated with muscles include fasciae, aponeuroses, and sheaths. Fasciae can be divided into two zones: superficial and deep. The zone of **superficial fasciae**, composed of loose tissue, is located directly under the dermis (skin). This zone contributes to the mobility of the skin, acts as an insulator, and may contain

muscles that connect to skin such as the platysma in the neck. The zone of **deep fasciae** is composed of compacted and regularly arranged collagenous fibers. The deep fasciae attach to muscles and bones and may form tracts, bands, or retinaculae. For example, the deep femoral fascia in the lower extremity forms a tract known as the **iliotibial tract** or **band.** This tract transmits the pull of two of the lower extremity muscles (the tensor fascia lata and the gluteus maximus) to the tibia (Fig. 3–10). **Retinaculae** are formed by localized transverse thickenings of the fasciae. Each retinaculum forms a loop that is attached at both ends to bone through which tendons pass (Fig. 3–11). The retinaculae maintain the relationship of the tendons to their respective joints and joint axes, also preventing tendons from bowing out of position during muscle action. Sometimes deep fasciae are indistinguishable from aponeuroses, which are sheets of dense, white, compacted collagen fibers that attach directly or indirectly to muscles, fasciae, bones, cartilage, and other muscles. Aponeuroses distribute forces generated by the muscle to the structures to which they are attached.[40]

FIGURE 3-10

Iliotibial tract. A lateral view of the left lower limb showing the deep fascial iliotibial tract extending from the tubercle of the iliac crest to the lateral aspect of the tibia.

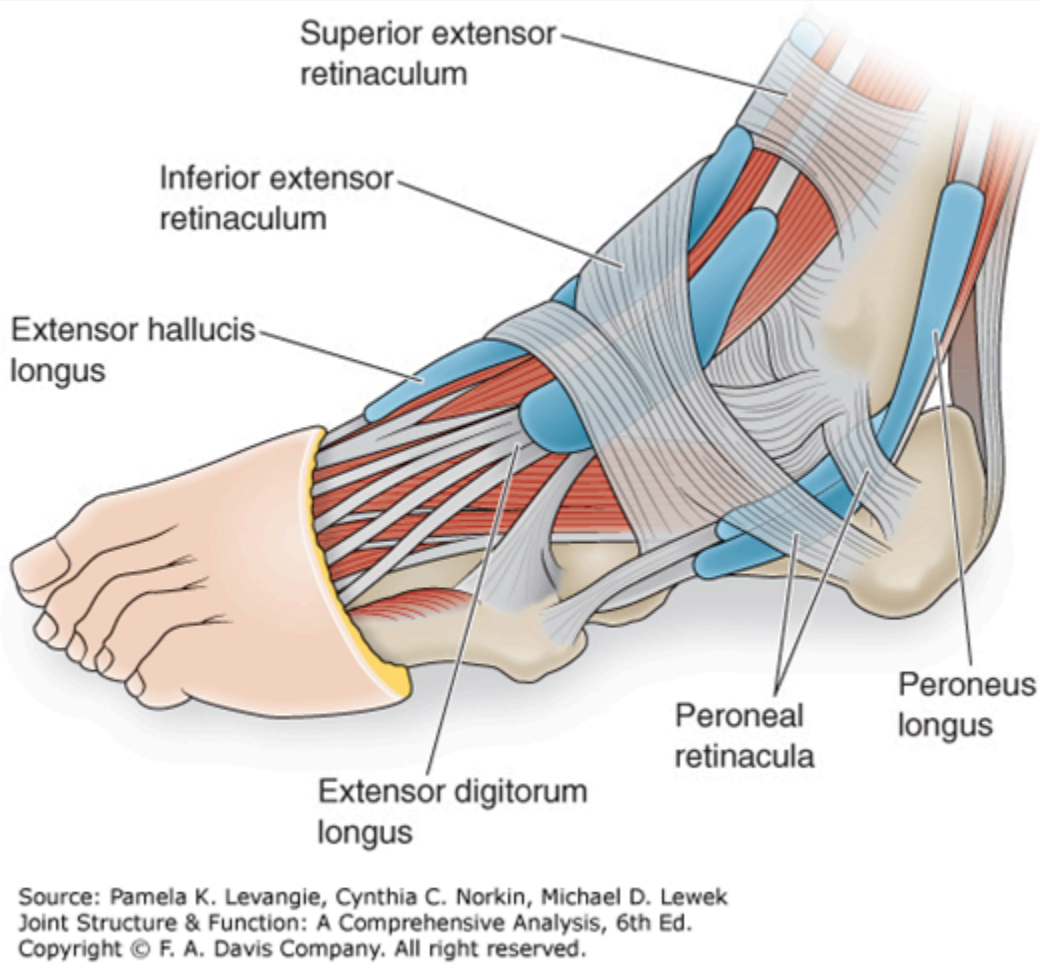


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FIGURE 3-11

The superior and inferior retinaculae and the peroneal retinaculae form tunnels for the muscles entering the foot, which maintain the

position of each of the tendons relative to the joints and joint axes they cross.



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Parallel and Series Elastic Components of Muscle

All of the connective tissues in a muscle are interconnected and constitute the parallel and series **passive elastic components** of a muscle. The connective tissues that surround the muscle, plus the sarcolemma, the elastic protein titin, and other structures (i.e., nerves and blood vessels), form the **parallel elastic component** of a muscle. When a muscle lengthens or shortens, these tissues also lengthen or shorten because they function in parallel with the muscle contractile unit. For example, the

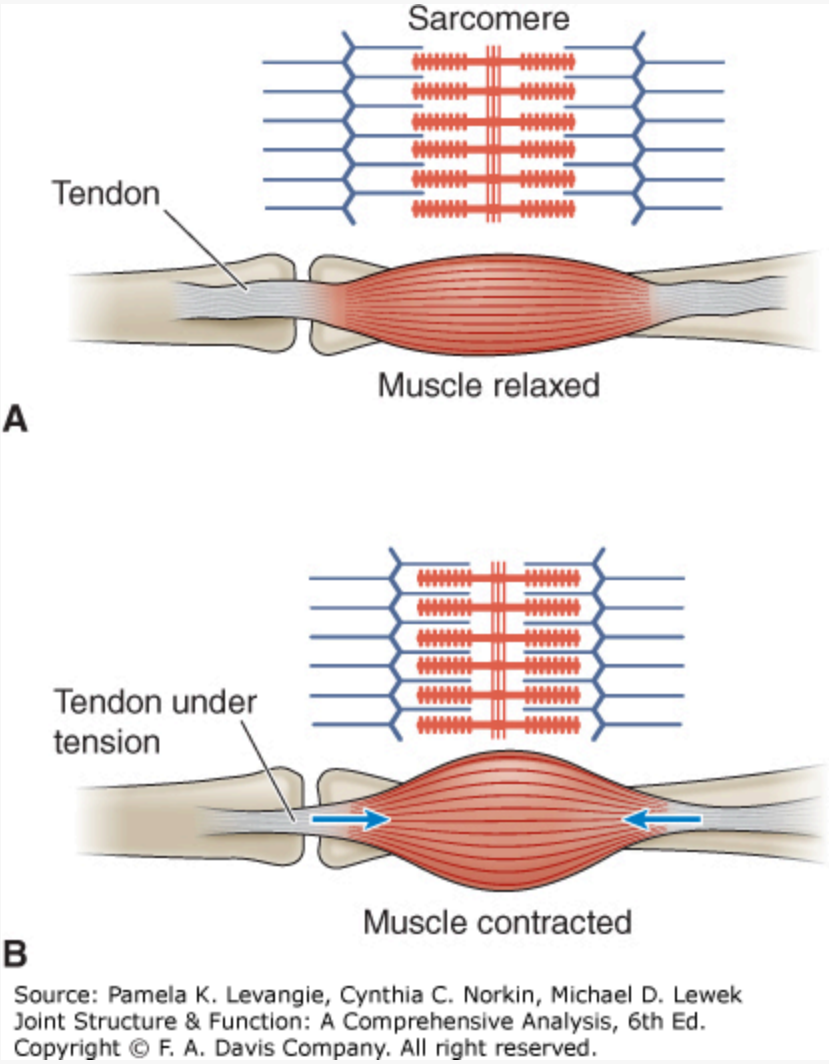
collagen fibers in the perimysium of fusiform muscles are slack when the sarcomeres are at rest, but straighten out and become taut as sarcomere lengths increase during an eccentric contraction. As the perimysium is lengthened, it also becomes stiffer (resistance to further elongation increases). The increased resistance of perimysium to elongation may prevent overstretching of the muscle fiber bundles and may contribute to the tension the muscle produces at the tendon.[41–44] When sarcomeres shorten from their resting positions, collagen fibers within the parallel elastic component that are slack at rest can **buckle (crimp)** even further. Whatever tension might have existed in the parallel elastic components at rest is diminished by the shortening of the sarcomere. Because of the many parallel elastic components of a muscle, the increase or decrease in passive tension can substantially affect the total (active plus passive) tension output of a muscle. This relationship between length and tension will be addressed in the next section.

In contrast to the parallel elastic component, the tendon is the primary structure comprising the **series elastic component** of the muscle (Fig. 3–12). The tendon of the muscle is considered to function in series with the contractile elements. When a muscle is at resting length, the tendon(s) are relaxed and may be crimped (slack) (Fig. 3–12A). The tendon will be under tension when the muscle actively produces tension. When the contractile elements in a muscle actively shorten, they exert a pull on the tendon (Fig. 3–12B). The active pull of the muscle fibers must be of sufficient magnitude to take up the slack (compliance) in the tendon so that the muscle pull can be transmitted through the tendon to the bony lever. Fortunately, the **compliance** (or **extensibility**) of the tendon is relatively small (about 3%–5% in human muscles). Thus, most of the muscle force can be used for moving the bony lever and is not dissipated in stretching the

tendon. The tendon is also under tension when a muscle is controlling or braking the motion of the lever in an eccentric contraction. A tendon is under reduced tension only when a muscle is completely relaxed and in a relatively shortened position.

FIGURE 3-12

Series elastic component. **A.** The muscle is shown in a relaxed state with the tendons slack (crimping or buckling of collagen fibers has occurred). The sarcomere depicted above the muscle shows minimal overlap of thick and thin filaments because the muscle is at resting length. **B.** The muscle in an actively shortened position shows that the tendons are under tension. The sarcomere depicted above the muscle shows extensive overlap of filaments and cross-bridge formation.



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Muscle Function

Muscle Tension

The most important characteristic of a muscle is its ability to develop tension and to exert a force on the bony lever. Tension in a muscle can be either active or passive, and the **total tension** that a muscle can develop includes both active and passive components. Total tension, which was identified in Chapter 1 as muscle force (Fms), is a vector quantity that has:

1. Magnitude
2. Two points of application (at the proximal and distal muscle attachments)
3. An action line
4. Direction of pull

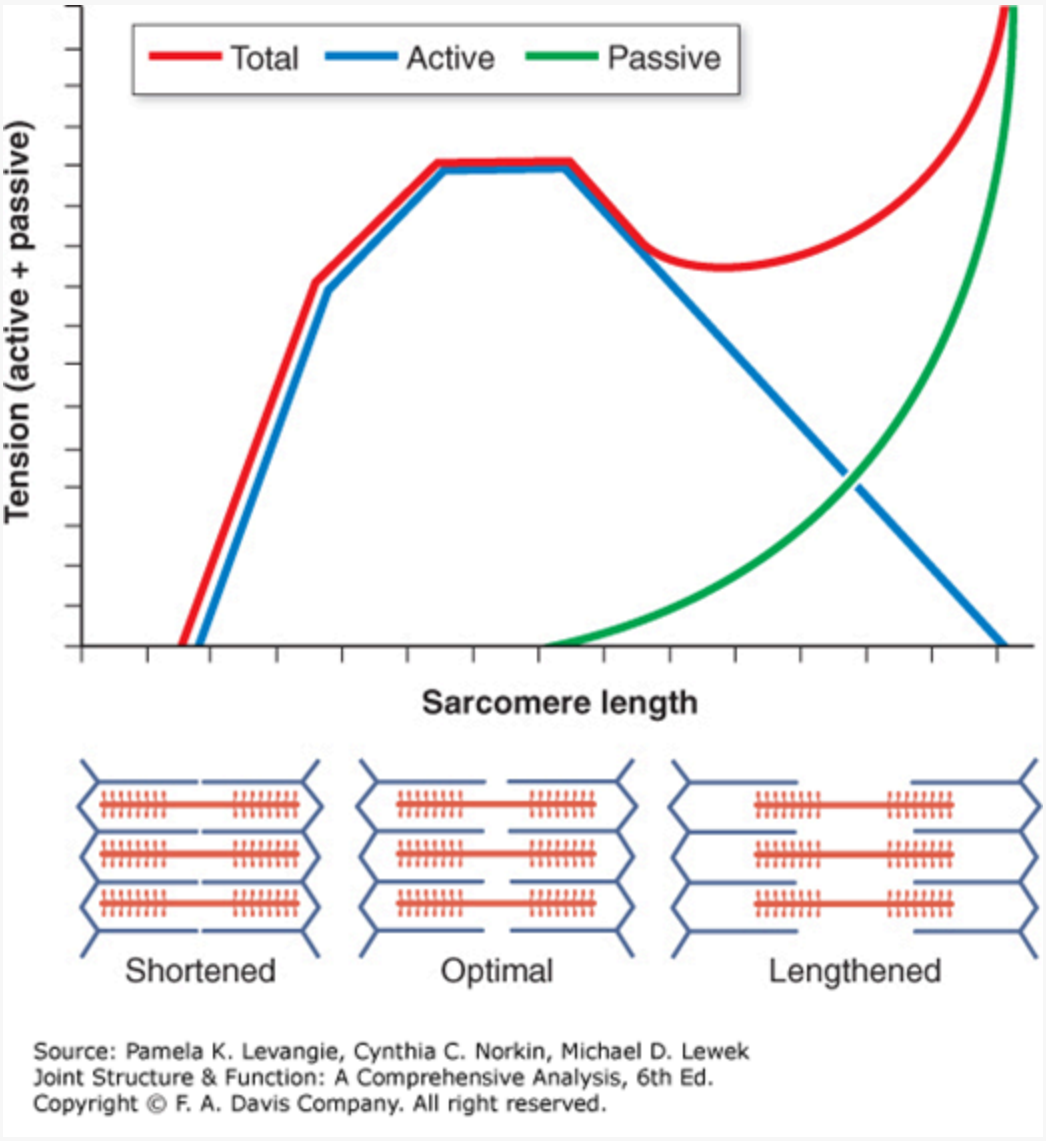
The point of application, action line, and direction of pull were the major part of the discussion of muscle force in Chapter 1, but we now need to turn our attention to the determinants of the total muscle tension (the magnitude of Fms), which will include a combination of the passive (non-contractile) and active (contractile) elements of a muscle.

Passive Tension

Passive tension refers to tension developed in the parallel elastic components of the muscle. Passive tension in the parallel elastic components is created by lengthening the muscle beyond the slack length of the tissues, in other words, by stretching the muscle. The parallel elastic components of the muscle will augment the active tension produced by the muscle fibers when the muscle is lengthened and the parallel elastic components are stretched (Fig. 3–13). As a muscle shortens, the parallel components will become increasingly less stretched (tensed). Once the parallel elastic elements are slack in a shortened muscle, they will contribute little or nothing to the total muscle tension.

FIGURE 3-13

The skeletal muscle isometric sarcomere length-tension relationship. The plateau of the active tension curve occurs at optimal sarcomere length. Active tension decreases as the muscle is lengthened because fewer cross-bridges can be formed. Active tension also decreases as the muscle is shortened because of interdigitation of the thin filaments. Passive tension increases (once crimp is picked up) with elongation of the muscle as the parallel elastic elements become increasingly stretched. Passive plus active tension results in the total tension developed by the muscle fiber.



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Expanded Concepts 3–3

Passive Muscle Stiffness

Passive muscle stiffness is an important property of skeletal muscle. The passive stiffness of an isolated muscle (not connected to bones and joints) is the slope of the passive length-tension relationship. The steeper the slope, the greater the stiffness in the muscle. The passive stiffness of a muscle attached to bone and crossing a joint is the slope of the torque-angle relationship. Titin is the primary component of the muscle that accounts for the stiffness (the resistance to stretching throughout the ROM) of the muscle.[9,45,46] On the other hand, the connective tissues in and around

the muscle (perimysium and endomysium) are responsible for the extent to which the muscle can be elongated.[41] This is often referred to as the muscle **extensibility** or **flexibility.**

Active Tension

Active tension refers to tension developed by the contractile elements of the muscle. Active tension in a muscle is initiated by cross-bridge formation and movement of the thin filaments on the thick filaments. The amount of active tension that a muscle can generate depends on neural factors and mechanical properties of the muscle fibers. The neural factors that can modulate the amount of active tension include the frequency, number, and size of motor units that are recruited. The mechanical properties of muscle that influence the amount of active tension are the isometric length-tension relationship and the force-velocity relationship.

Isometric Length-Tension Relationship

One of the fundamental concepts in muscle physiology is the direct relationship between isometric tension development in a muscle fiber and the length of the sarcomeres in a muscle fiber.[47] Identification of this relationship was, and continues to be, the primary evidence supporting the sliding filament theory of muscle contraction. The isometric sarcomere length-tension relationship was experimentally determined with isolated single muscle fibers under very controlled circumstances. There is an optimal sarcomere length at which a muscle fiber is capable of developing maximal total isometric tension (Fig. 3–13). Muscle fibers develop maximal isometric tension at optimal sarcomere length because the thick and thin filaments are positioned so that the maximum number of cross-bridges within the sarcomere can be formed. If the muscle fiber is lengthened or shortened beyond optimal length (Fig. 3–13), the amount

of active tension that the muscle fiber is able to generate decreases.

When a muscle fiber is lengthened beyond optimal length, there is less overlap between the thick and thin filaments, fewer possibilities for cross-bridge formation, and reduced active tension. However, the passive tension in the parallel elastic components may increase as these elements are stretched in the elongating or elongated muscle. This passive elastic tension is added to the active tension produced by the sarcomeres, resulting in greater total tension (Fms) in spite of the decrease in active tension (Fig. 3–13).

When a muscle fiber is shortened from its optimal sarcomere length, there is a similar decrease in potential isometric active tension in the muscle fiber. When the sarcomere is at shorter lengths, the distance between the Z discs decreases and there is interdigitation of the filaments. The interdigitation of the thick and thin filaments may interfere with the formation of cross-bridges from the myosin molecules, thus decreasing the active force. In the instance of a shortened muscle fiber, however, the passive parallel elastic components are relatively or completely slack, thus contributing little or nothing to the already reduced active tension of the shortening muscle fiber. The net effect is that the total muscle tension (Fms) is least in a shortening muscle fiber (Fig. 3–13).

It must be remembered that the sarcomere length-tension relationship was determined with isometric contractions and therefore should apply, in the strict sense, only to isometric muscle contraction. In addition, as we will see in the following section, the full range of sarcomere lengthening and shortening may be possible only in experiments with isolated muscle. Sarcomere length obviously changes during dynamic

contractions (concentric and eccentric contractions) that affect the tension that can be developed in the muscle. However, during dynamic contractions, *the length-tension relationship must be combined with the force-velocity relationship to determine the effect that both length and velocity have on muscle tension.*

APPLICATION OF THE LENGTH-TENSION RELATIONSHIP

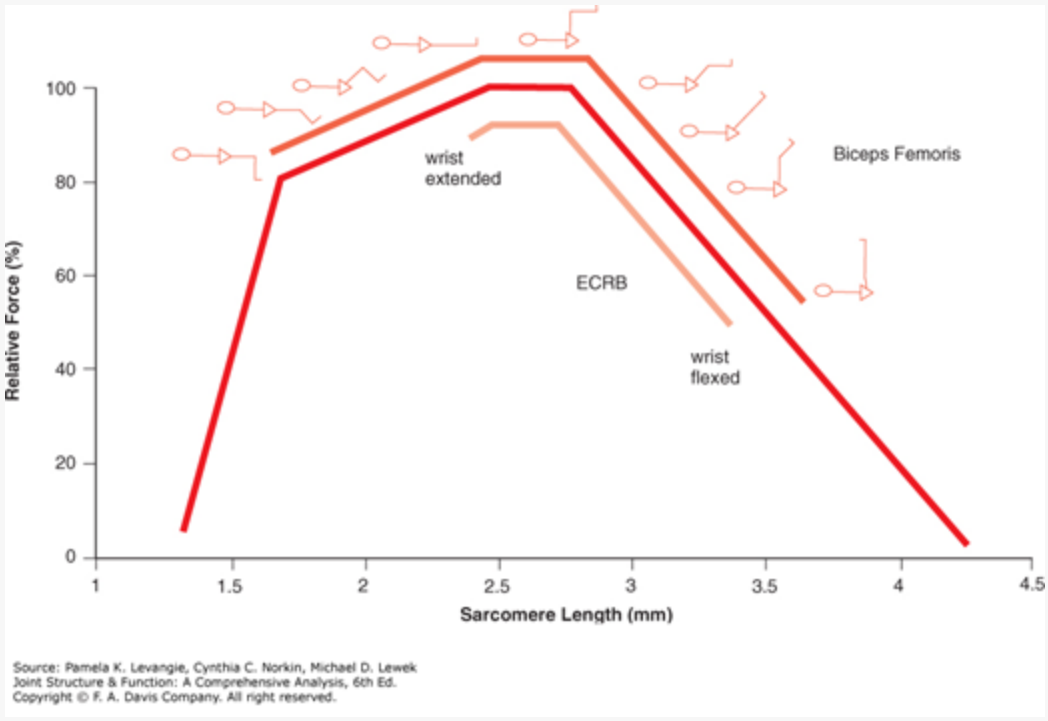
Applying the length-tension relationship to a whole muscle and ultimately to muscle-joint systems is not a simple matter. For example, sarcomere length is not homogeneous throughout the muscle, let alone between muscles with similar functions. This means that for any particular muscle length at a particular joint position, there may be sarcomeres at many different lengths corresponding to different points on the length-tension relationship. Also, when the muscle is acting at a joint, the torque produced is not only a function of the muscle force (which depends on muscle length) but also a function of the moment arm of the muscle. This means that at a certain joint angle, the muscle length may be short (which suggests that force will be low), but the moment arm may be relatively long, thus providing a higher joint torque. From these examples it is clear that the sarcomere length-tension relationship is important in our understanding of muscle physiology, but there are other important factors when whole muscle and joint systems are considered.

Only a few experiments have attempted to determine the isometric sarcomere length-tension relationship in intact human muscle.[48–52] In experiments on human wrist and thigh muscles, the range of sarcomere length changes that are seen with normal joint motion is quite small and is located around optimal length. This design appears to be quite beneficial in that the muscle is not disadvantaged by being too long or too short.

Figure 3–14 shows the estimated length-tension relationship for the extensor carpi radialis brevis and the biceps femoris long head.[37,48]

FIGURE 3-14

The sarcomere length-tension relationship for the human extensor carpi radialis brevis (ECRB) and the long head of biceps femoris, as each contracts from its most shortened position (left) to its most lengthened position (right). The dark red line is the presumed length-tension relationship of human muscle, based on the known lengths of human thin and thick filaments. Neither muscle was found to use the full range of that presumed length-tension relationship. The data for the ECRB were determined from intraoperative laser diffraction measurement of the sarcomere length, and the data from the biceps femoris were calculated on the basis of ultrasound measurements of the change in fascicle length with joint position.[35] *(Adapted from Chleboun GS, France AR, Crill MT, et al: In vivo measurement of fascicle length and pennation angle of the human biceps femoris muscle. Cells Tissues Organs 169:401, 2001; with permission.)*



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One empirical application of the sarcomere length-tension relationship is the observation that a muscle has a diminished ability to produce isometric tension at the extremes of joint motion. This probably occurs only in muscles that cross more than one joint (two-joint or multi-joint muscles) because muscle length excursion is relatively greater than in single-joint muscles. A decrease in the torque produced by the muscle may be encountered when the full ROM is attempted simultaneously at all joints crossed by a multi-joint muscle. This decrease in torque production is often referred to as **active insufficiency,** especially when the active muscle is unable to shorten sufficiently to complete the full available range at all joints crossed by the muscle. Although the decrease in torque can be explained by the length changes that result in decreased muscle force, other factors, such as the change in the moment arm and the passive restraint of the lengthened antagonists, also play a substantial role. Because

sarcomere length appears to stay close to the optimal length during joint movements, the influences of the moment arm and passive restraint of the antagonists may be more important than once thought.[48–54] Therefore, the term *active insufficiency* refers to a concept that is much broader than just the reduction in active length-tension properties of the muscle.

Patient Application 3–3

Angie Frenden is a 39-year-old woman who sustained a crush injury to her radial nerve resulting from a displaced fracture of the mid-shaft of her humerus. Although recovery may occur over time, Angie is currently having problems effectively gripping objects with the affected hand.

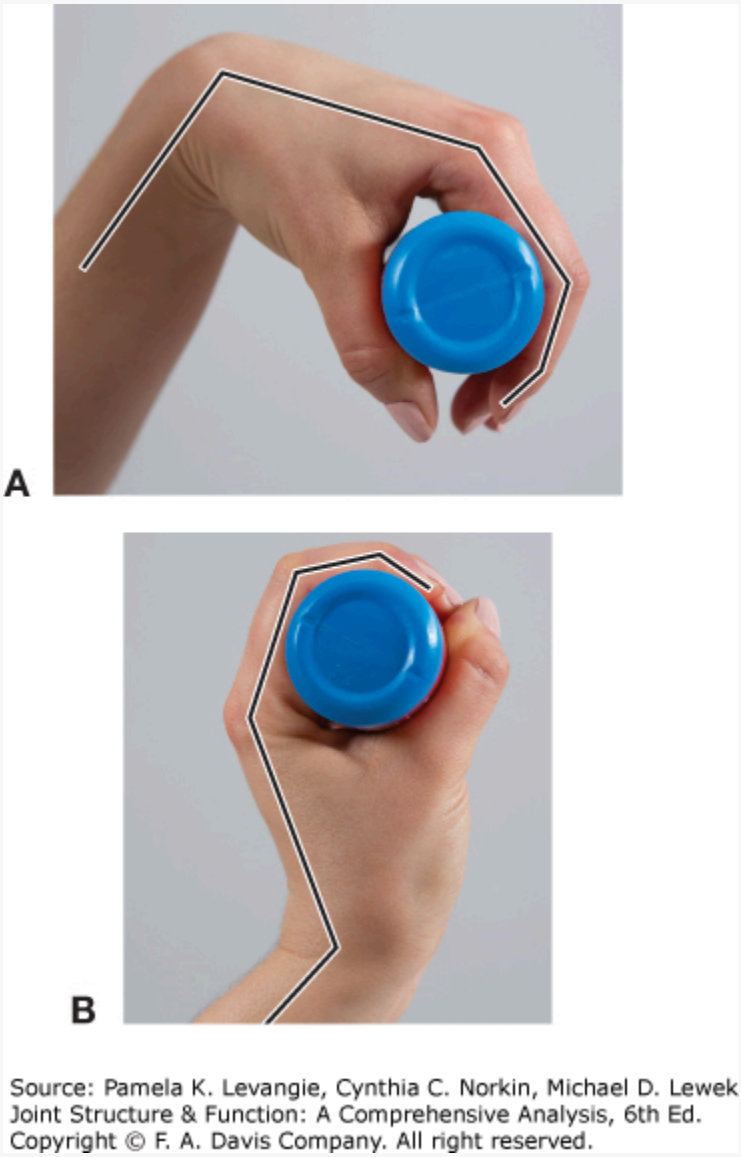
The finger flexors cross the wrist, carpometacarpal, metacarpophalangeal, and interphalangeal joints. If the finger flexors simultaneously flex all the joints at the same time, the maximally shortened finger flexors will generate less torque and are unlikely to be able to complete the flexion ROM at all joints; this will make grip less effective (Fig. 3–15A). The problem is aggravated by the stretch of the passive finger extensors over all the flexed joints that may be restricting motion and creating an opposing passive force.

When Angie's radial nerve recovers, the radial nerve innervated wrist extensors can prevent the finger flexors from flexing the wrist, thus maintaining a slight stretch of the finger flexors at the wrist. With a more favorable length-tension, the finger flexors can shorten sufficiently to complete the finger flexion ROM, rendering grip more effective (Fig. 3–15B). The potentially opposing passive tension of the finger extensors is also released somewhat by the wrist extension position.

FIGURE 3-15

Decrease in active tension with muscle shortening. A. The individual is attempting to grasp the object but is unable to do so effectively because the finger flexors are attempting to shorten over the flexed

wrist as well as over the flexed fingers. In addition, the finger extensors are stretched over the flexed wrist and fingers, potentially restricting flexion range. **B.** By stabilizing the wrist in slight extension, the finger flexors are *stretched* at the wrist, enabling them to shorten more at the fingers and increasing the effectiveness of grasp. The slight wrist extension also reduces possible resisting tension in the finger extensors.



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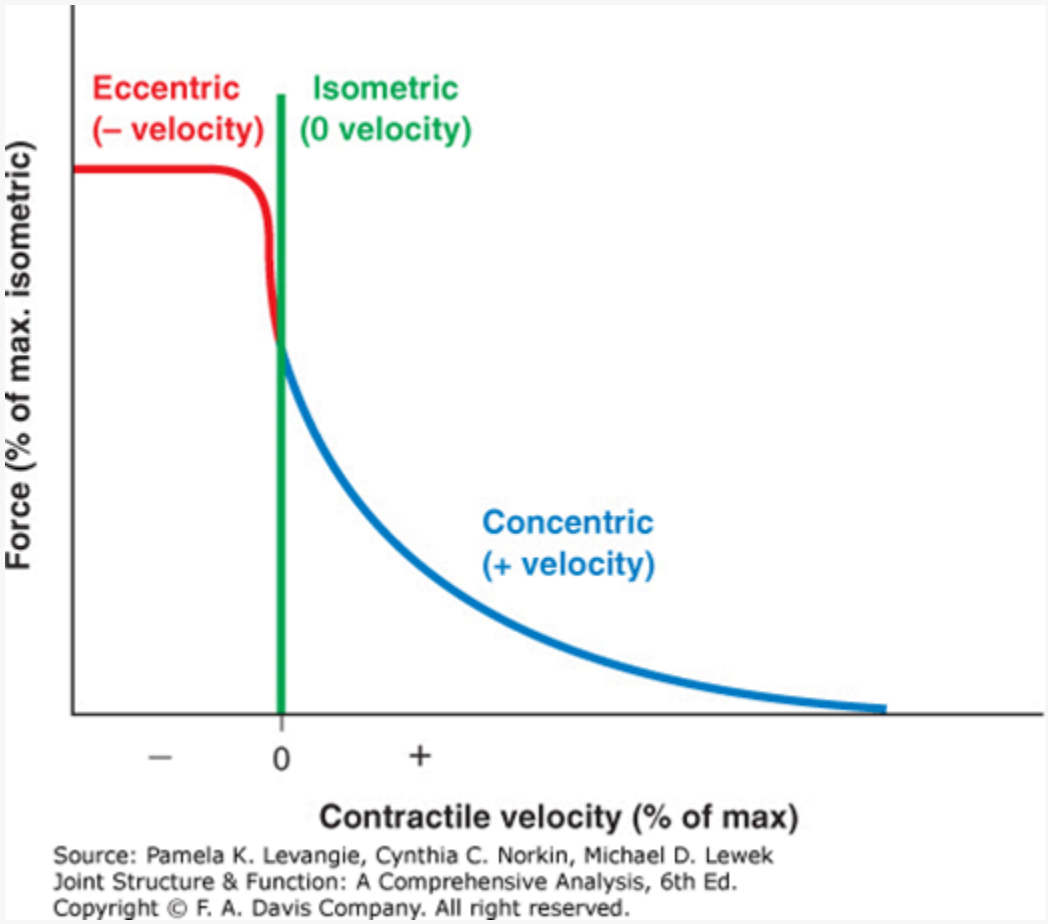
Force-Velocity Relationship

Another factor that affects the development of tension within a muscle is the speed of shortening of the myofilaments. The speed of shortening is the rate at which the myofilaments slide past one another while forming and re-forming cross-bridges. The ability of a muscle to generate force is affected by the speed at which a concentric or eccentric contraction occurs—known as the **force-velocity relationship.**

Based on experiments on isolated muscles, the force-velocity relationship indicates that the velocity of a muscle contraction is a function of the load being lifted.[55] From a clinical perspective, the relationship can be stated with the variables reversed; that is, the force generated is a function of the velocity of the muscle contraction (Fig. 3–16). In a concentric muscle contraction, as the contractile velocity of the contraction decreases, the force production by the muscle increases. In an isometric contraction, the contractile velocity is zero and force production is potentially greater than for a concentric contraction. In an eccentric contraction, as the contractile velocity increases, the potential force produced by the lengthening muscle increases and then plateaus. The net effect is that the potential for force generation is greatest for an eccentric contraction (regardless of speed) and least for a concentric contraction (regardless of speed). Not only is this relationship seen in experimental conditions with isolated muscles lifting a load, but it is also seen, to some degree, in intact muscles moving bony levers.[56–58] Lastly, remember that the speed of shortening is related to muscle fiber type as well as to the length of the muscle fiber. Note that muscle strain injuries often occur during high velocity eccentric muscle actions such as sprinting. In such activities, it is assumed that the force-velocity curve would predict that the muscles were producing high forces, which potentially lead to injury of the muscle or tendon.

FIGURE 3-16

The skeletal muscle force-velocity curve. At maximum velocity of muscle shortening (concentric contraction), no force is produced (in other words, maximum velocity of shortening can be attained only with no load on the muscle). As shortening velocity decreases, the force that the muscle can develop during a concentric contraction increases. At zero velocity, the muscle contracts isometrically. Force increases dramatically and then plateaus when the muscle is lengthened actively (eccentric contraction).



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It was noted earlier that the length-tension relationship must be combined with the

force-velocity relationship to understand force production because both sarcomere length and velocity of contraction affect the development of muscle tension. The length-tension curve data is based on isometric contractions (constant velocity). The fact that most human movements do not occur at a constant velocity of contraction complicates our ability to fully understand force production because the force will vary with changing velocity and changing length.



Foundational Concepts 3–3

Factors Affecting Active Muscle Tension

Adding to our understanding of active tension development, the muscle force will be modulated by several additional factors:

1. The number of muscle fibers in a motor unit (which affects the magnitude of the response to a stimulus).
2. The diameter of the axon innervating a motor unit (which determines the conduction velocity of the nerve impulse).
3. The number of motor units that are firing at any one time (which affects the total force response of the muscle).
4. The frequency of motor unit firing (which affects the total force response of the muscle).
5. The greater the number of cross-bridges that are formed, the greater the tension.
6. Muscles that have large physiological cross sections are capable of producing more tension than are muscles that have small cross sections.
7. Tension increases as the velocity of active shortening decreases and as the velocity of active lengthening increases.

Types of Muscle Action

Isometric, concentric, and eccentric muscle contractions were previously introduced in

relation to what was occurring at the sarcomere level. To review: When a muscle fiber is activated so that cross-bridges form, the sarcomeres in the fiber will either stay at constant length, shorten, or lengthen, depending on the load that is applied. An isometric contraction occurs when the muscle is activated and the sarcomere does not change length; a concentric contraction occurs when the sarcomere shortens; and an eccentric contraction occurs when the sarcomere lengthens (the load is greater than the force of the sarcomere). An older term, *isotonic contraction,* is not used here because it refers to equal or constant tension, which rarely if ever occurs during normal human movements.

What happens at the sacromere or fiber level can be applied to the whole muscle. When the whole muscle is activated and the bones it is attached to do not move, the muscle action is called an isometric contraction. Holding a hand weight without changing the elbow joint angle means that the muscle is contracting isometrically. During an isometric contraction, no "work" is being done because the joint is not moving. The formula for work is $W = F \times d$, where W is work, F is the force created by the muscle, and d is the distance that the object is moved. Despite the fact that no work is being done, energy is being expended through the hydrolysis of ATP (see Fig. 3–5).

During a concentric contraction, the bones to which the muscle attaches move closer together as the whole muscle shortens. **Positive work** is being done by the muscle because the joint moves through a ROM. Concentric contractions expend the highest amount of energy of the three types of muscle contraction. During an eccentric contraction, the contiguous bones move away from each other as the muscle tries to control the external force that is creating movement of the bony segment(s), with the

muscle elongating (lengthening) as the joint moves through its ROM. The work that is being done during an eccentric contraction is called **negative work;** the work is done *on* the muscle rather than *by* the muscle because of an external force that is larger than the internal muscle force. In eccentric contractions, the muscle expends the least amount of energy compared with concentric and isometric contraction.

The amount of tension that can be developed in a muscle varies according to the type of contraction, as was seen in the force-velocity relationship. A greater amount of tension can be developed in an isometric contraction than in a concentric contraction because there is time to recruit more motor units.[59] In general, the tension developed in an eccentric contraction is greater than what can be developed in an isometric or concentric contraction.[60] However, this relationship may not hold true for all muscles at all points in a joint's angle of motion.[57,61,62] The greater tension development in a muscle during an eccentric contraction than during a concentric contraction may be due, in part, either to mechanical factors in the attachment and detachment of cross-bridges or to alterations in the neural activation of the muscle.[63]

We typically think of a muscle contraction as occurring with the proximal segment (so-called "origin") fixed while the distal segment (so-called "insertion") moves. During normal functional activities, however, the proximal segment often moves while the distal segment is stationary. For example, during a pull-up exercise (Fig. 3–17), the brachialis and biceps brachii move the humerus while the forearm (ulna and radius) remain basically stationary. We sometimes refer to this as the **reverse action** of the muscle. Whether the distal or proximal segment moves when a muscle contracts is dependent upon the desired motion, the external forces, and the ability to stabilize

other joints. Therefore, in the example of a pull-up, the brachialis and biceps brachii are shortening (concentric contraction) to raise the humerus (and the attached body).

FIGURE 3-17

In a pull-up exercise, the brachialis and biceps brachii flex the elbow by moving the proximal segment (humerus) toward the distal segment (forearm), sometimes referred to as the “reverse action” of the muscle.



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Production of Torque

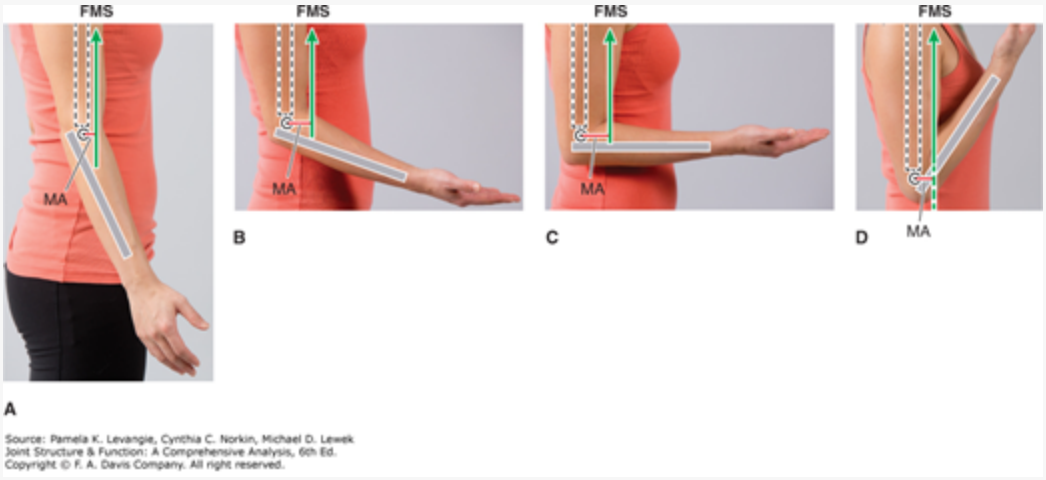
Clinicians often assess a patient's muscle strength. Whether strength is assessed using an instrument (such as an isokinetic device) or by manual resistance, we are actually assessing the amount of joint torque that the muscle can produce. In many physiological experiments on muscle, the muscle is removed from the bone so that the actual muscle force can be measured as the muscle is activated. When the muscle is

attached to bones in the body, the muscle still produces a force, but it is the rotational force (torque) on the segment that is being assessed. The muscle's ability to produce torque (joint rotation) is a function of both muscle force and the moment arm of the muscle.

As we know, the moment arm of the muscle can change with joint position, thereby affecting the torque being produced. If the biceps brachii is producing a consistent 10 N of force, as shown in Figure 3–18, the torque (muscle "strength") will be greatest in Fig. 3–18C and least in Fig. 3–18A. We now know, however, that muscle length is also changing and that the muscle's ability to produce force (total muscle tension or Fms) will not remain constant but will vary as the muscle is lengthened or shortened. In addition, during dynamic movements, the velocity of the shortening or lengthening affects the amount of active force that the muscle produces, thus affecting the total muscle force and torque production.[4,57]

FIGURE 3-18

The torque generated by the muscle changes as the moment arm of the muscle also changes during joint motion, even if the force production (Fms) remains constant. However, both the active and the passive tension will vary with the length of the active muscle, as well as with the type and velocity of the muscle contraction.

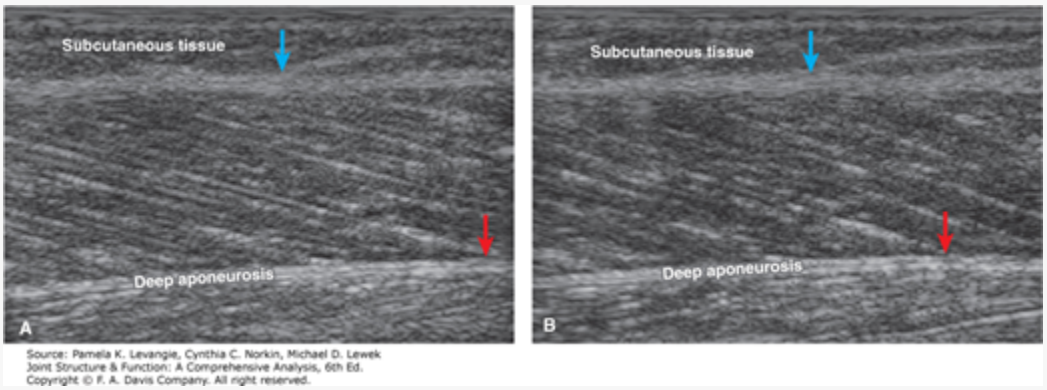


Interaction of Muscle and Tendon

The interaction of muscle and tendon (including the aponeurosis) during muscle contraction and movement has some important functional implications. Let us begin with a simple example. During an isometric contraction, the muscle actually shortens slightly and the tendon lengthens slightly (see Fig. 3–12B). In many muscles, the tendon may lengthen by as much as 10% of its resting length during an isometric contraction (Fig. 3–19).[64–68] The compliance of the tendon (or ability to lengthen under load) is important in augmenting the torque production of the muscle. This is the basis for **plyometric exercises**, in which the muscle/tendon complex is stretched before a forceful concentric contraction. The stretch immediately before the concentric contraction helps produce a much greater torque during the concentric contraction. Although the exact mechanism for the increase in torque is debated, evidence shows that the muscle fibers tend to stay at a relatively constant length (isometric) while the tendon lengthens, storing energy to be used during the concentric contraction.[69–73]

FIGURE 3-19

Ultrasound image of the medial gastrocnemius. **A.** Relaxed. **B.** During an isometric contraction. In both **A.** and **B.,** the blue arrow is a fixed reference point in the subcutaneous fat while the red arrow is a fixed point on the aponeuroses of the tendon. Note how the insertion point on the aponeurosis (*red arrow*) in **B.** moves to the left (proximal) compared to the same reference point in **A.** as the tendon stretches during an isometric contraction.



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Isokinetic Exercise and Testing

Advances in technology have led to the development of testing and exercise equipment that provide for the manipulation and control of some of the variables that affect muscle function. In isokinetic exercise and testing or isokinetic muscle contraction, the angular velocity of the bony component is preset and kept constant by a mechanical device throughout joint ROM.[74] The concept of an "isokinetic contraction" may not be so much a type of muscle action as it is a description of joint motion because the muscle fascicles do not shorten at constant velocity during an isokinetic movement.[75] Because the angular velocity of joint motion is constant ($\Sigma T = 0$) with the isokinetic

device, the resistance torque is equivalent to the torque produced by the muscle at all points in ROM. Therefore, as the torque produced by a muscle changes, the magnitude of the torque of the resistance changes equivalently. This provides an excellent means of testing muscle strength (torque). Isokinetic devices such as a Biodex are used in many clinics.

The proposed advantage of isokinetic exercise over free weight lifting is that isokinetic exercise theoretically accommodates for the changing torque of the muscle as the muscle's moment arm and length-tension vary throughout ROM. As long as the preset speed is achieved, the isokinetic device consistently matches the torque of the muscle.[74]

The maximum isokinetic torque for a concentric contraction obtained at high-angular velocities will be less than the torque produced at low-angular velocities. This decline in torque with increasing contraction velocity is expected on the basis of the force-velocity relationship of muscle. In fact, for any particular point in the joint range of motion, isometric torque values are higher than concentric torque values at any velocity for that same point in the joint range. Therefore, the closer the angular velocity of a concentric isokinetic contraction approaches to zero (0 = isometric), the greater the isokinetic torque.[76,77]

Isokinetic equipment is used extensively for determining the amount of joint torque that a muscle can develop at different velocities, for strength training, and for comparing the relative strength of one muscle group with another. Most isokinetic devices also permit quantification of eccentric muscle torque. Isokinetic evaluation of muscle strength has provided much important information about muscle function, and

isokinetic exercise is effective for gaining strength. However, there are some limitations to isokinetic evaluation and exercise. For example, at higher speeds, the amount of range within which movement is at constant velocity is decreased, and functional movements are rarely performed at constant velocity. Some research shows that isokinetic testing or exercising may differentiate performance in functional tasks or may enhance the training effect for functional activities, and some research shows that it does not.[78–81]



Expanded Concepts 3–4

At various stages of rehabilitation for an injured muscle or muscle group, it is advantageous to limit the force that a muscle produces because we do not want to injure healing muscle or tendon tissue. The most obvious way to decrease muscle force is to decrease the applied resistance. However, based on the discussions in this section, the clinician may choose to exercise the patient on an isokinetic device (or through some other mechanism that provides a constant external resistance), at a higher velocity which decreases the muscle force. Although this may not be a common choice for exercise training, it may prove useful in certain situations.



Foundational Concepts 3–4

Summary of Intrinsic and Extrinsic Factors Involving Active Muscle Tension

The velocity of muscle contraction is affected by:

- *Recruitment order of the motor units:* Units with slow conduction velocities are generally recruited first.
- *Type of muscle fibers in the motor units:* Units with type II muscle fibers can develop maximum tension more rapidly than units with type I muscle fibers; rate of cross-bridge formation, breaking, and re-formation may vary.
- *The length of the muscle fibers:* Long fibers have a higher shortening velocity than do shorter fibers.
- *The magnitude of the resistance:* For a given muscle force production, a greater resistance to the muscle action will result in a slower contraction.

The magnitude of the active tension is affected by:

- *Size of motor units:* Larger units produce greater tension.
- *Number and size of the muscle fibers in a cross section of the muscle:* The larger the cross section, the greater the amount of tension that a muscle may produce.
- *Number of motor units firing:* The greater the number of motor units firing in a muscle, the greater the tension.
- *Frequency of firing of motor units:* The higher the frequency of firing of motor units, the greater the tension.
- *Sarcomere length:* The closer the length is to optimal length, the greater the amount of isometric tension that can be generated.
- *Fiber arrangement:* A pennate fiber arrangement gives a greater number of muscle fibers and potentially a larger cross-sectional area, and therefore a greater amount of tension may be generated in a pennate muscle than in a parallel muscle.
- *Type of muscle contraction:* An isometric contraction can develop greater tension than a concentric contraction; an eccentric contraction can develop greater tension than an isometric contraction.
- *Velocity:* As the speed of shortening increases, tension decreases in a concentric contraction. As speed of active lengthening increases, tension increases in an eccentric contraction.

Classification of Muscles

Individual muscles may be named in many different ways based on shape (rhomboids like a rhombus, the deltoid like a delta or triangle), the number of heads (*bi*ceps, *tri*ceps, *quad*riceps), location (biceps *femori*s, *tibia*lis *anterior*), or a combination of location and function (*extensor digit*orum *long*us, *flexor pollicis* (thumb) *brevis*). Groups of muscles are categorized either by the actions they perform or the particular role they serve during specific actions. When muscles are categorized by action, they can be considered for any particular action as “flexors”, “extensors”, or “rotators”. When muscles are categorized according to role, individual muscles or groups of muscles may be referred to as the **agonists, antagonists,** or **synergists** for a particular motion. In this

section, we explore the classification of muscle based on the role of the muscle during movement as well as the classification based on muscle architecture and moment arm.

Based on the Role of the Muscle in Movement

The term **prime mover (agonist)** is used to designate a muscle whose role is to produce a desired motion at a joint. If flexion is the desired action, the flexor muscles are the prime movers. However, it is common to limit the term *prime mover* to those flexors that are first recruited or that produce the action with greatest potential force. When flexion is desired, the extensor muscles that would produce the opposite motion are called the **antagonists.** The desired motion is not opposed by an active contraction of the antagonist(s), but the antagonistic muscles have the *potential* to oppose the action.

Ordinarily, when an agonist (for example, the hamstrings) is called on to perform a desired motion (knee flexion), the antagonist muscle (quadriceps) does not contract concomitantly. If, however, the agonist and the potential antagonist contract simultaneously, then **co-contraction** occurs (Fig. 3–20). Co-contraction of muscles (purposeful simultaneous contraction of an agonist and antagonist) around a joint can help to provide stability for the joint and represents a form of synergy that may be necessary in certain situations. Co-contraction of muscles can be undesirable when a desired motion is prevented by involuntary co-contraction, such as occurs in disorders affecting the control of muscle function (e.g., cerebral palsy).

FIGURE 3-20

Co-contraction of the quadriceps and the hamstrings provide simultaneous flexion and extension torques at the knees to stabilize

the knees as the hips extend in a bridging exercise.



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Muscles that help the agonist (prime mover) to perform a desired action are called **synergists.** The anterior deltoid muscle is a strong flexor of the glenohumeral joint and typically is considered the prime mover. The much smaller coracobrachialis can also produce shoulder flexion but much more weakly. The coracobrachialis, therefore, is sometimes referred to as a synergist or may be referred to as a co-agonist for shoulder flexion.

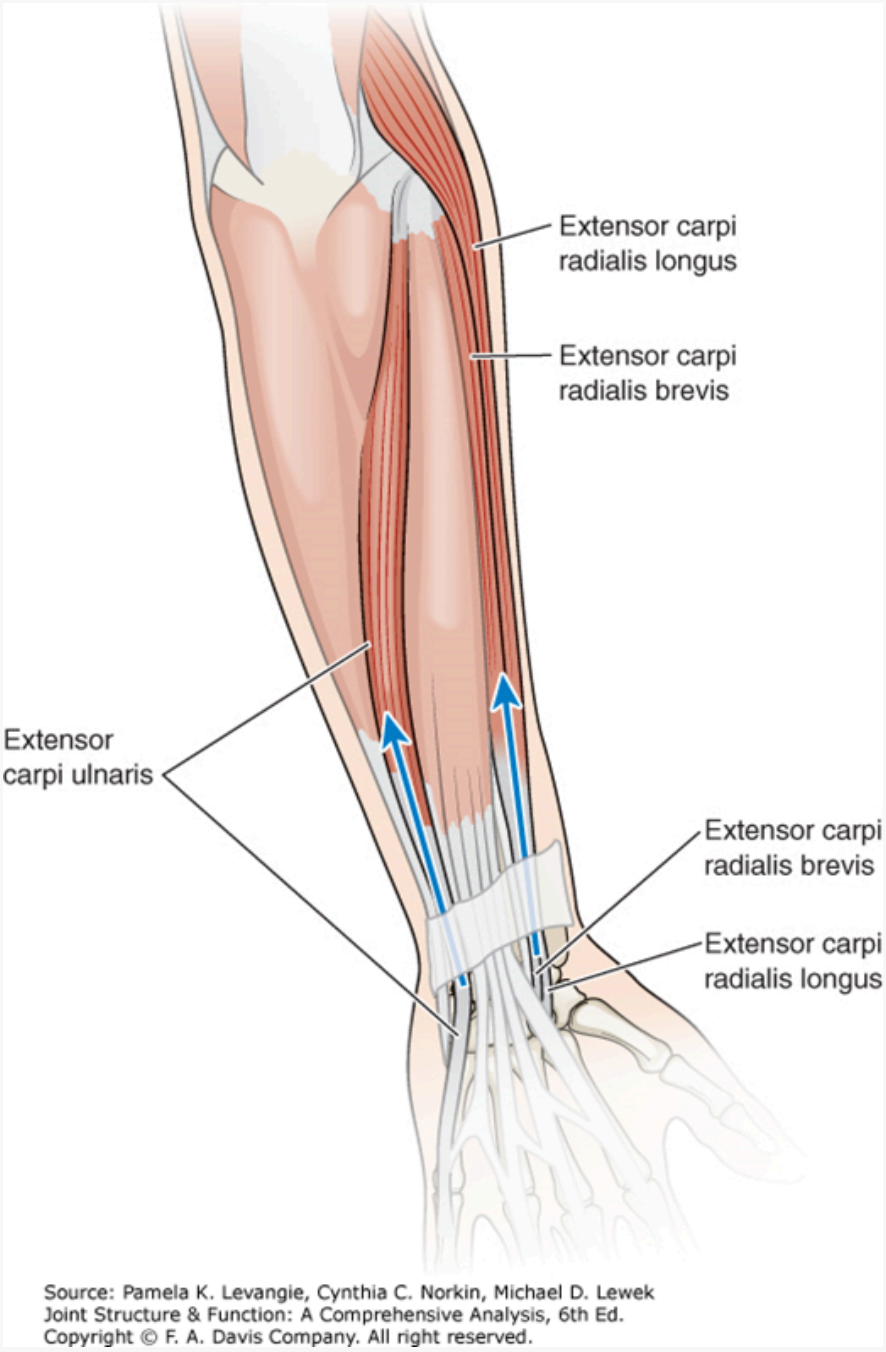
Synergists may assist the agonist directly by helping to perform the desired action (e.g., the coracobrachialis), or the synergists may assist the agonist indirectly either by stabilizing a segment or by preventing an undesired action. In Figure 3–15B we saw the example where the wrist extensors worked synergistically with the finger flexors to

maintain a length-tension in finger flexors to facilitate effective grasp. The wrist extensors prevent unwanted wrist flexion (as in Fig. 3–15A) that would weaken the finger flexors.

Sometimes the synergistic action of two muscles is necessary to produce a motion that neither could produce alone. The extensor carpi radialis longus and brevis by themselves will produce simultaneous wrist extension and radial deviation. The extensor carpi ulnaris by itself will produce simultaneous wrist extension and ulnar deviation. Working together, the extensor carpi radialis longus and brevis and the extensor carpi ulnaris produce pure wrist extension, with radial and ulnar deviation components of the muscles cancelling each other out (Fig. 3–21). During wrist extension, the long finger extensors (extensor digitorum communis) may assist with wrist extension as a synergist.

FIGURE 3-21

Synergistic muscle activity. When the extensor carpi radialis longus and brevis work together with the extensor carpi ulnaris, the deviations of the wrist (radial and ulnar, respectively) offset each other and the muscles work synergistically to produce wrist extension.



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Based on Muscle Architecture

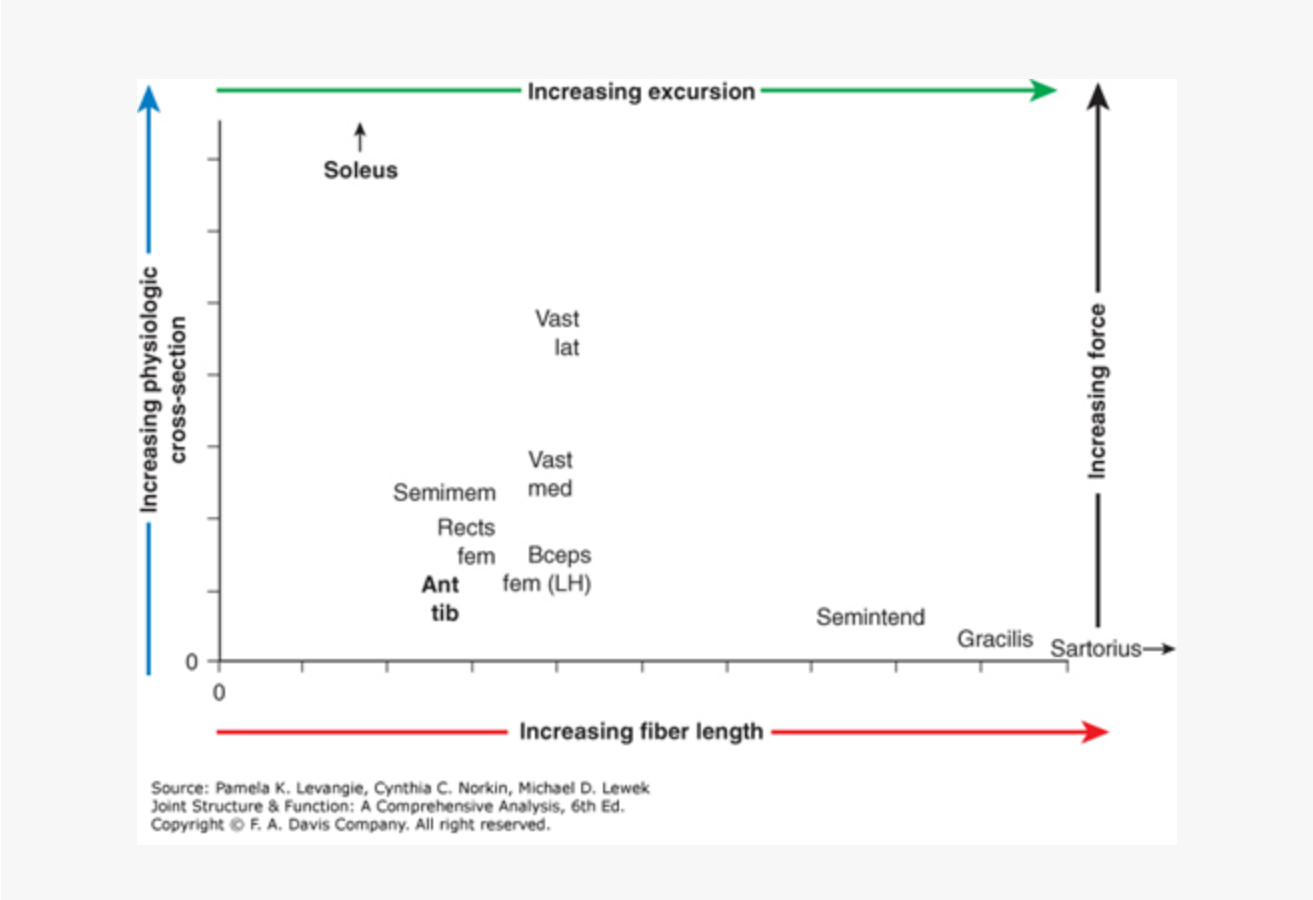
Placing muscles into functional categories, such as flexor/extensor or agonist/antagonist, helps to simplify the task of describing the many different muscles and explaining their actions. However, muscles can change roles. A potential antagonist in

one instance may be a synergist in another situation. An example of this can be found in the preceding discussion. The extensor carpi radialis longus is an antagonist to the extensor carpi ulnaris during ulnar deviation of the wrist but, as noted, is a synergist during wrist extension. Despite this apparent change in role, muscles that have similar functions also have similar architectural characteristics. This is intuitive, because muscle architecture plays such an important role in determining the potential force and velocity of muscle contraction, and several studies have confirmed that the functional groups of muscles have similar architecture. Examples of this may be seen in experiments with both animals and humans.[4,39,82–85]

In the lower extremity, the knee extensors have a shorter fiber length and large cross-sectional area as compared with knee flexors like the semitendinosus, gracilis, and sartorius that have a longer fiber length and smaller cross-sectional area. Whereas these knee flexors correspondingly have a decreased ability to produce force compared with the knee extensors, the ability of longer muscles to shorten (increased excursion) over both the hip and the knee joint allow those muscles to complete a greater range of motion and, therefore, a greater velocity of contraction (Fig. 3–22). The soleus (a plantarflexor) has a short fiber length but a large cross-sectional area that sets it apart from the anterior tibialis (an ankle dorsiflexor) that has longer fiber length but a smaller cross-sectional area. Although we can classify many muscles based on architecture, there are obvious exceptions. For example, the semimembranosus (a knee flexor) has a similar fiber length to the knee extensor muscles.

FIGURE 3-22

Muscle architectural relationships.[4,82] The soleus (a plantarflexor) has a large physiologic cross-sectional area (PCSA) (left y-axis) that is off-scale, with a corresponding ability to produce substantial force (right y-axis) that contrasts with the small PCSA and low force production capacity of the anterior tibialis (a dorsiflexor). However, the longer fibers of the anterior tibialis (Ant tib) (lower x-axis) give it a greater excursion (ability to shorten) (upper x-axis). The vastus lateralis (vast lat) and vastus medialis (vast med) are knee extensors with substantially greater PCSA and force generating capability than knee flexors like the semitendinous (semintend), gracilis, and sartorius (with the fiber length of the sartorius being off-scale). However, those knee flexors have much greater shortening capability (excursion) that enhances their ability to produce range across both the hip and the knee in contrast to the one-joint vasti muscles, biceps fem (LH), biceps femoris, long head; Rectus fem, rectus femoris; semimem: semimembranosus.



Based on Length of the Moment Arm

The relationship of a muscle to the joint has also been used to classify muscles into groups. The length of the muscle's moment arm is an important component of determining the joint torque and, in combination with the fiber length, the range through which the muscle can move the joint.[4] The ratio of the fiber length to the moment arm provides a way of identifying which factor plays a greater role in the production of the joint torque and in determining the resulting ROM at the joint. For example, the ratio of fiber length to the moment arm is much higher in the wrist extensor muscles compared with the wrist flexor muscles. This suggests that wrist extensor fiber length plays a greater role than does the moment arm and that the wrist flexor moment arm plays a greater role than fiber length.[48]

Although all skeletal muscles adhere to a general basic structural design, a considerable amount of variability exists among muscles regarding the number, size, arrangement, and type of muscle fibers. Therefore, attempts to classify muscles into only a few groups may be inappropriate. According to the evidence that subpopulations of motor units from a muscle, rather than from groups of muscles, appear to work together for a particular motor task, a more appropriate way of describing muscle action might be in terms of motor units.[21] However, more research needs to be performed in this area before a motor unit classification system can be widely used and accepted.

Factors Affecting Muscle Function

In addition to the large number of factors affecting muscle function that have been examined thus far, a few other factors need to be considered:

- Types of joints and location of muscle attachments
- Number of joints crossed by the muscle
- Passive insufficiency
- Sensory receptors

Types of Joints and Location of Muscle Attachments

The type of joint affects the function of a muscle because the structure of the joint determines the type of motion that will occur (flexion and extension) as well as the range of motion. The muscle's location or line of action relative to the joint determines which motion the muscle will perform. In general (but with exceptions), muscles that cross the anterior aspect of joints are flexors, whereas muscles located on the posterior

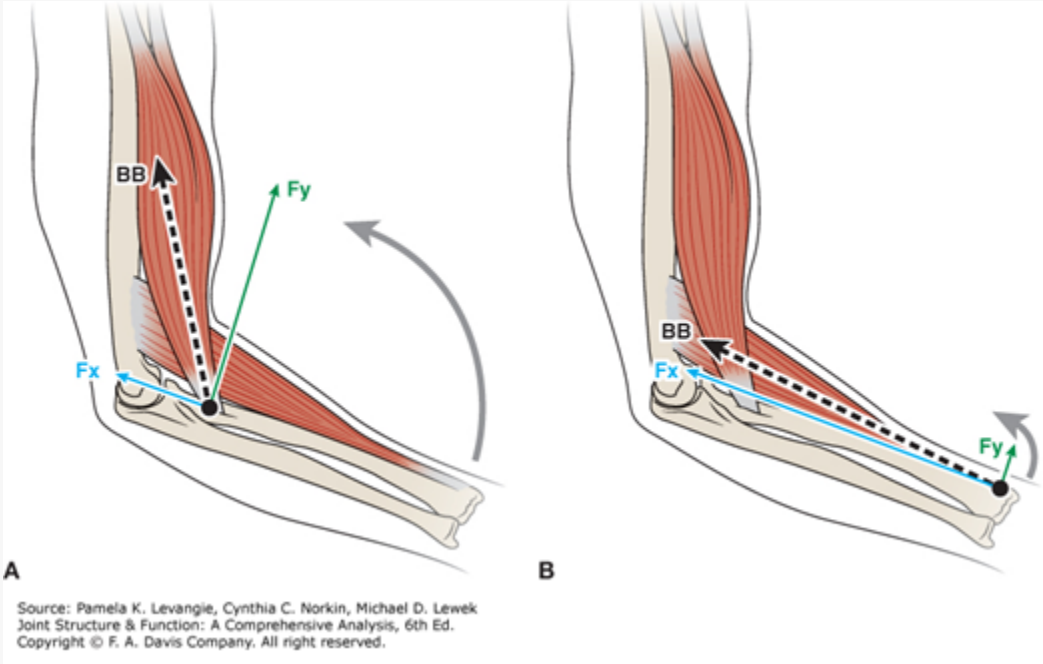
aspect of joints are typically extensors. Muscles located laterally and medially serve as abductors and adductors, respectively, and may also serve as rotators.

Muscles like the biceps brachii, which has a distal attachment that lies close to a joint axis, are usually able to produce a greater range of the bony lever (Fig. 3–23A) than those like the brachioradialis, which has a distal attachment at a distance from the joint axis (Fig. 3–23B). However, muscles with attachments farther from the joint are designed to provide stability for the joint because most of their force is directed toward the joint compression ($F_X > F_Y$) throughout the joint range. As has already been identified in past chapters, the biceps brachii's relative contribution to stability will be less at 90° than when the elbow is in full extension (see Fig. 1–36). At full extension, a greater proportion of the biceps brachii force is directed toward the joint (F_x), resulting in greater compression. A muscle provides maximum joint stabilization at the point at which its compressive component is greatest and, correspondingly, its rotary component is least.

FIGURE 3-23

Location of muscles. **A.** Because the attachment of the biceps brachii (BB) on the radius lies close to the elbow joint, its substantial rotary component (Fy) will create a large arc of motion for distal forearm. **B.** The brachioradialis (BR) is attached to the radius far from the elbow joint: its small rotary component will create only a small arc of motion for the distal forearm. It should be noted, however, that the compressive component of the brachioradialis (Fx) is substantially

greater than the compressive component of the biceps, making the brachioradialis a stronger stabilizer.



Usually one group of muscles acting at a joint can produce more torque than another group of muscles acting at the same joint. Disturbances of the normal ratio of agonist-antagonist strength may create a muscle imbalance at the joint and may place the joint at risk for injury. For example, weakness of the hip external rotators causes a strength imbalance between hip external and internal rotation that may result in excess hip internal rotation during gait. Agonist-antagonist strength ratios for normal joints are often used as a basis for establishing treatment goals after an injury to a joint.

Number of Joints

Many functional activities require the coordinated movement of several joints that are controlled by combinations of muscles that cross the joints requiring coordination. To produce a purposeful movement pattern, many believe that motor control minimizes

necessary muscle force to accomplish the task (least motor unit activity) and thus minimizes energy expenditure and muscle fatigue. These strategies of motor control attempt to ensure that movement is done efficiently. There are several theories that attempt to explain differences in how the one joint and multi-joint movements are controlled.[86] This is a current topic of research that will likely continue for several years.

Single-joint muscles tend to be recruited for force and work production, more often as concentric and isometric contractions. Multi-joint muscles, in contrast, tend to be recruited to control the fine regulation of torque during dynamic movements involving eccentric more often than concentric muscle actions,[87–89] although there are exceptions to these general assumptions. In some animals, for example, the biarticular muscles tend to contract concentrically, producing positive work whereas the single-joint muscles contract eccentrically.[90]

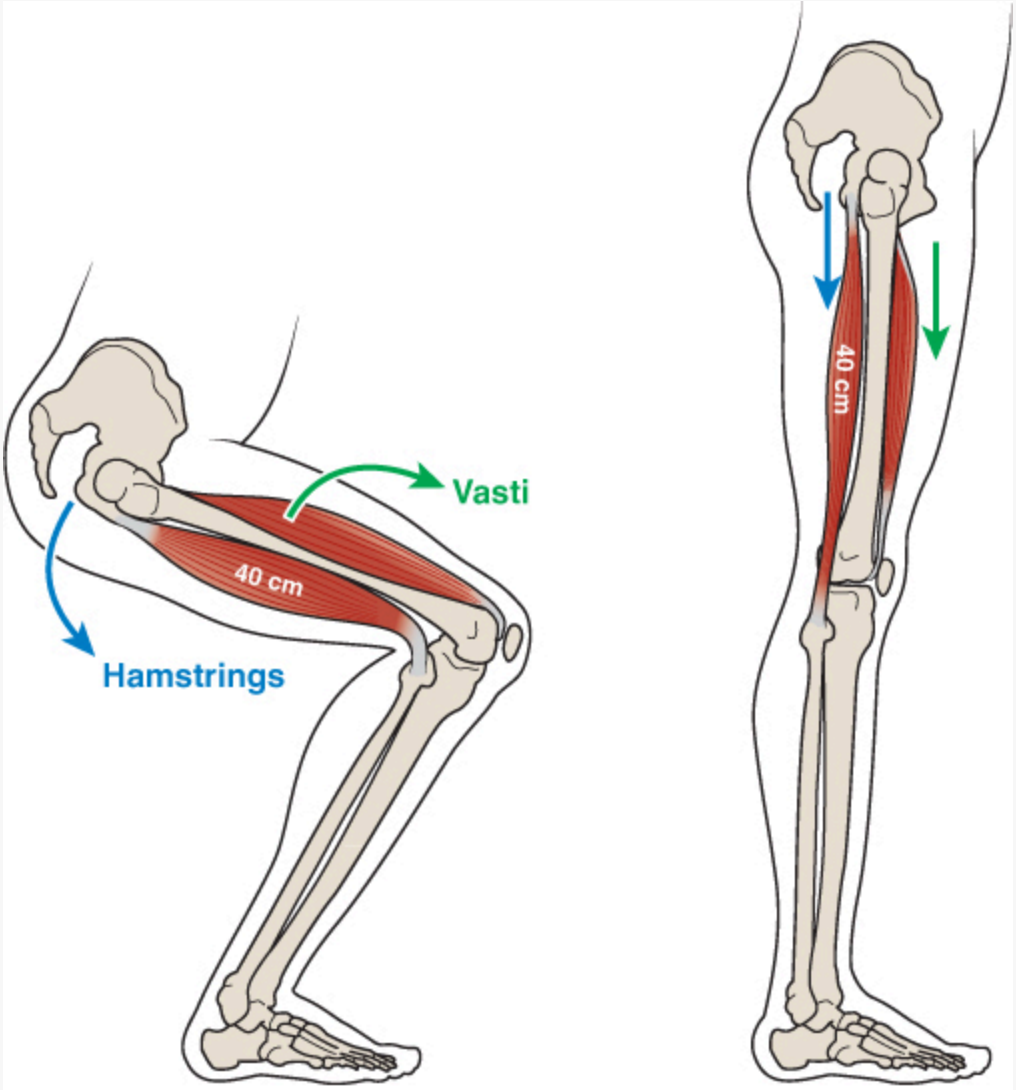
Multi-joint muscles tend to be recruited during more complex motions requiring movement around multiple axes. When elbow flexion with concurrent supination is needed, the biceps brachii (a multi-joint muscle) will function with the added contribution of the brachialis (a single-joint muscle).[91] However, if elbow flexion with the forearm in pronation is desired, this will be accomplished primarily with the brachialis. When the biceps participates in that movement, the undesired supination function of the biceps brachii would necessitate simultaneous activity of a forearm pronator.

Single-joint and multi-joint muscles may also work together in such a way that the single-joint muscle can assist in the movement of a joint that it does not cross.[89,92] We saw this in Chapter 1 (Fig. 1–45) where the gluteus maximus helped extend the knee. In

another example, the movement of standing up from a chair requires simultaneous knee and hip extension. The single-joint knee extensors (vastus medialis, intermedius, and lateralis) may actually “assist” the biarticular hamstrings (and the one joint gluteus maximus) with hip extension, although the vasti do not cross the hip. Consider that the hamstrings are active and simply are attempting to maintain a constant length (e.g., 40 cm) as the person rises from the chair. As the vasti muscles shorten and extend the femur on the weightbearing tibia, the extending knee will pull on the active hamstring muscles that essentially act as an inextensible rod. With increased knee extension, the hamstrings will pull the pelvis downward posteriorly, resulting in hip extension with little or no shortening of the hamstrings. (Fig. 3–24).

FIGURE 3-24

Single joint quadriceps muscles contributing to hip extension. The vasti muscles of the quadriceps extend the knee while the active hamstring muscles maintain a constant length (40 cm, for example); the knee extension action of the vasti muscles pulls on the “inextensible” hamstrings that then pull on the posterior pelvis to produce hip extension.



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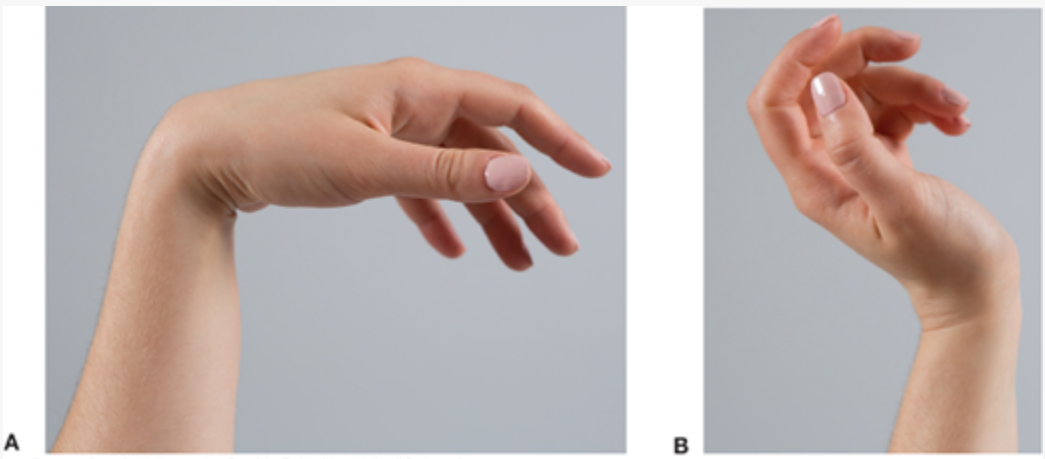
Passive Insufficiency

If a person's elbow is placed on a table with the forearm in a vertical position and the relaxed hand drops forward into wrist flexion, the fingers tend to extend (Fig. 3–25A). Extension of the fingers occurs because the finger extensors are being stretched over the flexed wrist and are insufficiently long (extensible) to permit the fingers to remain

flexed. The insufficient length in a passive muscle is termed **passive insufficiency.** If the person moves his or her wrist backward into wrist extension, the relaxed fingers will tend to flex (Fig. 3–25B). Flexion of the fingers occurs because the finger flexors are stretched over the extended wrist and are insufficiently long to permit the fingers to remain extended. The “insuffiency” part of passive insufficiency might imply an abnormal situation, but passive insufficiency can, in fact, be quite normal.

FIGURE 3-25

Passive insufficiency. **A.** When the wrist is flexed, the inactive finger extensors are stretched over the wrist with enough passive tension to cause the metacarpophalangeal (MCP), proximal interphalangeal (PIP), and distal interphalangeal (DIP) joints to extend. **B.** When the wrist is extended, the inactive finger flexors are stretched over the wrist with enough passive tension to cause the MCP, PIP, and DIP joints to flex. These are examples of “passive insufficiency” because the extensibility of the passive muscles and tendons is insufficient to allow fingers to remain flexed (A) or extended (B).



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Joint Structure & Function: A Comprehensive Analysis, 6th Ed.
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Under normal conditions, one-joint muscles rarely, if ever, are of insufficient extensibility to limit full ROM at the joint. Two-joint or multi-joint muscles, however, frequently are of insufficient extensibility to permit full ROM simultaneously at all joints crossed by these muscles. The passive tension developed in these stretched muscles is sufficient to either cause motion (as shown by finger flexion and extension in Fig. 3–25) or limit motion. For example, the biarticular rectus femoris (a hip flexor and knee extensor) may limit the available range of knee flexion if the rectus femoris is stretched over an extended hip. Similarly, the biarticular hamstrings (hip extensors and knee flexors) may limit full knee extension if the hamstrings are stretched over a fully flexed hip. Passive insufficiency can also occur simultaneously with active insufficiency. In Figure 3–15A, the finger flexion ROM was insufficient to allow effective grasp in the wrist flexed position. Not only were the active finger flexors unable to shorten sufficiently to complete flexion at the wrist and finger joints at the same time (active insufficiency), but the inactive finger extensors were simultaneously passively insufficient. That is, the finger extensors were being passively stretched over all of the joints that they cross and were not sufficiently extensible to allow full flexion at the joints. In this example, the active insufficiency of the finger flexors is actually accentuated by the passive insufficiency in the finger extensors. Whereas problems with passive insufficiency (with or without concomitant active insufficiency) are typically not encountered in usual activities of daily living, issues with passive insufficiency may be encountered in sports activities.

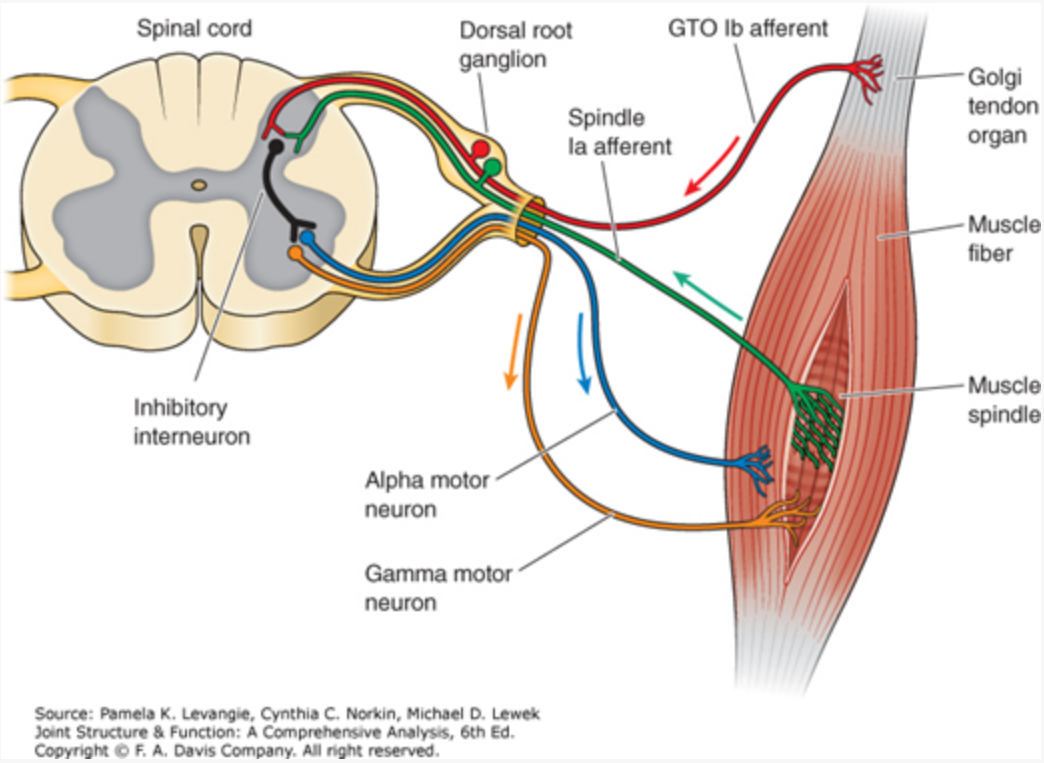
Sensory Receptors

Normal motor control resulting in voluntary movements depends on the coordination of descending motor pathways from the cortex, muscle actions, and a constant flow of

sensory information. When performing or participating in athletic events, highly skilled dancers and athletes always seem to make it look so easy. To achieve this level of skill, the movements were practiced frequently, requiring constant sensory information from many systems. In the muscle, feedback comes from particularly two important sensory receptors: the **Golgi tendon organ (GTO)** and the **muscle spindle.** The Golgi tendon organs, located in the tendon at the myotendinous junction (Fig. 3–26), are sensitive to tension and may be activated either by an active muscle contraction or by an excessive passive stretch of the muscle. When the Golgi tendon organs are excited, they send a message to the central nervous system via Ib afferent neurons to adjust the muscle tension. The fine-tuning of muscle tension is not accomplished alone, however, because other reflex systems (muscle spindles) are also providing feedback to the muscle.

FIGURE 3-26

The function of the Golgi tendon organ during gait.



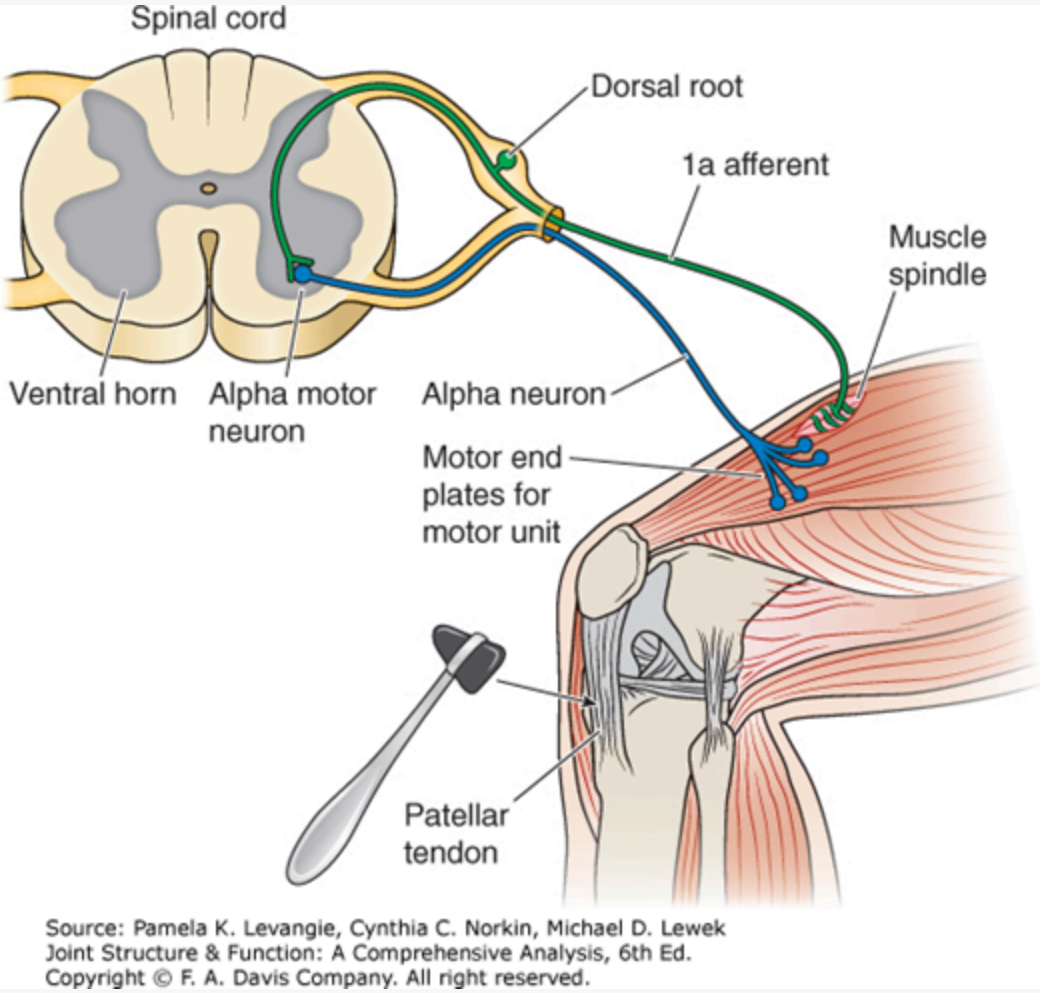
The **muscle spindles** consist of 2 to 10 specialized muscle fibers called **intrafusal fibers** that are enclosed in a connective tissue sheath and are interspersed throughout the muscle. These spindle fibers are sensitive to the length and the velocity of lengthening of the muscle fibers (also known as **extrafusal fibers**). The spindles send messages to the central nervous system about the state of stretch of the muscle. When the muscle is passively stretched, the spindles send a message to the spinal cord and central nervous system via the group Ia afferent neurons (Fig 3–26).Depending upon the extent of this message and the other inputs into the spinal cord, movement will be affected. When the muscle fiber shortens, the spindles stop sending messages because they are no longer stretched. When the muscle spindle signal decreases, the central nervous system sends a message via the gamma motor neurons to the intrafusal muscle fibers in the spindle to contract, which stretches the center of the spindle slightly to increase the

sensitivity so that the spindle is once again able to respond to the length change in a now shorter muscle.

The muscle spindle is responsible for sending the message to the muscle in which it lies to contract when the tendon of the muscle is tapped with a hammer (Fig. 3–27). The quick stretch of the muscle caused by tapping the tendon activates the muscle spindles, and the muscle responds to the unexpected spindle message with a brief contraction. This response is called by various names: for example, **deep tendon reflex (DTR), muscle spindle reflex (MSR),** or simply **stretch reflex.** Both the Golgi tendon organs and the muscle spindles provide constant feedback to the central nervous system during movement so that appropriate adjustments can be made, and they help protect the muscle from injury by monitoring changes in muscle length.

FIGURE 3-27

The stretch reflex. When the muscle spindle is stretched by the tap of the hammer, the 1a afferent neuron can send a message directly to the alpha motor neuron, which results in contraction of the stretched muscle. *(Adapted from Smith LK, Weiss EL, Lehmkuhl LD (eds): Brunnstrom's Clinical Kinesiology (ed. 5). Philadelphia, FA Davis, 1996;100; with permission.)*



The presence of the stretch reflex is beneficial for preventing muscle injury but presents a problem in treatment or fitness programs, in which stretching of a muscle is desirable for improving flexibility and restoring a full range of joint motion. Muscle contraction or reflex activation of motor units during intentional stretching of a muscle may create a resistance to the stretching procedure and makes stretching more difficult and possibly ineffective. Methods of stretching that may prevent reflex activity and motor unit activation during stretching have been investigated.[93,94] The non-contractile components of a muscle also provide resistance to stretching and need to be considered when a muscle-stretching program is implemented.

Receptors that lie in joint capsules and ligaments may have either a positive or a negative influence on muscle activity through their signals to the central nervous system. **Nociceptors** and other receptors in and around the joint can have flexor excitatory and extensor inhibitory action. Even a small joint effusion that is undetectable by the naked eye or pinching of a capsule can cause inhibition.[95–97]

Control of voluntary movements is not only accomplished via feedback systems such as Golgi tendon organs and the muscle spindles. **Feed-forward control** plays an important role in voluntary movement because it allows for anticipatory control of muscle actions. For example, in preparation for movements of the extremities, the abdominal and pelvic floor muscles contract to stabilize the trunk.[98,99] Feed-forward control relies on sensory input from the visual and auditory systems to learn appropriate movement patterns as well as to anticipate the control of movement.

Effects of Immobilization, Injury, and Aging

Immobilization

Immobilization affects both muscle structure and function. The effects of immobilization depend on immobilization position (shortened or lengthened), percentage of fiber types within the muscle, and length of the immobilization period.

In Shortened Position

A muscle immobilized in a shortened position adapts to the new resting position through the following structural changes:

- Decrease in the number of sarcomeres with a compensatory increase in sarcomere

length[100–102]

- Increase in the amount of perimysium[103]
- Thickening of endomysium[103]
- Collagen fibril orientation becoming more circumferential[104]
- Increase in ratio of connective tissue to muscle fiber tissue
- Decreased muscle mass and muscle atrophy[101,105,106]

Changes in function that result from immobilization in the shortened position reflect the structural changes. The decrease in the number of sarcomeres, coupled with an increase in the length of sarcomeres, displaces the length-tension relationship of the muscle so that the maximum tension generated by the muscle now corresponds to the new shortened position rather than the “typical” optimal length for that muscle. Although this altered capacity for developing tension may be beneficial while a muscle is immobilized in the shortened position, the muscle (often referred to as “adaptively shortened”) will not be able to return to typical function immediately after cessation of the immobilization. A muscle that has adapted to its shortened state will resist lengthening, thus potentially limiting joint motion. Furthermore, the overall tension-generating capacity of the muscle is decreased and the increase in connective tissue in relation to muscle fiber tissue results in an increase in stiffness to passive stretch.

In Lengthened Position

Muscles immobilized in the lengthened position exhibit fewer structural and functional

changes than do muscles immobilized in the shortened position. The primary structural changes are:

- An increase in the number of sarcomeres, resulting in a decrease in sarcomere length at the lengthened position
- Increased endomyseal and perimyseal connective tissue
- Muscle hypertrophy that may be followed by atrophy[101,103,107]

The primary functional changes in a muscle immobilized in a lengthened rather than in a shortened position are an increase in maximum tension-generating capacity and displacement of the length-tension curve close to the longer immobilized position. Passive tension in the muscle (often referred to as “adaptively lengthened”) approximates that of the muscle before immobilization.[101]

Prevention of the effects of immobilization in the shortened position may require only short periods of daily movement. With only 30 minutes of daily ROM activities out of the cast, the negative effects of immobilization in a shortened position were eliminated in animal models.[108,109] This beneficial effect of movement is not apparent when the frequency of movement is decreased to once per week over 3 weeks of immobilization.[110] If daily motion is not possible (e.g., due to fracture), then intensive re-mobilization with active and passive motion after immobilization can reverse the sarcomere number and connective tissue alterations that occur with immobilization.[111,112] Recall, however, that connective tissue structures are more likely to fail if remobilization is overly aggressive. In summary, a word of caution concerning

the interpretation of the response of muscle to immobilization: the studies on sarcomere adaptation to immobilization have all been done with specific muscles in animals. Human skeletal muscle sarcomere numbers may be capable of adaptation similar to that of animals in situations of clinical bone lengthening to compensate for a short limb. When lengthened very slowly over several weeks in a case study, the vastus lateralis muscle adapted with an increase in number of sarcomeres.[113] Despite this one case study, it is not clear whether these changes are apparent in all human muscles and under what conditions.

Injury

Overuse

Overuse may cause injury to tendons, ligaments, bursae, nerves, cartilage, and muscle. The common etiology of these injuries is repetitive trauma that does not allow time for complete repair of the tissue. The additive effects of repetitive forces lead to microtrauma which, in turn, triggers the inflammatory process and results in swelling. The tissue most commonly affected by overuse injuries is the musculotendinous unit.[114] Muscle and tendons can fatigue with repetitive submaximal loading, with rapidly applied loads, and when the active (contracting) musculotendinous unit is stretched by external forces.[115,116] Bursae may become inflamed with resultant effusion and thickening of the bursal wall as a result of repetitive trauma. Nerves can be subjected to compression injuries by muscle hypertrophy, decreased flexibility, and altered joint mechanics.[117]

Muscle Strain

Muscle strain injuries can occur from a single high-force contraction of the muscle while

the muscle is lengthened by external forces (such as body weight). The muscle usually fails at the interface between the muscle and tendon (myotendinous junction).[28,118,119] Subsequently, there is localized bleeding and a significant acute inflammatory response, resulting in swelling, redness, and pain.[120]

Eccentric Exercise-Induced Muscle Injury

Injuries to muscles may occur with even a single bout of eccentric exercise.[121] After 30 to 40 minutes of eccentric exercise (walking downhill) or as few as 15 to 20 repetitions of high-load eccentric contractions, significant and sustained reductions in maximal voluntary contractions occur. Also, a loss of coordination, **delayed-onset muscle soreness (DOMS),** swelling, and a dramatic increase in muscle stiffness have been reported. The DOMS reaches a peak 2 to 4 days after exercise.[122–124] DOMS occurs in muscles performing eccentric exercise but not in muscles performing concentric exercise.[122] The search for a cause of DOMS is still under investigation. It is known to be related to the forces experienced by muscles and may be a result of mechanical strain in the muscle fibers or in their associated connective tissues.[123,125–128] Morphological evidence shows deformation of the Z disc (Z-disc streaming) and other focal lesions after eccentric activity that induces soreness.[129] Biomechanical and histochemical studies have demonstrated evidence for collagen breakdown and for other connective tissue changes.[122]

Aging

Fiber Number and Fiber Type Changes

As a person ages, skeletal muscle strength decreases as a result of **sarcopenia** (the loss of muscle mass). Sarcopenia occurs as result of a loss of muscle fibers and a decrease in

the size of existing fibers. Some muscles (e.g., vastus lateralis) show a 25% to 30% loss of fibers in persons in their 70s and 80s.[130,131] It is commonly thought that there is a gradual decrease in the number and size of type II fibers, leaving the muscle with a relative increase in type I fibers. But more recent data using immunohistochemical methods of fiber typing (identifying the specific MHC content in the muscle) suggests that the proportion of type I and type II fibers remains relatively constant.[132] The maintenance of the proportion of fiber types may occur to a greater degree in elderly people who stay active, whereas the elderly who are inactive may have a greater proportion of type I fibers.[132] There is also a decrease in the number of motor units, and the remaining motor units have a higher number of fibers per motor unit.[133–136]

Connective Tissue Changes

Aging also increases the amount of connective tissue (endomysium, perimysium, and epimysium) within a muscle. It is generally assumed that the increased connective tissue results in decreased ROM and increased muscle stiffness,[137,138] although there have been reports that muscle stiffness may not change or may decrease with aging.[139–142] All of these changes in the muscle result in decreased muscle strength and, more importantly, a loss in muscle power. This loss of muscle power, or the ability to contract the muscle with high force and high velocity, may be a contributing factor to falls in the elderly.[143,144] Resistance exercise training in the elderly appears to have positive effects on aging muscles, causing an increase in the size of muscle fibers and an increase in strength and functional performance.[145–150] However, the response to resistance training is more limited in the elderly than in the young.[151]

Summary

- There are many factors that affect the function of the muscles. From the individual proteins and whole muscle architecture that determine the structure of the muscle to the neural and biomechanical relationships that determine performance of the muscle, the interrelationships between structure and function in muscles are complex and often indistinguishable.

- Muscles are more adaptable and, in many ways, more complex than the joints that they serve. Artificial joints have been designed and used to replace human joints, but it is as yet impossible to design a structure that can be used to replace a human muscle.

- All skeletal muscles adhere to the general principles of structure and function that have been presented in this chapter. Muscles produce the forces that power an incredible array of movements. During human movements, muscles not only provide the force to move the limbs but also provide the force for stabilization.

In subsequent chapters, the structure and function of specific muscles and the relationship between muscles and specific joints will be explored. The way in which muscles support the body in the erect standing posture and provide movement during walking will be examined in the last two chapters of this book.

Study Questions

1. What are the contractile and non-contractile elements of muscle?

2. How does the relationship between the thick and thin filaments change during isometric, concentric, and eccentric muscle contractions?

3. What is the function of titin in active muscle contraction and in passive muscle lengthening?

4. How is motor unit size related to the production of muscle force?

5. What is the fiber type distribution in the soleus muscle and how does this affect the functional characteristics of the soleus compared with the gastrocnemius muscle?

6. How is the function of the quadriceps similar to or different from the hamstrings muscles on the basis of the architectural characteristics of each muscle group?

7. What are the factors that could affect the development of active muscle tension? Suggest positions of the upper extremity that would limit tension development for each of the following muscles: biceps brachii, triceps brachii, and flexor digitorum profundus. What are the positions of the upper extremity in which the same muscles could passively limit motion?

8. Which upper extremity muscles are involved in lowering oneself into an armchair using one's arms? Is the muscle contraction eccentric or concentric? Explain your answer.

9. When testing strength using manual muscle testing, what exactly are we measuring? Beside changes in muscle length, why does muscle strength change at different points in the ROM?

10. Diagram the changes in the moment arm of the biceps brachii muscle from full

elbow extension to full flexion. How does the change in moment arm affect the function of the muscle?

11. What are the agonists, antagonists, and synergists in each of the following motions: abduction at the shoulder, flexion at the shoulder, and abduction at the hip?

12. Based on muscle architecture, what is the difference in function between the finger flexors and the finger extensors?

13. How does isokinetic exercise differ from other types of exercise, such as dynamic (free) weight lifting and isometric exercise?

14. What are the effects of immobilization when the muscle is maintained in a shortened position vs. a lengthened position?

15. What adaptations occur in skeletal muscle with aging?

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Chapter 4: The Vertebral Column

Diane Dalton

Anatomy Overview

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MUSCLE ACTIONS OF THE VERTEBRAL COLUMN (INTERVERTEBRAL AND CRANIOVERTEBRAL JOINTS)		
TABLE KEY:	Primary movers	Secondary movers
Sagittal Plane	Flexion	Extension
	Rectus abdominis External oblique Internal oblique Sternocleidomastoid Longus capitis Longus colli Anterior scalene Rectus capitis anterior	Splenius cervicis/capitis Iliocostalis group Longissimus group Semispinalis group Multifidus Rectus capitis posterior Major/ Minor Oblique capitis superior/ inferior Spinalis Rotatores Interspinalis

MUSCLE ACTIONS OF THE VERTEBRAL COLUMN (INTERVERTEBRAL AND CRANIOVERTEBRAL JOINTS)		
TABLE KEY:	Primary movers	Secondary movers
Sagittal Plane	Flexion	Extension
		Intertransversarii Levator scapulae Trapezius Quadratus lumborum
Transverse Plane	Ipsilateral Rotation	Contralateral Rotation
	Splenius Cervicis/Capitis Internal oblique Longissimus group Iliocostalis group Levator scapulae	External oblique sternocleidomastoid Trapezius Semispinalis capitis/cervicis Rotatores Multifidus
Frontal Plane	Lateral Flexion	
	Iliocostalis group Internal oblique External oblique Semispinalis group	

MUSCLE ACTIONS OF THE VERTEBRAL COLUMN (INTERVERTEBRAL AND CRANIOVERTEBRAL JOINTS)		
TABLE KEY:	Primary movers	Secondary movers
Sagittal Plane	Flexion	Extension
	Longissimus group Trapezius Multifidi Intertransversarii Quadratus lumborum Splenius capitis/ cervicis Scalenes Levator scapulae Sternocleidomastoid	

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VERTEBRAL COLUMN MUSCLE ATTACHMENTS		
Muscles	Proximal Attachment	Distal Attachment
Splenius capitis	Nuchal ligament and spinous	Fibers run superolaterally to

VERTEBRAL COLUMN MUSCLE ATTACHMENTS		
Muscles	**Proximal Attachment**	**Distal Attachment**
	processes of C7–T6 vertebrae	mastoid process of temporal bone and lateral third of superior nuchal line of occipital bone
Splenius cervicis	Nuchal ligament and spinous processes of C7–T6 vertebrae	Transverse processes of C1–C3 vertebrae
Iliocostalis (erector spinae)	Arises by a broad tendon from posterior part of iliac crest, posterior surface of sacrum, sacroiliac ligaments, sacral and inferior lumbar spinous processes, and supraspinous ligament	Lumborum, thoracis, cervicis; fibers run superiorly to angles of lower ribs and cervical transverse processes
Longissimus (erector spinae)		Thoracis, cervicis, capitis; fibers run superiorly to ribs between tubercles and angles, to transverse processes in thoracic and cervical regions, and to mastoid processes of temporal bone
Spinalis (erector spinae)		Thoracis, cervicis, capitis; fibers run superiorly to spinous processes in the upper thoracic region and to cranium
Semispinalis (transversospinalis)	Transverse processes of C4–T12 vertebrae	Thoracis, cervicis, capitis; fibers run superomedially to occipital bone and spinous processes in thoracic and

VERTEBRAL COLUMN MUSCLE ATTACHMENTS		
Muscles	**Proximal Attachment**	**Distal Attachment**
		cervical regions, spanning 4–6 segments
Multifidus (transversospinalis)	Arises from posterior sacrum, PSIS of ilium, aponeurosis of erector spinae, sacroiliac ligaments, mammillary processes of lumbar vertebrae, transverse processes of T1–T3, articular processes of C4–C7	Thickest in lumbar region; fibers pass obliquely superomedially to entire length of spinous processes, spanning 2–4 segments
Rotatores: brevis and longus (transversospinalis)	Arise from transverse processes of vertebrae; best developed in thoracic region	Fibers pass superomedially to attach to spinous process of vertebra immediately (brevis) or 2 segments (longus) superior
Interspinalis	Superior surfaces of spinous processes of cervical and lumbar vertebrae	Inferior surface of spinous process of vertebra of proximal attachment
Intertransversarii	Transverse processes of cervical and lumbar vertebrae	Transverse processes of adjacent vertebrae
Quadratus lumborum	Medial half of inferior border of 12th ribs and lumbar transverse processes	Iliolumbar ligament and lip of iliac crest
Trapezius	Medial third of superior nuchal line, external occipital protuberance, nuchal ligament, spinous processes	Lateral third of clavicle, acromion, and spine of scapula

VERTEBRAL COLUMN MUSCLE ATTACHMENTS		
Muscles	**Proximal Attachment**	**Distal Attachment**
	of C7–T12 vertebrae	
Levator scapula	Transverse processes of C1–C4 vertebrae	Superior aspect of medial border of the scapula
Anterior scalene	Transverse processes of C3–C6 vertebrae	1st rib
Middle scalene	Transverse processes of C5–C7 vertebrae	Superior surface of 1st rib
Posterior scalene	Transverse processes of C5–C7 vertebrae	External border of 2nd rib
Sternocleidomastoid	Lateral surface of mastoid process of temporal bone and lateral half of superior nuchal line	Sternal head: anterior surface of manubrium of sternum Clavicular head: superior surface of medial third of clavicle
Rectus capitis posterior major (suboccipital muscles)	Spinous process of C2 vertebra	Lateral aspect of inferior nuchal line
Rectus capitis posterior minor (suboccipital muscles)	Posterior tubercle of posterior arch of C1 vertebra	Medial aspect of inferior nuchal line
Obliquus capitis inferior (suboccipital muscles)	Posterior tubercle of posterior arch of C2 vertebra	Transverse process of C1 vertebra
Obliquus capitis	Transverse process of C1	Occipital bone between

VERTEBRAL COLUMN MUSCLE ATTACHMENTS		
Muscles	**Proximal Attachment**	**Distal Attachment**
superior (suboccipital muscles)	vertebra	superior and inferior nuchal lines
Rectus capitis anterior	Base of cranium, just anterior to occipital condyle	Anterior surface of lateral mass of C1 vertebra
Rectus capitis lateralis	Jugular process of occipital bone	Transverse process of C1 vertebra
Longus colli	Bodies of C1–C3 and transverse processes of C3–C6 vertebrae	Bodies of C5–T3 vertebrae; transverse processes of C3–C5 vertebrae
Longus capitis	Basilar part of occipital bone	Transverse processes of C3–C6
Rectus abdominis	Pubic symphysis and pubic crest	Xyphoid process and 5th–7th costal cartilage
Internal oblique	Thoracolumbar fascia, anterior 2/3 of iliac crest	Inferior borders of 10th–12th ribs, linea alba
External oblique	External surfaces of 5th–12th ribs	Linea alba, pubic tubercle, and anterior half of iliac crest

Introduction

The vertebral column is an amazingly complex structure that must meet the seemingly contradictory demands of mobility and stability for the trunk and extremities while protecting the spinal cord. Although the pelvis is not always considered part of the

vertebral column, it will be included in this chapter because of the anatomical and functional interrelationships between the lumbar spine, sacrum, and innominate bones.

Structure and Function of the Vertebral Column

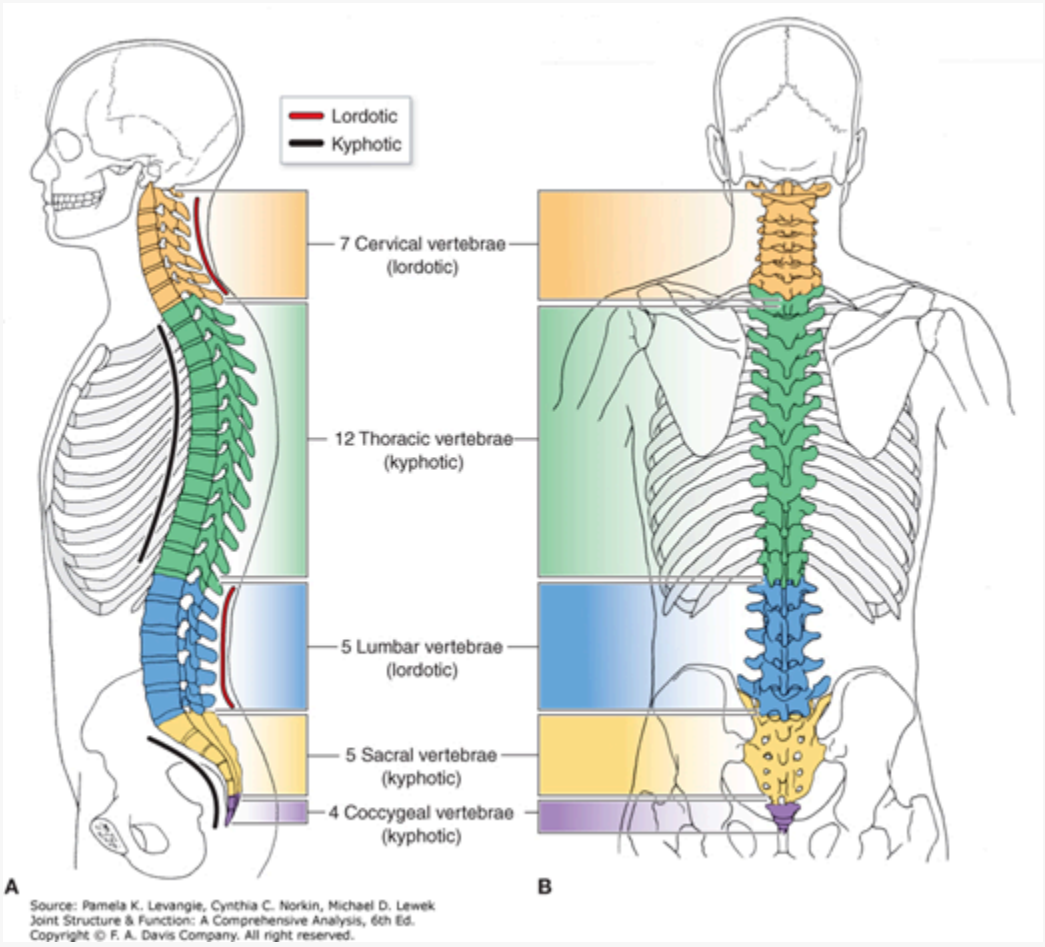
Structure

The vertebral column resembles a curved rod, composed of 33 vertebrae and 23 intervertebral discs. The vertebral column is divided into five regions: cervical, thoracic, lumbar, sacral, and coccygeal (Fig. 4–1). The vertebrae adhere to a common basic structural design but show regional variations in size and configuration that reflect the functional demands of a particular region. The vertebrae increase in size from the cervical to the lumbar regions and then decrease in size from the sacral to coccygeal regions. Twenty-four of the vertebrae in adults are individual bones: seven vertebrae are located in the cervical region, twelve in the thoracic region, and five in the lumbar region. Five of the remaining nine vertebrae are fused to form the sacrum, and the remaining four constitute the coccygeal vertebrae. When viewed from the side, four curves are evident (Fig. 4–1A). Curves that have a posterior convexity (anterior concavity) are referred to as **kyphotic curves;** curves that have an anterior convexity (posterior concavity) are called **lordotic curves.** The vertebral column bisects the trunk when viewed from the posterior aspect (Fig. 4–1B).

FIGURE 4-1

The five regions of the vertebral column include cervical, thoracic,

lumbar, sacral, and coccygeal. **A.** In a side view, two lordotic curves (cervical and lumbar) and two kyphotic curves (thoracic and sacral) are evident. **B.** In a posterior view, the vertebral column bisects the trunk.

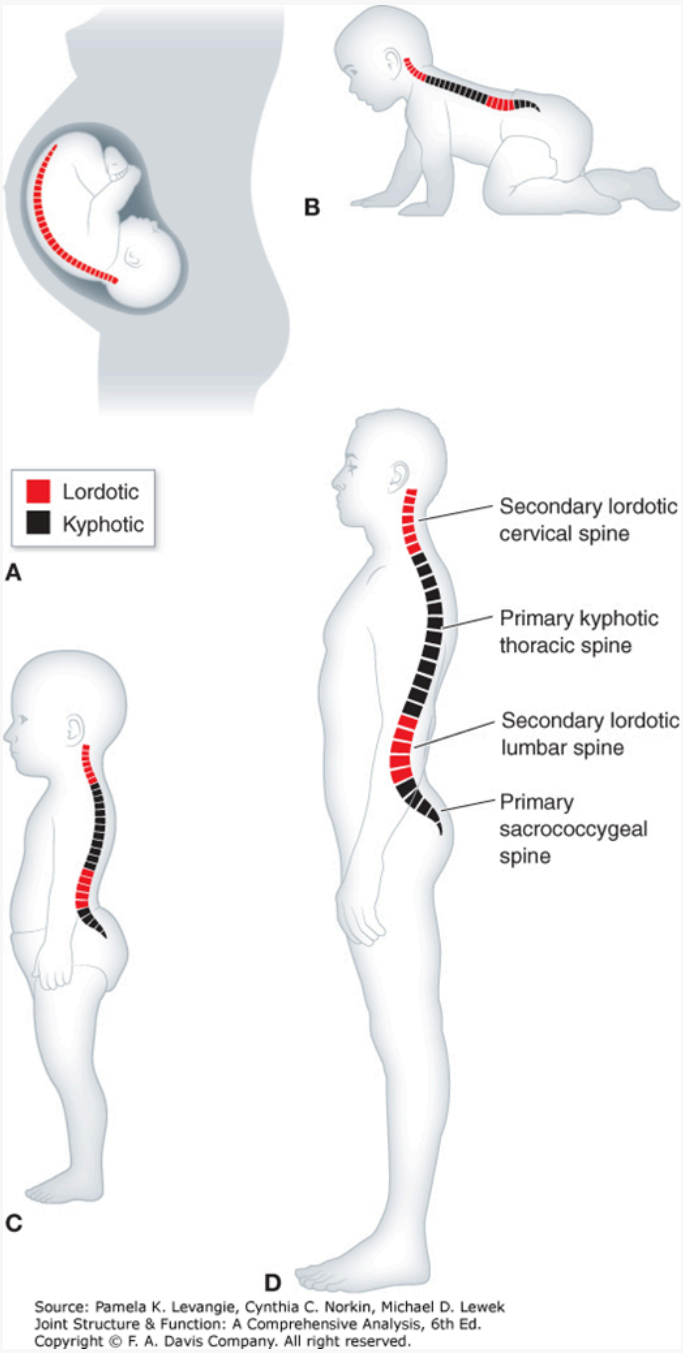


The four curves of the vertebral column develop over time (Fig. 4–2). The curve of the vertebral column of a fetus exhibits one long curve that is convex posteriorly (Fig. 4–2A). As the child achieves developmental milestones such as crawling (Fig. 4–2B) and walking (Fig. 4–2C), the curves of the vertebral column adapt to the demands of upright posture. Four distinct and relatively balanced anteroposterior curves are eventually

evident (Fig. 4–2D). The two thoracic and sacrococcygeal curves that retain the original posterior convexity (kypohosis) throughout life are called **primary curves,** whereas the two cervical and lumbar curves that show a reversal of the original posterior convexity (lordosis) are called **secondary curves.** The anteroposterior curves of the vertebral column provide a significant advantage over a straight rod because reciprocal curves are able to resist much higher compressive loads.[1]

FIGURE 4-2

Primary and secondary curves. **A.** The entire spine is kyphotic in the fetus in utero. **B.** As the baby begins to orient his or her head to vertical, the secondary cervical lordosis appears first. **C.** In the toddler all four curves are evident, often with an exaggeration of the secondary lumbar lordosis. **D.** In the typical adult spine, the primary thoracic and sacrococcygeal kyphoses are evident, as are the secondary cervical and lumbar lordoses.



When standing, the vertebral column typically functions as a closed chain, with the upright head at one end and the weightbearing feet at the other. We can readily see how the contact of the feet to the ground “fixes” one end of the chain. The need for the head at the other end of the chain to remain vertically oriented in a somewhat stable

position is less obviously “fixed.” As we move, we must allow the sensory organs, particularly the eyes and ears, to be optimally positioned for function. Each of the many separate but interdependent components of the vertebral column is designed to contribute to the overall function of the total unit, as well as to perform specific tasks.

The first section of this chapter will cover the general components of a mobile segment, followed by regional variations and the sacroiliac joints. The second section of the chapter will cover the function of the muscles of the vertebral column and pelvis.

The Mobile Segment

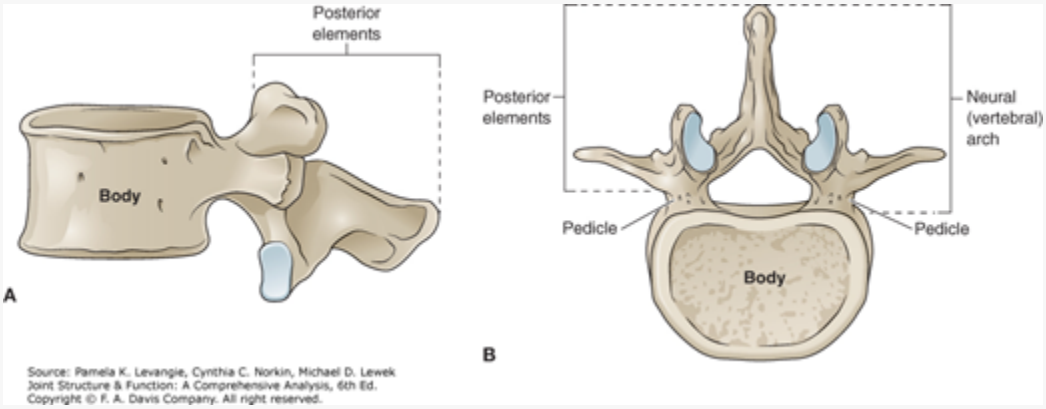
It is generally held that the smallest functional unit in the spine is the **mobile segment.** A mobile segment consists of any two adjacent vertebrae, the intervening intervertebral disc (if there is one), and all the soft tissues that secure them together. Vertebral movements are named by what the cranial vertebra is doing and by what the anterior aspect of the vertebral body is doing (not what the spinous process is doing).

A Typical Vertebra

The structure of a typical vertebra consists of two major parts (Fig. 4–3): an anterior, cylindrically shaped vertebral body (Fig. 4–3A) and a posterior, irregularly shaped neural arch (Fig. 4–3B). The vertebral body is designed to be the weightbearing structure of the spinal column. It is suitably designed for this task, given its block-like shape with generally flat superior and inferior surfaces. To minimize the weight of the vertebrae and allow dynamic load-bearing, the vertebral body is not a solid block of bone but a shell of cortical bone surrounding a cancellous-filled cavity (Fig. 4–4).[2] The cortical shell is reinforced by trabeculae in the cancellous bone that help the cortical bone resist compressive forces.

FIGURE 4-3

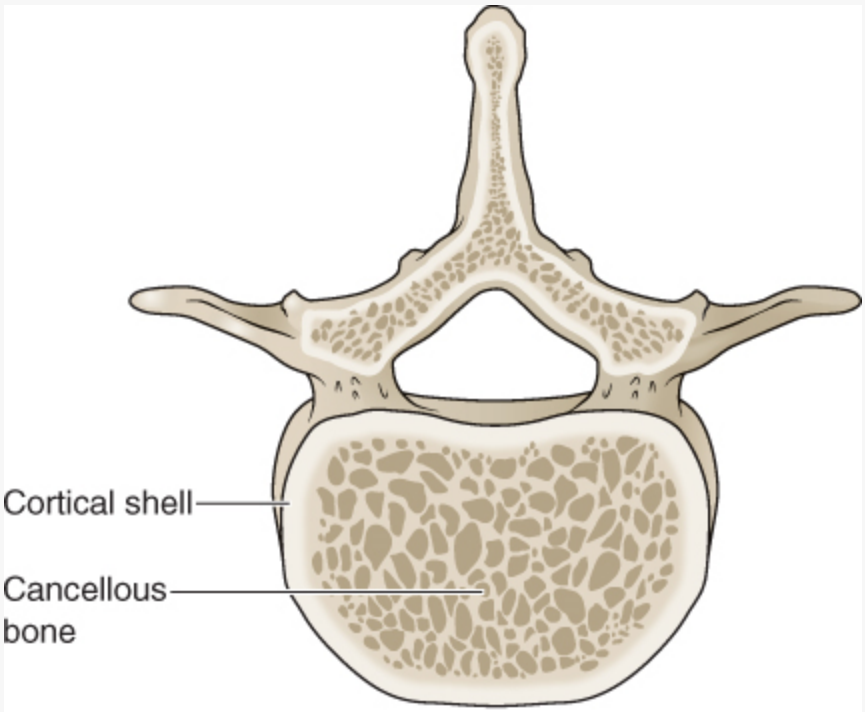
A. The anterior portion of a vertebra is composed of the vertebral body. **B.** The posterior portion of a vertebra is called the vertebral or neural arch. The neural arch is attached to the body via the pedicles.



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FIGURE 4-4

The vertebral body is comprised of a shell of cortical bone surrounding cancellous with reinforcing trabeculae bone.

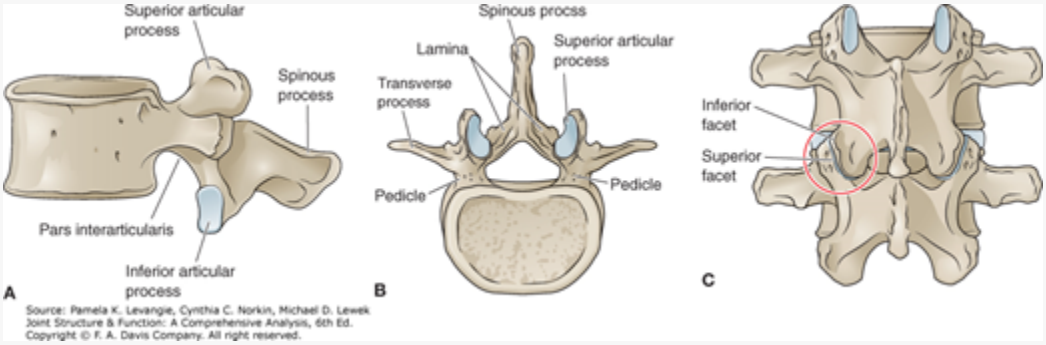


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The **neural arch** can be divided into the pedicles and the posterior elements (Fig. 4–5). The **pedicles** (Fig. 4–5B) are the portion of the neural arch that lies between the articular processes and the vertebral body, connecting the posterior elements to the body. The pedicles are short, stout pillars with thick walls and are well structured to transmit tension and bending forces from the posterior elements to the vertebral body. In general, the pedicles increase in size from the cervical to lumbar regions, which makes sense because greater forces are transmitted through the pedicles in the lumbar region. The orientation of the pedicle helps to define the size and shape of the vertebral canal through which the spinal cord and cauda equina run.

FIGURE 4-5

A and **B.** The posterior elements of a vertebra are the laminae, the articular processes, the spinous process, and the transverse processes. The pars interarticularis is the portion of each lamina that lies between the articular processes. **C.** The facets of the inferior articular processes on the vertebra above articulate with the facets of the superior articular processes on the vertebra below.



The remaining posterior elements are the laminae, the articular processes, the spinous process, and the transverse processes (Fig. 4–5B). The **laminae** serve as origination points for the rest of the posterior elements. The laminae are thin, vertically oriented pieces of bone that serve as the “roof” to the neural arch that protects the spinal cord. In addition, the laminae transmit forces from the posterior elements to the pedicles anteriorly and, via the pedicles, on to the vertebral body. This force transfer occurs through a region of the laminae called the **pars interarticularis; which,** as its name suggests, is the portion of the laminae that is between the superior and inferior articular processes (Fig. 4–5A).

The **articular processes** for each vertebra include two **superior** and two **inferior facets**

(Fig. 4–5A); the superior facets articulate with the inferior facets of the vertebra above while the inferior facets articulate with the superior facets of the vertebra below (Fig. 4–5C). In the sagittal plane, these articular processes form a supportive column, frequently referred to as the **articular pillar.** The pars interarticularis between the articular facets is subjected to bending forces that are transmitted from the vertically oriented lamina to the more horizontally oriented pedicles. The pars interarticularis is most developed in the lumbar spine, where the forces are the greatest in magnitude. Typically an increase in cortical bone occurs to accommodate the increased forces in this region. If the cortical bone is insufficient, the pars interarticularis is susceptible to stress fractures.



Foundational Concepts 4–1

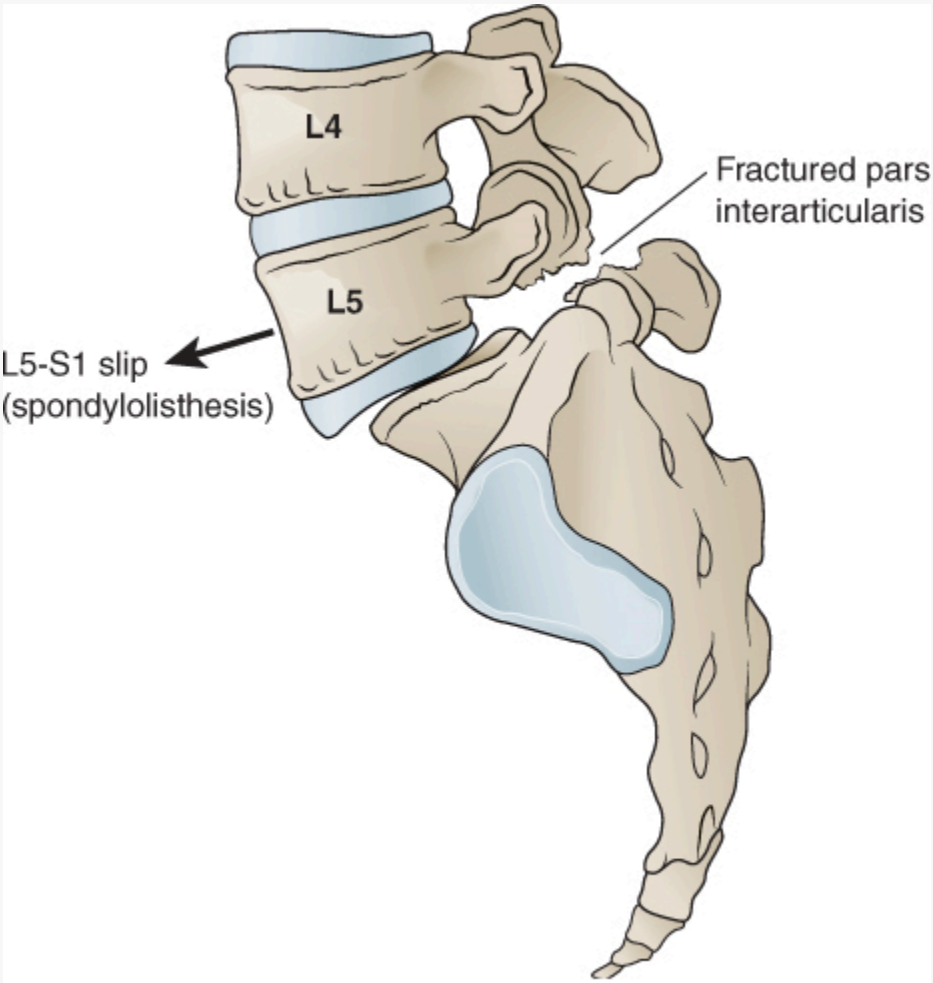
Spondylolytic Spondylolisthesis

When fractures of the pars interarticularis occur bilaterally, the result may be **spondylolytic spondylolisthesis.** In this condition, the posterior elements are separated from the remainder of the neural arch and the vertebral body. The vertebral body may then begin to slip forward on the vertebra below (Fig. 4–6). This occurs most frequently at the L5–S1 mobile segment because of the angulation of the segment and the anterior shear forces that exist there.

The altered location of the slipped vertebra changes its relationship to adjacent structures and creates excessive stress on supporting ligaments and joints. Overstretched ligaments may lead to a lack of stability of the mobile segment. The forward slippage of the vertebra may cause compression on the nerves of the cauda equina. Pain may arise from excessive stress on any of the following pain-sensitive structures: anterior and posterior longitudinal ligaments, interspinous ligament, spinal nerves, dura mater, vertebral bodies, zygapophyseal joint capsules, synovial linings, or the muscles.

FIGURE 4-6

In this lateral view, a spondylolisthesis is evident, resulting from failure of the pars interarticularis. That failure allows the vertebra above to slide anteriorly on the vertebra below, most commonly occurring as a slip of L5 on S1.



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In the typical vertebra, the **spinous process** and two **transverse processes** are sites for muscle attachments and serve to increase the lever arm for the muscles of the vertebral column. Table 4–1 summarizes the components of a typical vertebra and Table 4–2

summarizes the regional variations in vertebral structure.

Table 4-1

Components of a Typical Vertebra

Table 4-2

Regional Variations in Vertebral Structure

The vertebrae are subjected to a wide variety of forces; however, they have a bony architecture that suggests a typical loading pattern.[3] Vertebral bone trabecular systems that develop in response to the stresses placed on the vertebral bodies and the neural arch are found within the spongy (cancellous) bone (Fig. 4–7). The vertebrae have vertically oriented trabeculae with horizontal connections near the vertebral endplates and with denser bone areas near the pedicle bases.[3,4] Vertical systems within the vertebral bodies help support the body's weight and resist compression forces. There are also fan-shaped trabeculae introduced into the vertebral body at the area of the pedicle in response to bending and shearing forces transmitted through this region.[3]

FIGURE 4-7

Schematic representation of the internal architecture of a lumbar vertebra (lateral view). The various trabeculae are arranged along the lines of force transmission.



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The Intervertebral Disc

The intervertebral disc has two principal functions: to separate two vertebral bodies, thereby increasing available motion, and to transmit load from one vertebral body to the next. The size of the intervertebral disc is related to both the amount of motion and the magnitude of the loads that must be transmitted. The intervertebral discs increase in size from the cervical to the lumbar regions. The disc thickness varies from approximately 3 mm in the cervical region where the weightbearing loads are the lowest, to about 9 mm in the lumbar region where the weightbearing loads are the highest.[1] Although the discs are smallest in the cervical region and largest in the lumbar region, it is the ratio between disc thickness and vertebral body height that determines the available motion. The greater the ratio, the greater the intersegmental mobility. The ratio is greatest in the cervical region, followed by the lumbar region, and the ratio is

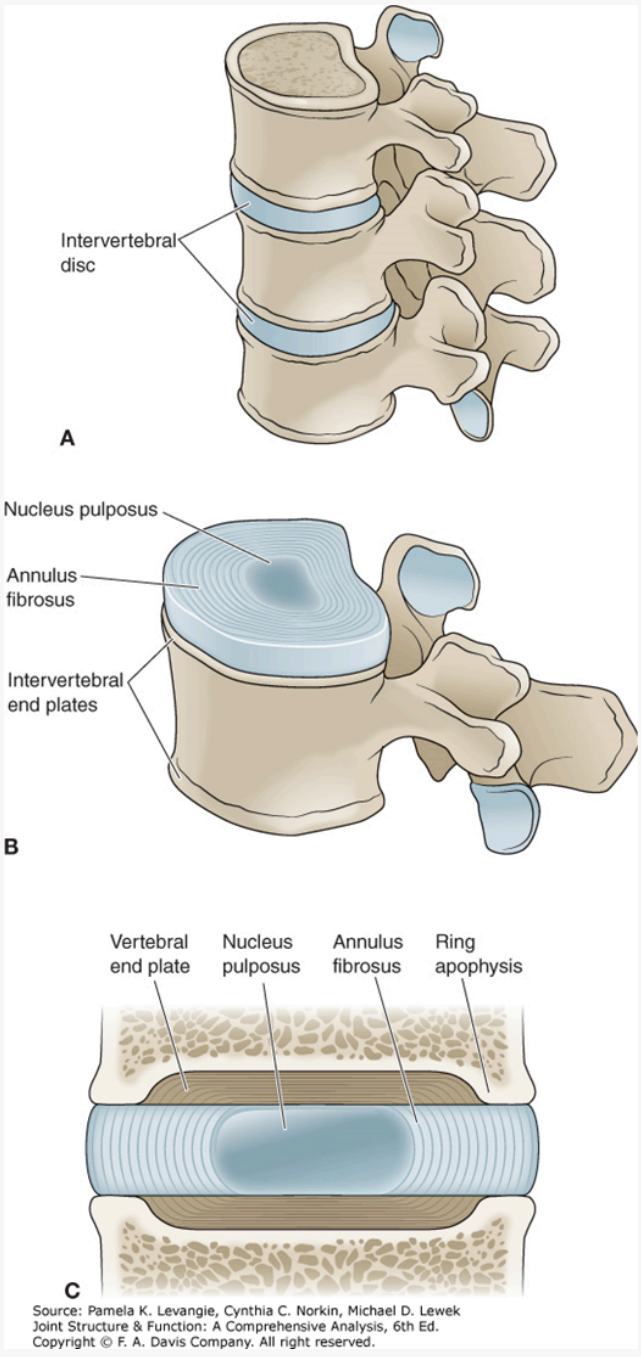
smallest in the thoracic region. This reflects the greater functional needs for mobility in the cervical and lumbar regions and for relative stability in the thoracic region.

Most of what we know about the structure and function of the intervertebral discs has been gleaned from studies of the lumbar region. It was long thought that the discs of the cervical and thoracic regions had a structure similar to discs of the lumbar region, although this may not be the case.[5,6] This section will describe the general structure and function of the intervertebral disc. The region-specific variations will be described in the regional structure sections of the chapter.

All intervertebral discs are composed of three parts: (1) the nucleus pulposus, (2) the annulus fibrosus, and (3) the vertebral endplate (Fig. 4–8). The **nucleus pulposus** is the gelatinous mass found in the center, the **annulus fibrosus** is the fibrous outer ring, and the **vertebral endplate** is the cartilaginous layer covering the superior and inferior surfaces of the disc, separating it from the cancellous bone of the vertebral bodies above and below. All three structures are composed of water, collagen, and proteoglycans; however, the relative proportions of each vary. Fluid and proteoglycan concentrations are highest in the nucleus and lowest in the outer annulus fibrosus and the outer vertebral endplate (closest to the vertebral body). Conversely, collagen concentrations are highest in the vertebral endplate and outer annulus and lowest in the nucleus pulposus. Although the nucleus pulposus is clearly distinct from the annulus fibrosus in its center and the annulus fibrosus is clearly distinct from the nucleus in its outer rings, there is no clear boundary separating the two structures where they merge. They are distinct structures only where they are furthest apart.

FIGURE 4-8

A. The intervertebral discs lie between each of the vertebrae. **B.** This schematic representation of a lumbar intervertebral disc shows the nucleus pulposus, the annulus fibrosus, and the vertebral endplate. **C.** On cross section, the relationship between the three components of the disc is evident, with the endplate located within the circumferential ring apophysis.



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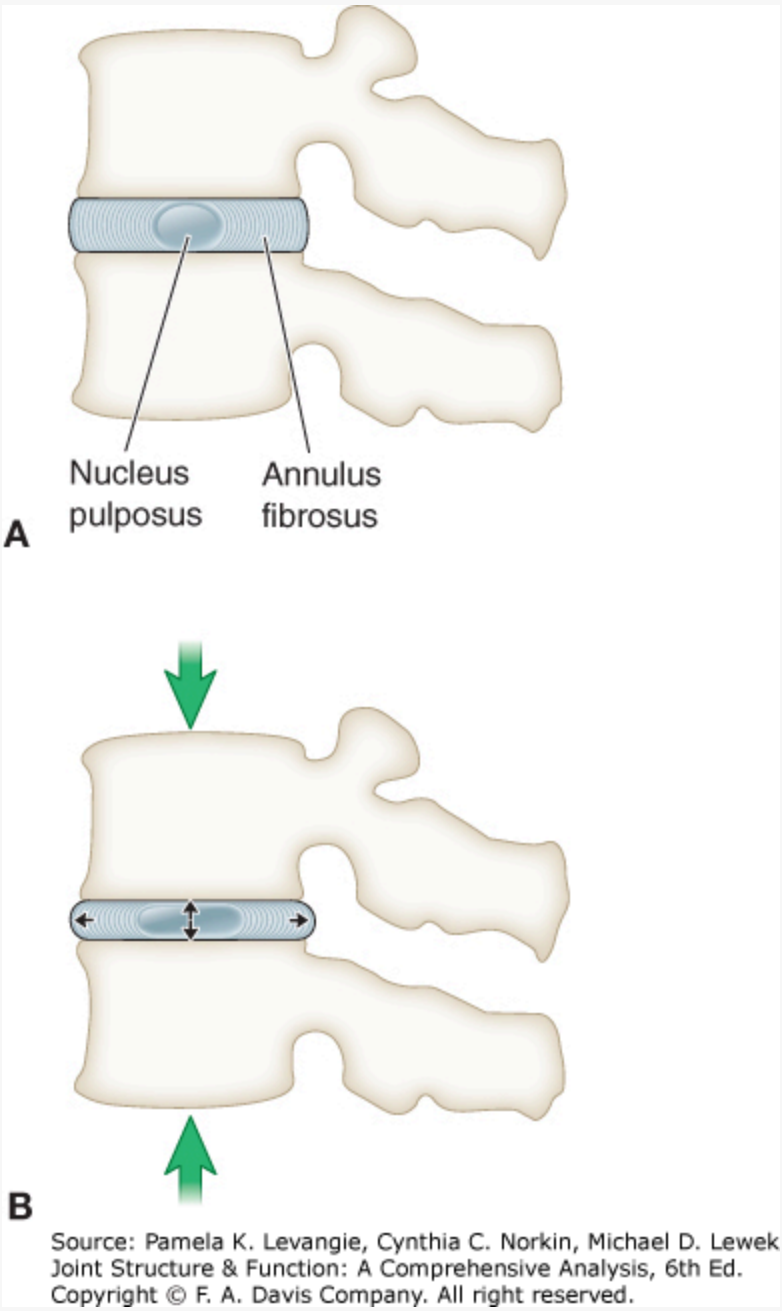
NUCLEUS PULPOSUS

The nucleus pulposus is approximately 80% water, depending on age and time of day.[2] Proteoglycans make up approximately 65% of the dry weight and have the ability to attract water molecules because of the presence of glycosaminoglycans; hence the high

water content. Collagen fibers contribute 15% of the dry weight, and the remainder of the dry weight is composed of collagen, chondrocytes, elastin fibers, proteins, and proteolytic enzymes.[2] The nucleus pulposus has both type I and type II collagen; however, type II predominates because of its ability to resist compressive loads. Very little, if any, type I collagen is present in the center of the nucleus pulposus.[2,7] The nucleus pulposus frequently has been likened to a water balloon. When compressed, the nucleus deforms, and the increased pressure stretches the walls of the nucleus pulposus in all directions (Fig. 4–9).

FIGURE 4-9

Schematic representation of compression of an intervertebral disc. Behaving somewhat like a water-filled balloon, the nucleus pulposus is contained by the annulus. **A.** In the unloaded spine, the nucleus remains rounded. **B.** Under compressive loading, the nucleus pulposus attempts to expand, placing the annulus fibrosus under tension that restrains further radial expansion of the nucleus. The nuclear pressure is transmitted by the annulus fibrosus to the vertebral endplates.



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ANNULUS FIBROSUS

The annulus fibrosus is approximately 60% water depending on age and time of day. Proteoglycans contribute approximately 20% and collagen fibers 50% of the dry weight.[2] The remainder of the dry weight is made up of elastin fibers and cells such as fibroblasts and chondrocytes.[2] Again, type I and type II collagen are present in its layers;

however, type I collagen predominates, particularly in the outer portions.[2,7] This collagen makeup reflects the need for the annulus fibrosus to resist greater proportions of tensile forces. The annulus fibers are attached by Sharpey fibers to the cartilaginous endplates on the inferior and superior vertebral plateaus of adjacent vertebrae and to the epiphyseal ring (ring apophysis) region.

VERTEBRAL ENDPLATES

The vertebral endplates are layers of cartilage that cover the region of the vertebral bodies encircled by the ring apophysis on both the superior and inferior surfaces (see Fig. 4–8). The endplates cover the nucleus pulposus but not the entire annulus fibrosus. The vertebral endplate is strongly attached to the annulus fibrosus but only weakly attached to the vertebral body, which is why it is considered a component of the disc.[2,7] Vertebral endplates consist of proteoglycans, collagen, and water, as does the rest of the disc. In addition, there are cartilage cells aligned along the collagen. The cartilage of the vertebral endplates is both hyaline cartilage and fibrocartilage. Hyaline cartilage is present closest to the vertebral body and is found mainly in young discs. Fibrocartilage is present closest to the nucleus pulposus. With increasing age, fibrocartilage becomes the major component of the vertebral endplate with little or no hyaline cartilage remaining. This presumably reflects the increasing need to tolerate higher compressive forces.

INNERVATION AND NUTRITION

The intervertebral discs are innervated in the outer one-third to one-half of the fibers of the annulus fibrosus by branches from the vertebral and sinuvertebral nerves. The sinuvertebral nerve also innervates the peridiscal connective tissue and ligaments associated with the vertebral column.[2,8]

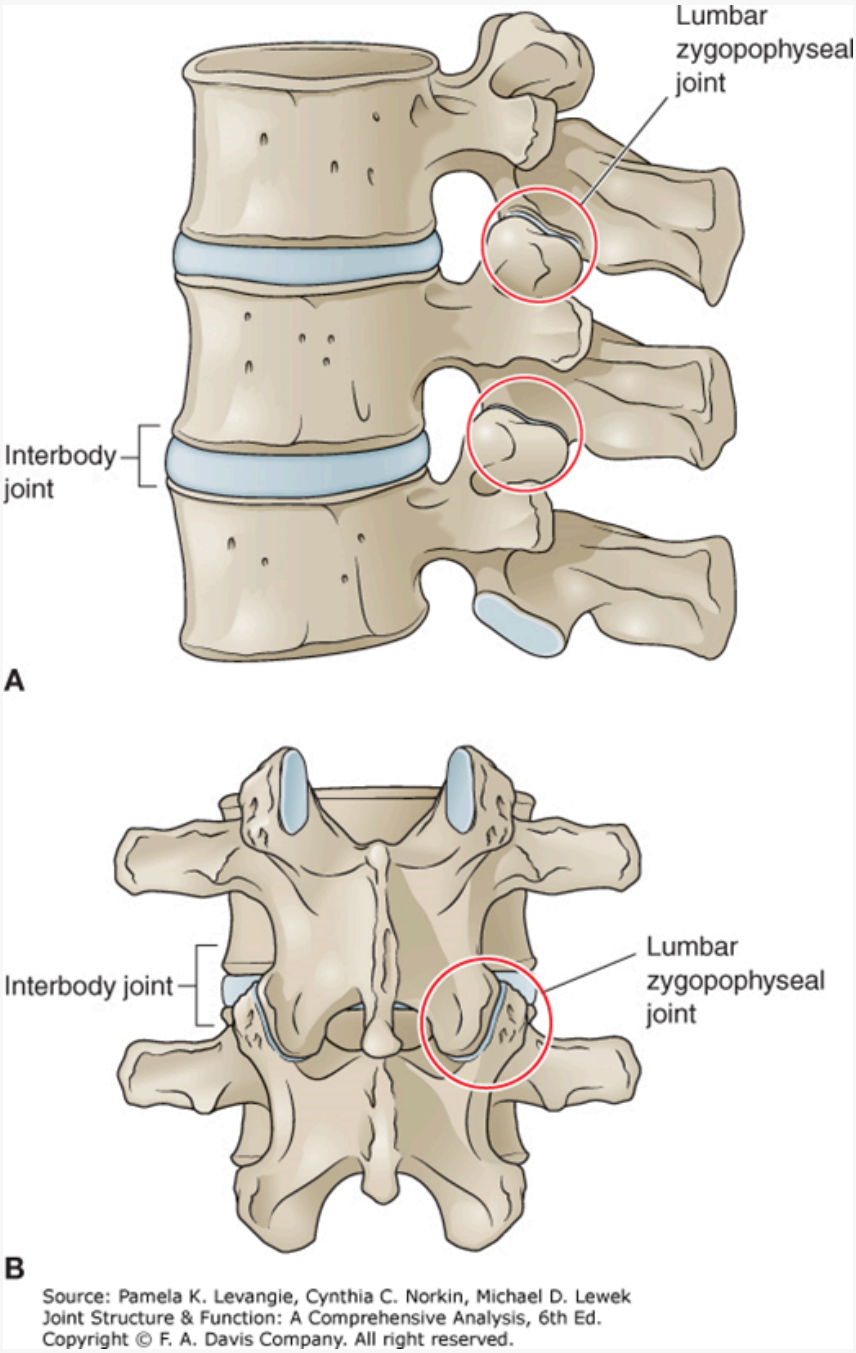
The intervertebral discs do not receive blood supply from any major arterial branches. The metaphyseal arteries form a dense capillary plexus in the base of the endplate cartilage and the subchondral bone deep to the endplate; small branches from these metaphyseal arteries supply the outer surface of the annulus fibrosus.[2] The remainder of the disc receives its nutrition via diffusion.

Articulations

Two main types of articulations are found in the vertebral column: cartilaginous joints of the symphysis type between the vertebral bodies, including the interposed discs, and synovial joints between the zygapophyseal facets located on the superior articular processes of one vertebra and the zygapophyseal facets on the inferior articular processes of an adjacent vertebra above. The joints between the vertebral bodies are referred to as the **interbody joints** and the joints between the zygapophyseal facets are called the **zygapophyseal** (**apophyseal** or **facet**) **joints** (Fig. 4–10). Synovial joints also are present where vertebrae articulate with the skull and with the pelvis at the sacroiliac joints, as well as where the ribs articulate with the thoracic vertebrae (see Chapter 5).

FIGURE 4-10

A. Lateral and **B.** Posterior views of interbody and zygapophyseal joints.



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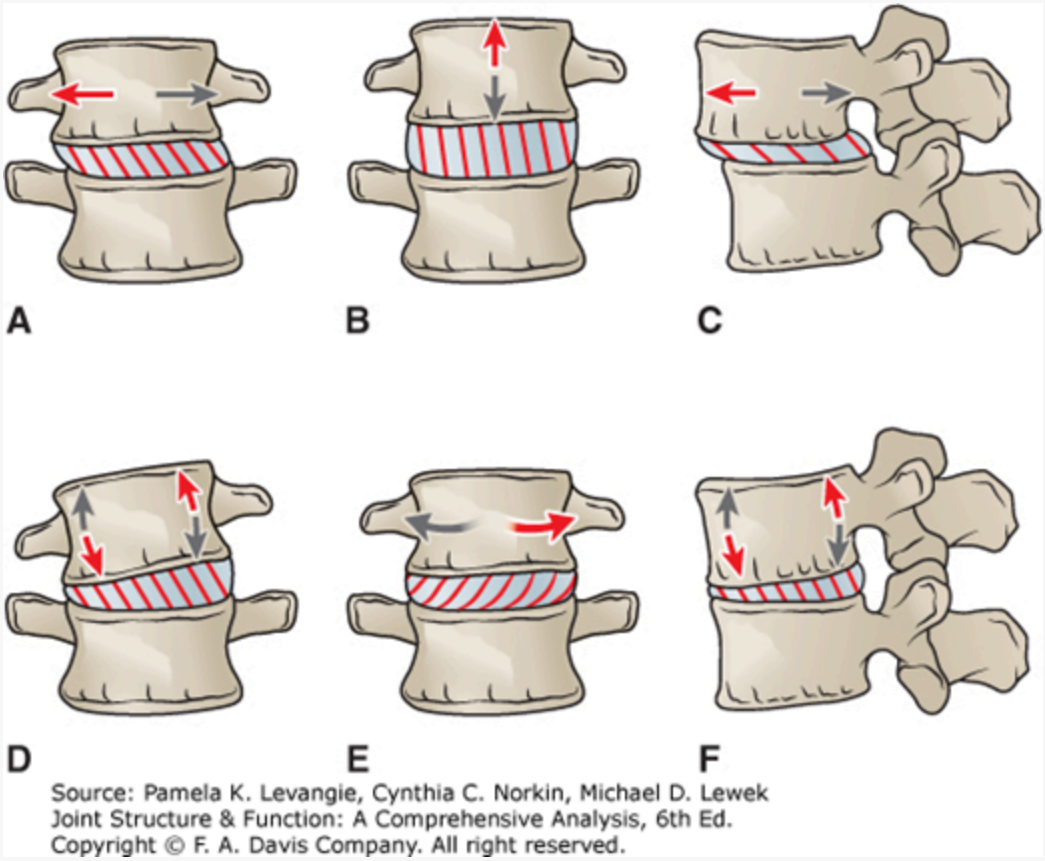
INTERBODY JOINTS

Available movements at the interbody joints include **sliding** (translation)**, distraction, compression,** and **tilting** (Fig. 4–11). Sliding motions can occur in the following directions: anterior to posterior, medial to lateral, and as axial rotation (torsion). Tilt

motions can occur in anterior to posterior and in lateral directions. These motions, together with distraction and compression, constitute six degrees of freedom.[9] The amounts of each of these motions vary by region according to structural differences in the discs and the vertebral bodies, as well as in the ligamentous supports. In addition, the zygapophyseal joints influence the total available motion of the interbody joints.

FIGURE 4-11

Available movements of the vertebra above on the vertebra below can take place as **A.** Side-to-side sliding (translation) in the frontal plane; **B.** Axial distraction and compression (superior-inferior) vertically; **C.** Anteroposterior sliding (translation) in the sagittal plane; **D.** Side-to-side tilting in the frontal plane around an anteroposterior axis; **E.** Axial rotation in the transverse plane around a vertical axis; or **F.** Anteroposterior tilting in the sagittal plane around a coronal axis.



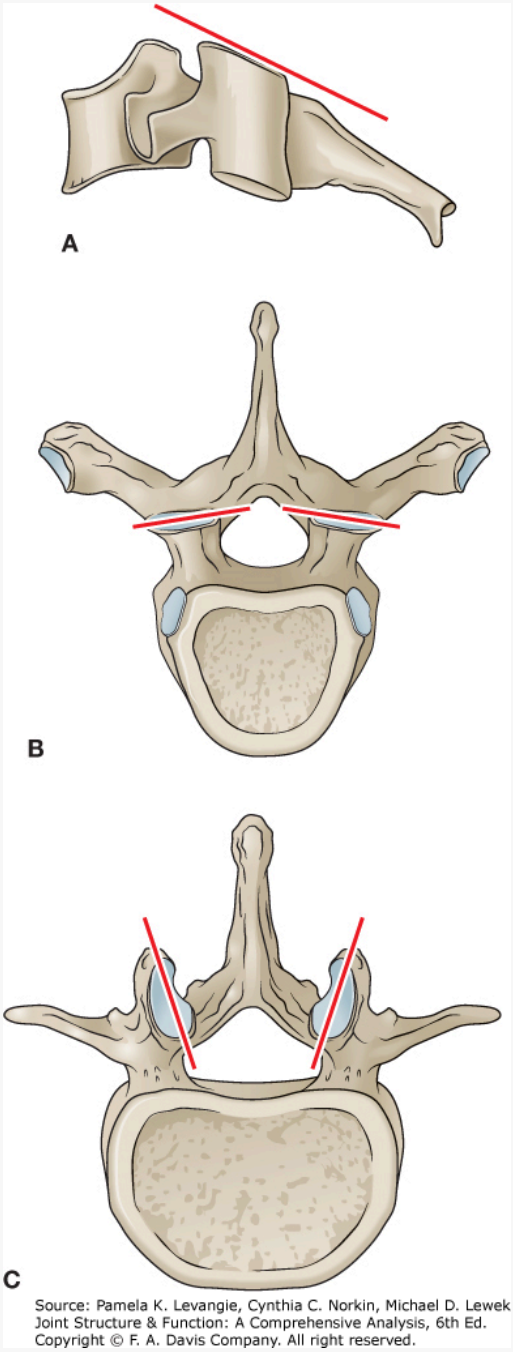
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ZYGAPOPHYSEAL ARTICULATIONS

The zygapophyseal joints are composed of the articulations between the right and left inferior articulating facets of a vertebra with, respectively, the right and left superior facets of the vertebra immediately below. The zygapophyseal joints are synovial joints and have regional variations in structure (Fig. 4–12). Intra-articular accessory joint structures have been identified in the zygapophyseal joints.[2,10,11] These accessory structures appear to be of several types, but most are classified as fibroadipose meniscoids. The structures most likely are involved in protecting articular surfaces that are exposed during flexion and extension of the vertebral column.

FIGURE 4-12

Orientation of the zygapophyseal joints varies regionally. **A.** A typical cervical spine orientation (lateral view); **B.** A typical thoracic spine (superior view); and **C.** A typical lumbar spine (superior view).



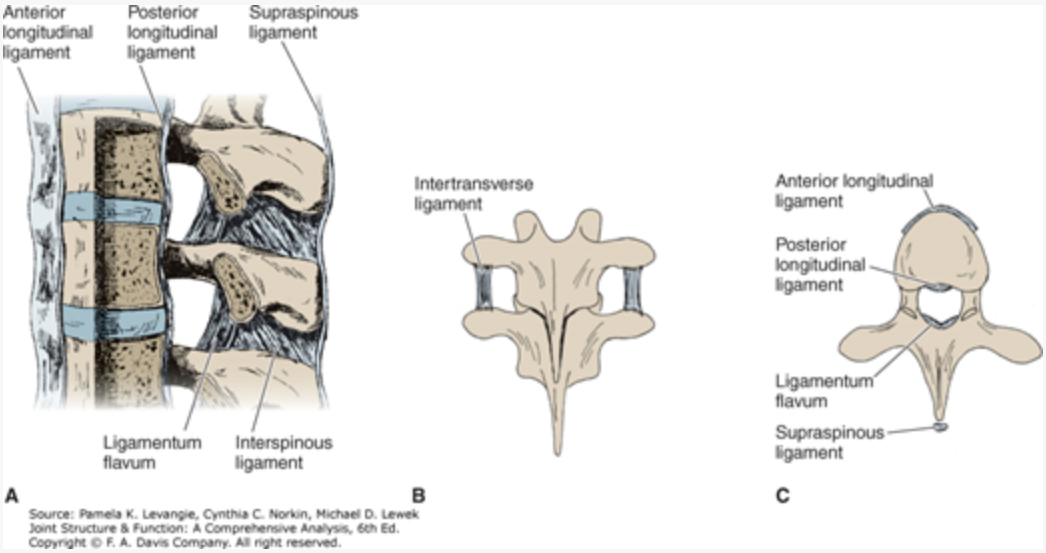
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Ligaments and Joint Capsules

The ligamentous system of the vertebral column is extensive and exhibits considerable regional variability. Six main ligaments are associated with the intervertebral and zygapophyseal joints. These are the anterior and posterior longitudinal ligaments, the ligamentum flavum, and the interspinous, supraspinous, and intertransverse ligaments (Fig. 4–13).

FIGURE 4-13

Ligamentous system of the vertebral column. **A.** The anterior and posterior longitudinal ligaments are located on the anterior and posterior aspects of the vertebral body, respectively. The ligament flavum runs from lamina to lamina on the posterior aspect of the vertebral canal. Portions of the bone have been removed to show the orientation of the ligament fibers. The supraspinous and interspinous ligaments run vertically along and between the spinous processes. **B.** The intertransverse ligament connects the transverse processes. **C.** The relative positions of the ligaments are shown in a superior view of the vertebra.



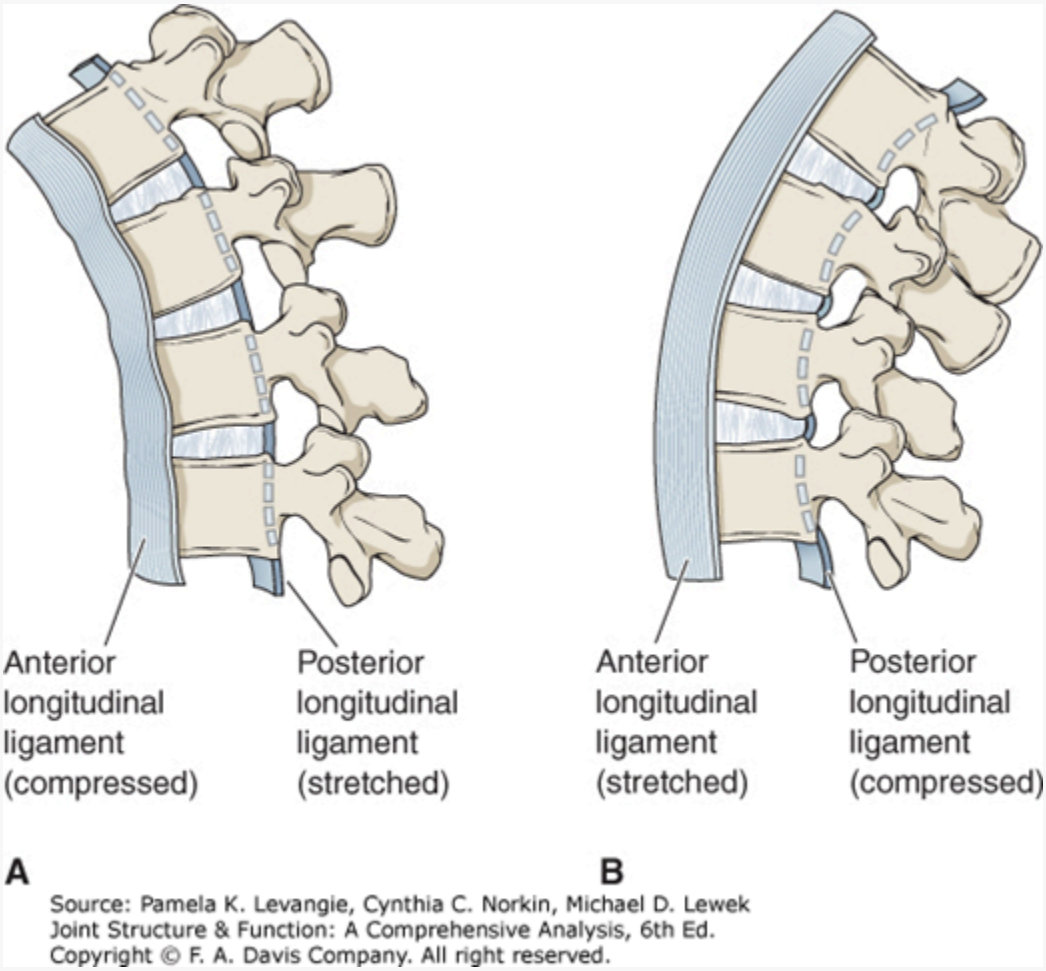
ANTERIOR AND POSTERIOR LONGITUDINAL LIGAMENTS

The anterior longitudinal ligament and posterior longitudinal ligament are associated with the interbody joints. The **anterior longitudinal ligament** (ALL) (Figs. 4–13A and C) runs along the anterior and lateral surfaces of the vertebral bodies from the sacrum to the second cervical vertebra. The anterior longitudinal ligament has two layers made up of thick bundles of collagen fibers; the fibers of the superficial layer are long and bridge several vertebrae, whereas the deep fibers are short and run between single pairs of vertebrae. The deep fibers blend with the fibers of the annulus fibrosus and reinforce the anterolateral portion of the intervertebral discs and the anterior interbody joints.[10] The anterior longitudinal ligament is well developed in the lordotic (cervical and lumbar) sections of the vertebral column but has little substance in the region of thoracic kyphosis. The tensile strength of the anterior longitudinal ligament is greatest in the lumbar region where it is reported to be twice as strong as the posterior longitudinal ligament (Fig. 4–14). The anterior longitudinal ligament is compressed in flexion (Fig. 4–14A) and stretched in extension (Fig. 4–14B). It may become slackened in

the neutral position of the spine when the normal height of the discs is reduced, as might occur with degenerative disc disease.

FIGURE 4-14

A. In flexion of the vertebral column, the anterior longitudinal ligament (ALL) is compressed whereas the posterior longitudinal ligament (PLL) is stretched. **B.** In extension of the vertebral column, the PLL is slack whereas the ALL is stretched.



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The **posterior longitudinal ligament** (PLL) (see Figs. 4–13A and 4–13C) runs on the posterior aspect of the vertebral bodies from C2 to the sacrum and forms the ventral

surface of the vertebral canal. It also consists of a superficial and a deep layer. In the superficial layer, the fibers span several mobile segments. In the deep layer, the fibers extend only to adjacent vertebrae, interlacing with the outer layer of the annulus fibrosus and attaching to the margins of the vertebral endplates.[10] In the lumbar region, the ligament narrows to a thin ribbon that provides little support for the interbody joints. The posterior longitudinal ligament is stretched in flexion (Fig. 4–14A) and is slack in extension (Fig. 4–14B).



Foundational Concepts 4–2

Potential Role of the Posterior Longitudinal Ligament in Disc Disorders

The narrow posterior longitudinal ligament in the lumbar region does not provide much support for the intervertebral discs, which is one of the factors contributing to the relatively high incidence of disc herniations in a posterolateral direction in the lumbar spine. In the lumbar spine, the posterior fibers of the annulus fibrosus are stressed most in combined flexion and rotation positions. Combined with excessive compressive loads such as occur with repetitive lifting and twisting with heavy objects, the posterior annulus can fail. The posterior annulus failure, combined with limited support from the posterior longitudinal ligament, can allow the nucleus pulposus to herniate in a posterolateral direction and possibly compress the exiting spinal nerve.

LIGAMENTUM FLAVUM

The **ligamentum flavum** is a thick, elastic ligament that connects lamina to lamina from C2 to the sacrum and forms the smooth posterior surface of the vertebral canal (see Figs. 4–13A and 4–13C). Some fibers extend laterally to cover the articular capsules of the zygapophyseal joints. Although the highest strain in this ligament occurs during flexion when the ligament is stretched,[2] this ligament is under constant tension even when the spine is in a neutral position because of its elastic nature. This highly elastic

nature serves two purposes. First, it creates a small but continuous compressive force on the discs. The raised pressure in the discs makes the discs stiffer and thus better able to provide support for the spine in the neutral position. Second, a highly elastic ligament in this location is advantageous because the ligament will not buckle on itself during movement. If the ligament did buckle on itself, it would compress the spinal cord in the vertebral canal, especially with any movement into extension.

Patient Application 4–1

Joyce is a 61-year-old woman with a 10 year history of intermittent neck pain. She has seen a physical therapist in the past for her neck pain and it has responded well to intervention. She presents to physical therapy now with familiar neck pain. However, she has new symptoms of weakness in both hands and has fallen three times in the last month. Examination confirms impaired balance and intrinsic hand muscle weakness, as well as identifying hyperreflexia of lower extremity deep tendon reflexes and a positive Hoffman's reflex. Compressive cervical myelopathy is suspected clinically and confirmed by an MRI that also shows a hypertrophied ligamentum flavum and ossified posterior longitudinal ligament.

The ligamentum flavum is subject to hypertrophy and loss of elastin due to aging, and this hypertrophy is accelerated with spondylosis (degeneration) of the spine. If the ligamentum flavum is hypertrophied and less elastic, it has the potential to buckle into the spinal canal and cause compression of the spinal cord, particularly with extension motions. Because the ligamentum flavum forms the posterior surface of the spinal canal, hypertrophy can produce central spinal stenosis (narrowing of the spinal canal). The buckling potential is greatest in the presence of degenerated intervertebral discs that also allow the ligament to be slack at rest. Spinal stenosis associated with ligamentum flavum hypertrophy is even greater if ossification of the posterior longitudinal ligament has occurred as well. If the spinal canal space becomes small enough, myelopathy will result. If the cross-sectional area of the canal is reduced by ≥ 30%, neurological symptoms will likely be present.[12]

INTERSPINOUS LIGAMENTS

The **interspinous ligament** connects spinous processes of adjacent vertebra (see Fig.

4–13A). It is a fibrous sheet consisting of type I collagen, proteoglycans, and profuse elastin fibers. The interspinous ligament is innervated by medial branches of the dorsal rami.[13–15] Most authors agree that the interspinous ligament resists flexion; however, it may also resist end-range extension and posterior shear of the superior vertebra on the vertebra below. Mahato found that the orientation of the fibers varied by level in the lumbar spine, with interspinous ligaments of the upper lumbar vertebrae having a more horizontal orientation and those of the inferior lumbar vertebrae having a more vertical orientation.[16] McGill suggests that the interspinous ligament produces anterior shear when the lumbar spine is in full flexion and that this should be considered in exercise prescription.[14]

SUPRASPINOUS LIGAMENT

The **supraspinous ligament** is a strong, cordlike structure that connects the tips of the spinous processes (see Figs. 4–13A and 4–13C).[2,17] The supraspinous ligament, like the interspinous ligament, is stretched in flexion and its fibers resist separation of the spinous processes. During hyperflexion, the supraspinous and interspinous ligaments are the first to fail.[18] The supraspinous ligament contains mechanoreceptors, and deformation of the ligament appears to play a role in the recruitment of spinal stabilizers such as the multifidus muscles.

INTERTRANSVERSE LIGAMENTS

The structure of the paired **intertransverse ligaments** is extremely variable by region. In general, the ligaments pass between transverse processes (see Fig. 4–13B) and are alternately stretched and compressed during lateral bending. The ligaments on the right side are stretched and offer resistance during lateral bending to the left, whereas the ligaments on the left side are slack and compressed during this motion. Conversely,

the ligaments on the left side are stretched during lateral bending to the right and offer resistance to this motion.

ZYGAPOPHYSEAL JOINT CAPSULES

The **zygapophyseal joint capsules** assist the ligaments in providing limitation to motion and stability for the vertebral column. The roles of the joint capsules also vary by region. The capsules are strongest in transition zones, those areas where the spinal configuration changes from lordotic to kyphotic (cervicothoracic junction) or from kyphotic to lordotic (thoracolumbar junction), presumably because of the potential for excessive stress in these areas. The joint capsules, like the supraspinous and interspinous ligaments, are vulnerable in extremes of flexion, especially in the lumbar region. Table 4–3 provides a summary of the ligaments and their functions.

Table 4-3

Major Ligaments of the Vertebral Column

Function

Kinematics

The motions available to the vertebral column as a whole are flexion/extension, lateral flexion, and rotation. These motions appear to occur independently of each other; however, at the level of the individual motion segment, these motions are often coupled. **Coupling** is defined as the consistent association of one motion about an axis with another motion around a different axis. The most predominant motions that

exhibit coupled behaviors are lateral flexion and rotation. It appears that pure lateral flexion and pure rotation do not occur in any region of the spine with the possible exception of the atlantoaxial joint, where rotation is the primary motion as the atlas pivots around the dens of the axis in a horizontal plane. Otherwise, for either lateral flexion or rotation to occur, at least some of the other motions must occur as well.[9,19]

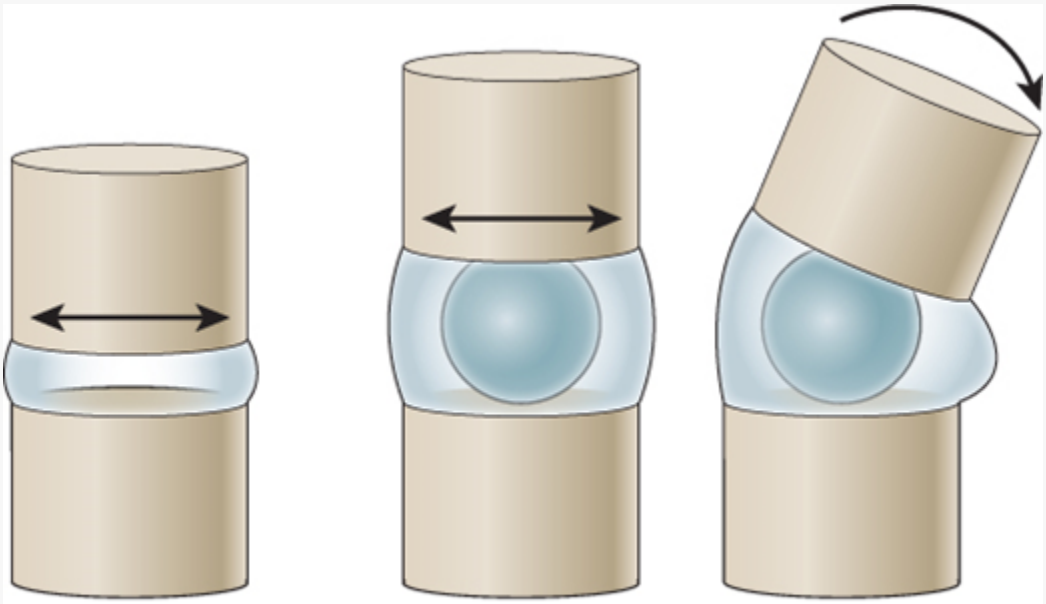
Coupling patterns, as well as the types and amounts of motion that are available, are complex. The patterns differ from region to region, depending on factors that include: the spinal posture and curves; the orientation of the articulating facets; the fluidity, elasticity, and thickness of the intervertebral discs; and the extensibility of the muscles, ligaments, and joint capsules.[19,20] It also appears that coupled motions vary widely among individuals and that there may be fewer patterns of coupled motions than once was believed.[21] The most consistent pattern of coupled motion occurs in the lower cervical spine where cervical lateral flexion to one side is accompanied by cervical rotation to the same side when either lateral flexion or rotation is initiated.[22]

Motions at the interbody and zygapophyseal joints are interdependent. The *amount* of motion available is determined primarily by the size of the discs, whereas the *direction* of the motion is determined primarily by the orientation of the facets. The intervertebral discs increase the amount of movement between two adjacent vertebrae. If the vertebrae were to lie flat against each other without discs, the movement between them would be limited to translation alone.[2] Vertebrae tilt on each other because the soft, deformable disc lies between them. As a consequence, discs add substantial (albeit differing) range of motion (ROM) to each mobile segment (Fig. 4–15). The fibers of the annulus fibrosus behave as a ligamentous structure and act as

restraints to motion of each mobile segment.

FIGURE 4-15

A. Without an intervertebral disc, only translatory motions could occur. **B.** The addition of an intervertebral disc separates the vertebral bodies, allowing a vertebra to both slide (translate) and tilt on the vertebra below, substantially increasing range of motion at the interbody joint.



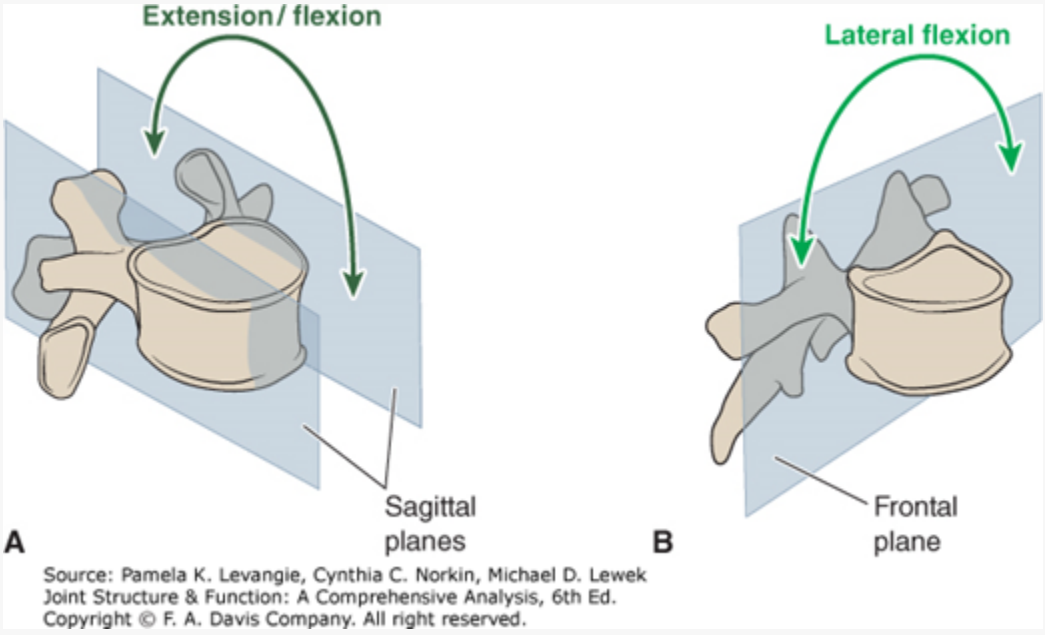
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The motions of flexion and extension occur as a result of the tilting and sliding of a superior vertebra over the inferior vertebra. As the superior vertebra moves through a ROM, it follows a series of different arcs, each of which has a different instantaneous axis of rotation.[23] Nucleus pulposus acts like a pivot but is able to undergo greater distortion than a mechanical pivot due to its viscoelastic composition.

A sliding motion occurs at the zygapophyseal joints as the vertebral body tilts over the disc at the interbody joint. The orientation of the zygapophyseal facet surfaces, varying from region to region (see Fig. 4–12), determines the direction of the tilting and sliding within a particular region (**Fig. 4–16**). If the superior and inferior zygapophyseal facet surfaces of adjacent vertebrae are oriented in the sagittal plane (as is approximated by the facets of a typical lumbar vertebra), the motions of flexion and extension are facilitated (**Fig. 4–16A**). On the other hand, if the zygapophyseal facet surfaces are oriented in the frontal plane (as is approximated by the facets of a typical thoracic vertebra), the predominant motion that is allowed is lateral flexion (**Fig. 4–16B**).

FIGURE 4-16

A. Zygapophyseal facet surfaces oriented in the sagittal plane (as seen in a typical lumbar vertebra) favor the motions of flexion and extension. **B.** Zygapophyseal facet surfaces oriented in the frontal plane (as seen in a typical thoracic vertebra) favor lateral flexion.



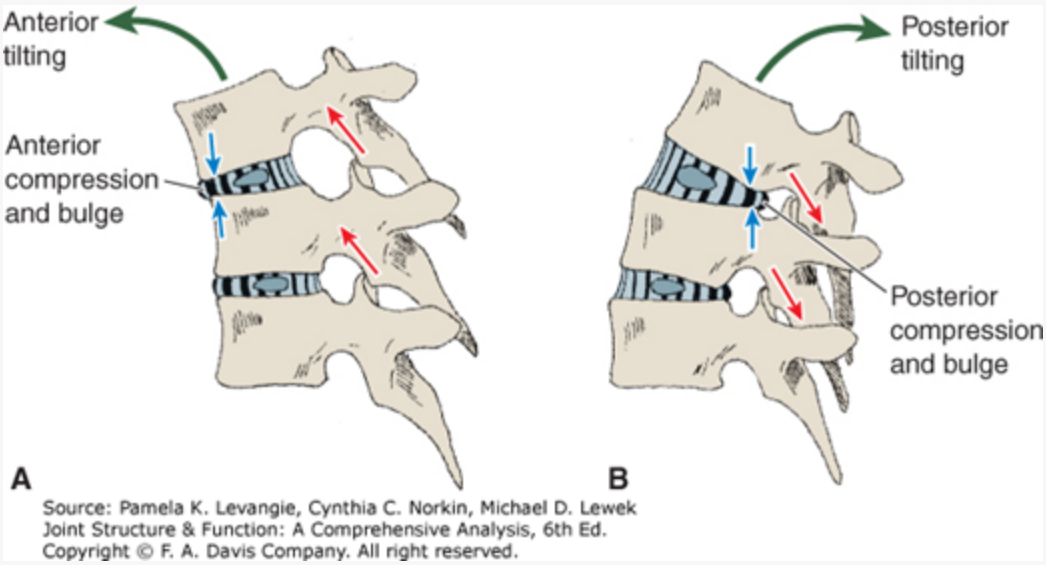
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FLEXION

In vertebral flexion, the vertebral body above anteriorly tilts and the inferior facets of that vertebra slide upwardly on the vertebral facets below, causing separation of the spinous processes and widening of the intervertebral foramen (Fig. 4–17). Although the amount of tilting depends partly on the size of the discs, supraspinous and interspinous ligament tension resists separation of the spinous processes and thus limits the flexion ROM. Passive tension in the zygapophyseal joint capsules, ligamentum flavum, posterior longitudinal ligament, and the spinal extensors also restrains flexion. With movement into flexion, the anterior portion of the annulus fibrosus is compressed whereas the posterior portion is stretched and resists separation of the vertebral bodies, restraining motion (Fig. 4–17A).

FIGURE 4-17

A. In flexion, the inferior facets of the superior vertebra slide up on the superior facets of the contiguous vertebra below whereas the vertebral body tilts anteriorly. The anterior tilting causes compression and bulging of the annulus fibrosus anteriorly and stretching of the annulus posteriorly. The intervertebral foramen widens. **B.** In extension, the inferior facets of the superior vertebra slide down on the superior facets of the contiguous vertebra below while the vertebral body tilts posteriorly. The anterior annulus fibrosus fibers are stretched, whereas the posterior portion of the disc is compressed and bulges posteriorly. The intervertebral foramen narrows.



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EXTENSION

In extension, posterior tilting and gliding of the superior vertebra on the contiguous inferior vertebra occurs, causing the spinous processes to approximate and the intervertebral foramen to narrow (Fig. 4–17B). The amount of motion available in extension is limited by bony contact of the spinous processes and passive tension in the zygapophyseal joint capsules, anterior fibers of the annulus fibrosus, anterior longitudinal ligament, and anterior trunk muscles. The only ligament that directly limits extension is the anterior longitudinal ligament, which is likely the reason that the anterior longitudinal ligament is stronger than the posterior longitudinal ligament.

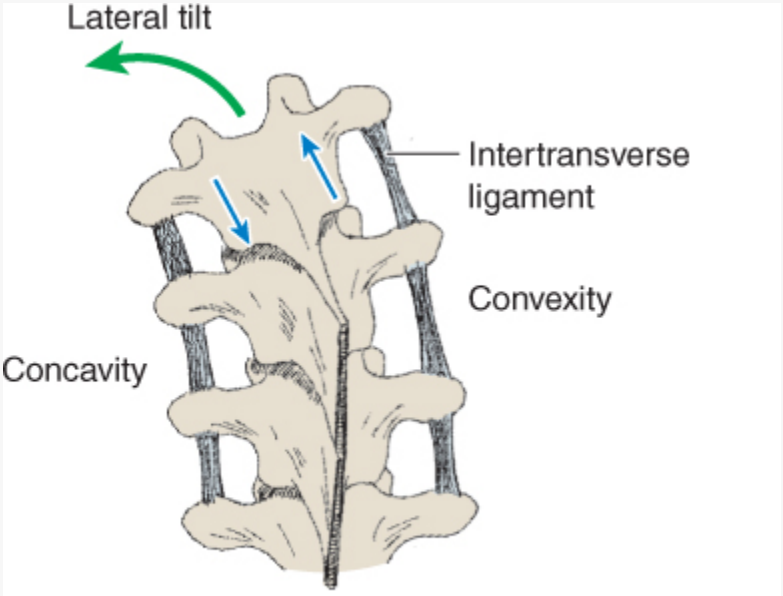
LATERAL FLEXION

In lateral flexion, the superior vertebra laterally tilts and translates over the contiguous vertebra below (Fig. 4–18). The intervertebral foramen is widened contralateral to the direction of lateral flexion and narrowed on the ipsilateral side. The annulus fibrosus is compressed on the concavity of the curve and stretched on the convexity of the curve. Passive tension in the annulus fibers, intertransverse ligaments, and anterior and

posterior trunk muscles on the convexity of the curve limit lateral flexion. The direction of rotation that accompanies lateral flexion differs slightly from region to region because of the differing orientation of the facets.

FIGURE 4-18

In lateral flexion to the left, the inferior facet on the concave (left) side of the bend slides down on the superior facet of the vertebra below whereas the inferior facet on the convex (right) side slides upward. The vertebral body tilts laterally, widening the intervertebral foramen on the convexity and narrowing that on the concavity. Lateral flexion is limited by tension in the intertransverse ligament on the convexity of the curve.



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ROTATION

Rotation is available in each spinal region, but due to the drastically different shapes of

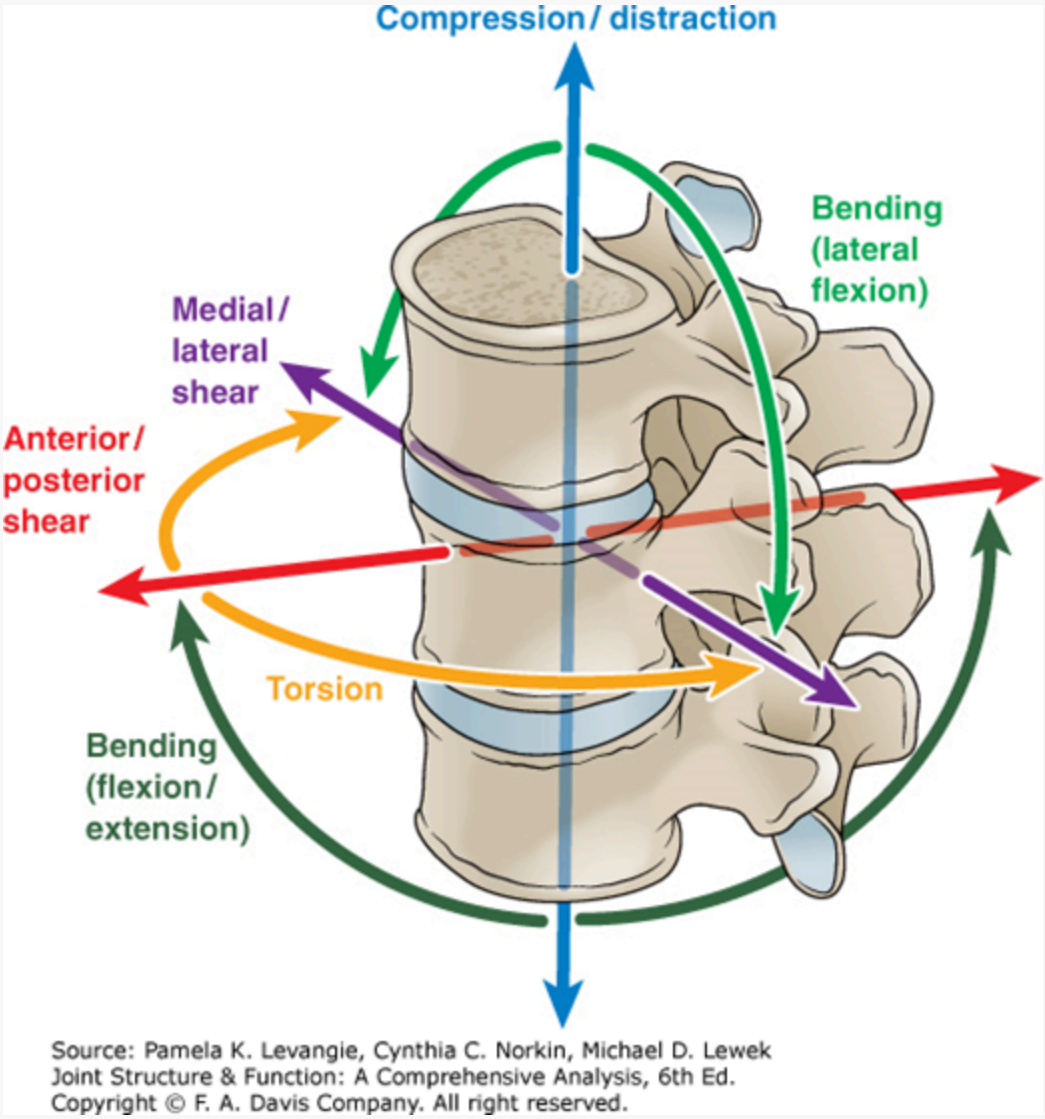
the zygapophyseal joints, the kinematics vary greatly by region and will be discussed by region in the following section.

Kinetics

The vertebral column is subjected to axial compression, tension (distraction), bending, torsion, and shear stress not only during normal functional activities but also at rest (Fig. 4–19). The column's ability to resist these loads varies among spinal regions and depends on: the type, duration, and rate of loading; the person's age and posture; the condition and properties of the various structural elements (vertebral bodies, joints, discs, muscles, joint capsules, and ligaments); and the integrity of the nervous system.

FIGURE 4-19

Each motion segment in the vertebral column is subjected to axial (compression/distraction), bending (flexion/extension, lateral flexion), torsional (rotation), and shear (anterior/posterior, medial/lateral) forces.



AXIAL COMPRESSION

Axial compression (force acting through the long axis of the spine at right angles to the discs) occurs as a result of the force of gravity, ground reaction forces, forces produced by the ligaments and muscular contractions, and any potential external load carried on the person's body. The discs and vertebral bodies resist most of the compressive force, but the neural arches and zygapophyseal joints share some of the load in certain postures and during specific motions. The compressive load is transmitted from the

superior vertebral endplate to the inferior endplate through the trabecular bone of the vertebral body and its cortical shell. The cancellous bone contributes much of the strength of a lumbar vertebra before the age of 40, and the cortical bone carries the remainder. After age 40, the cortical bone carries a greater proportion of the load, because the trabecular bone's compressive strength and stiffness decrease with decreasing bone density.[24] Depending on the posture and region of the spine, the zygapophyseal joints carry from 0% to 30% of the compression load. The spinous processes also may share some of the load when the spine is in hyperextension.

The nucleus pulposus acts as a ball of fluid that can be deformed by a compression force. The pressure created in the nucleus pulposus is actually greater than the force of the applied load.[25] When a weight is applied to the nucleus pulposus from above, the nucleus pulposus exhibits a swelling pressure and tries to expand outward toward the annulus fibrosus and the endplates (see Fig. 4–9). As the nucleus attempts to distribute the pressure in all directions, stress is created in the annulus fibrosus, and central compressive loading occurs on the vertebral endplates. Normally, the annulus fibrosus and the endplates are able to provide sufficient resistance to the swelling pressure in the nucleus pulposus to reach and maintain a state of equilibrium. The pressure exerted on the endplates is transmitted to the superior and inferior vertebral bodies. The discs and trabecular bone are able to undergo a greater amount of deformation without failure than either the cartilaginous endplates or cortical bone when subjected to axial compression. The endplates are able to undergo the least amount of deformation and therefore will be the first to fail (fracture) under high compressive loading. The discs will be the last to fail (rupture).

When the intervertebral discs are subjected to a constant load, the discs, like all connective tissues, exhibit creep (time-dependent deformation). This phenomenon produces typical diurnal changes in disc composition and function. Under sustained compressive loading such as that incurred in the upright posture, the increase in the swelling pressure in the disc causes fluid to be expressed from the nucleus pulposus and the annulus fibrosus, reducing the height of the disc. The amount of fluid expressed from the disc depends both on the size of the load and the duration of its application. The expressed fluid is absorbed through microscopic pores in the cartilaginous endplates. When the compressive forces on the discs are reduced again, the disc imbibes fluid back from the vertebral body, regaining height.[26] The creep of the disc with compression explains why a person is slightly shorter at the end of a day of prolonged axial compression from the upright position, and slightly taller in the morning after hours of reduced compression resulting from a horizontal position.



Patient Application 4–2

On March 1, 2016, U.S. Astronaut Scott Kelly returned to earth after spending 340 days on the International Space Station, setting a record for the longest consecutive time in space by a U.S. astronaut. NASA reported that Kelly gained 2 inches of height during his time in space given the zero gravity conditions that allowed discs to imbibe and maintain fluid, thereby lengthening the spine.



Expanded Concepts 4–1

Effects of Creep Loading on the Intervertebral Discs

Adams and colleagues reported mechanical changes in the disc with creep loading that include loss of intervertebral disc height, increased bulging of the disc, and more flexibility in bending.[27] The result of these changes is that the neural arch and the ligaments, especially the zygapophyseal joints, are subjected to large compressive and bending forces under conditions of creep loading of the disc. With prolonged

compressive forces, there is a shift in load from the nucleus pulposus to the annulus fibrosus, especially the posterior aspects of the disc. This increased load can cause buckling or prolapse of the annulus fibrosus. In addition, there is decreased exchange of fluid and a decrease in metabolism, thereby decreasing nutrition and potential healing of the disc.

BENDING

Bending causes both compression and tension on the structures of the spine. In forward flexion, the anterior structures (anterior portion of the disc, anterior ligaments, and muscles) are subjected to compression; the posterior structures are subjected to tension (see Fig. 4–14A). The resistance offered to the tensile forces by collagen fibers in the posterior outer annulus fibrosus, zygapophyseal joint capsules, and posterior ligaments helps to limit extremes of motion and thus provides stability in flexion. Creep occurs when the vertebral column is subjected to sustained loading, such as might occur in the flexed postures commonly assumed in sitting. The resulting deformation (elongation or compression) of supporting structures such as ligaments, joint capsules, and intervertebral discs may lead to an increase in the ROM beyond normal limits. If the creep deformation of tissues occurs within the toe or elastic regions of the stress-strain curve, the structures will return to their original dimensions either minutes or hours after cessation of the activity. If the deformation exceeds the elastic region, the vertebral structures may be at risk for injury.

In extension, the posterior structures generally are either unloaded or subjected to compression while the anterior structures are subjected to tension (see Fig. 4–14B). In general, resistance to extension is provided by the anterior outer fibers of the annulus fibrosus, the zygapophyseal joint capsules, passive tension in the anterior longitudinal ligament, and possibly by contact of the spinous processes. In lateral bending, the

ipsilateral side of the disc is compressed; that is, in right lateral bending, the right side of the disc is compressed and the outer fibers of the left side of the disc are stretched. Therefore, the contralateral fibers of the outer annulus fibrosus and the contralateral intertransverse ligament (see Fig. 4–18) help to provide stability during lateral bending by resisting extremes of motion.

TORSION

Torsional forces are created during axial rotation that occurs as a part of the coupled motions that take place in the spine. It has been suggested that the annulus fibrosus may be the most effective structure in the lumbar region for resisting torsion. However, the risk of rupture of the disc fibers is increased when there is a combination of torsion, heavy axial compression, and forward bending.[28]

When the disc is subjected to torsion, some of the annulus fibrosus fibers resist clockwise rotations whereas fibers oriented in the opposite direction resist counterclockwise rotations.

SHEAR

Shear forces occur parallel to the disc and tend to cause each vertebra to slide on the one below (translation anteriorly, posteriorly, or laterally). In the lumbar spine, the zygapophyseal joints resist some of the anterior shear force and the discs resist the remainder. When the load is sustained, the discs exhibit creep, and the zygapophyseal joints may have to resist all of the shear force. Table 4–4 summarizes vertebral function.

Table 4-4

Summary of Vertebral Function

Regional Structure and Function

The complexity of a structure that must fulfill many functions is reflected in the design of the structure's component parts, with more varied demands often requiring more complex structures. This concept is reflected in the vertebral column. Structural variations evident in the first cervical vertebra, first thoracic vertebra, fifth lumbar vertebra, and sacral vertebrae represent adaptations necessary for joining the vertebral column to adjacent structures. Differences in vertebral structure are also apparent at the cervicothoracic, thoracolumbar, and lumbosacral junctions where there is a transition from one type of vertebral structure to another. The vertebrae located at regional junctions are called **transitional vertebrae** and usually possess characteristics common to their contiguous regions. The cephalocaudal increase in the size of the vertebral bodies reflects the increased proportion of body weight that must be supported by the lower thoracic and lumbar vertebral bodies. Fusion of the sacral vertebrae into a rigid segment reflects the need for a firm base of support for the column. In addition to these variations, a large number of minor alterations in structure occur throughout the column. However, only the major variations are discussed here.

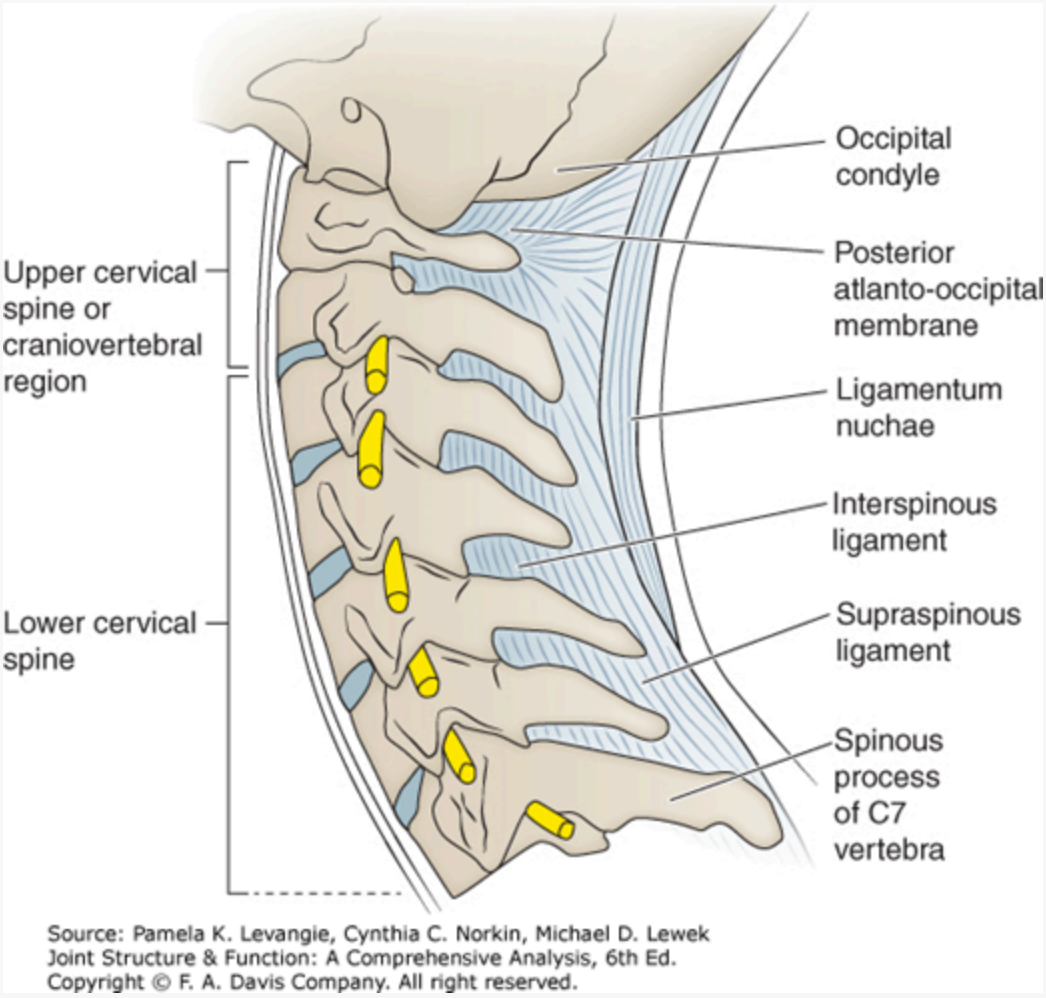
Structure of the Cervical Region

The cervical portion of the vertebral column consists of seven vertebrae. Morphologically and functionally, the cervical column is divided into two distinct

regions: the upper cervical spine, or craniovertebral region, and the lower cervical or subaxial cervical spine (Fig. 4–20). The **craniovertebral region** includes the occipital condyles and the first two cervical vertebrae (**C1** and **C2,** or, respectively, the **atlas** and **axis)** whereas the **lower cervical spine** includes the vertebrae of **C3 to C7** (Fig. 4–21). The vertebrae from C3 to C6 display similar characteristics and therefore are considered to be the typical cervical vertebrae. The atlas, axis, and C7 exhibit unique characteristics and are considered atypical cervical vertebrae. All of the cervical vertebrae have the unique feature of a **transverse foramen** located on the transverse processes that serve as passage for a vertebral artery.

FIGURE 4-20

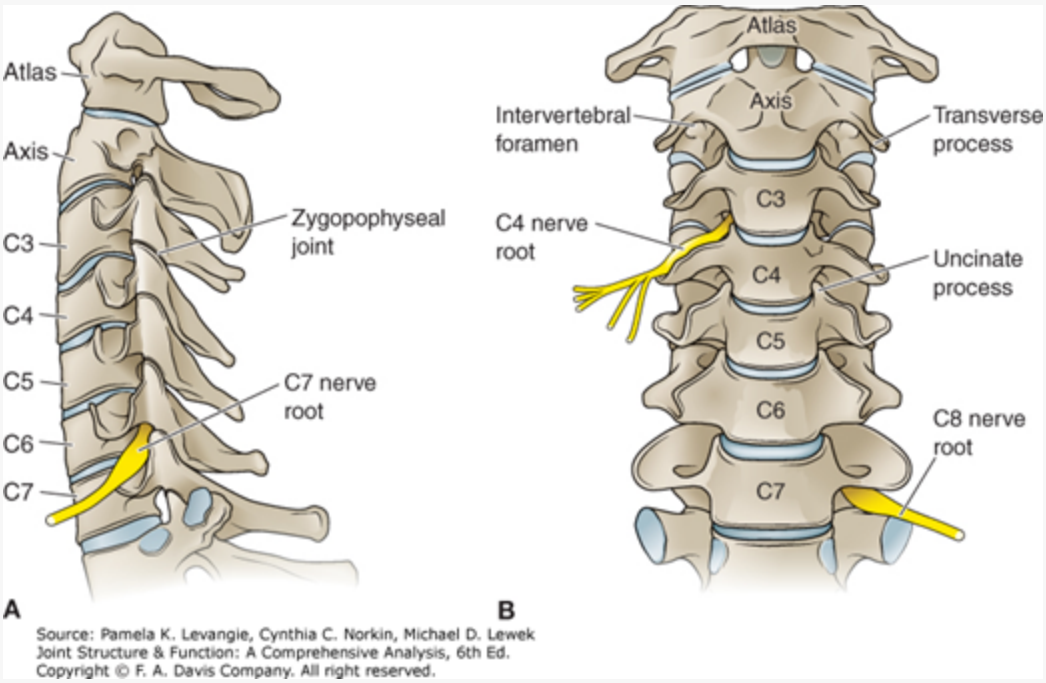
The cervical region consists of the upper cervical spine (craniovertebral region) and the lower cervical (subaxial) spine.



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FIGURE 4-21

Vertebrae of the cervical spine. **A.** Anterior view. **B.** Lateral view.



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Craniovertebral Region

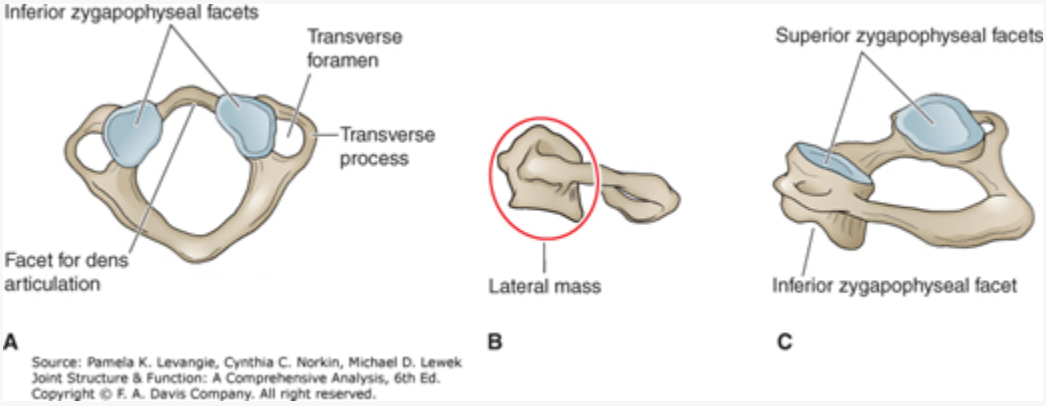
ATLAS

The atlas (C1) might be described as a firm ring sitting between the occipital condyles and the axis (Fig. 4–22). The functions of the atlas are to cradle the occiput and to transmit forces from the occiput to the lower cervical vertebrae. These functions are reflected in the bony structure. The atlas is different from other vertebrae in that it has no vertebral body or spinous process and is shaped like a ring (Fig. 4–22A). There are two large lateral masses on the atlas formed by the two superior and two inferior zygapophyseal facets (Figs. 4–22B and 4–22C) oriented to each occipital condyle to serve the function of maximizing the transmission of forces. These lateral masses are connected by an anterior and posterior arch that together form the ring structure and also create large transverse processes for muscle attachments.[5] The transverse processes, like those elsewhere in the cervical spine, each include a transverse foramen

(see Fig. 4–22A) through which the vertebral artery passes on each side. The superior zygapophyseal facets are large, typically kidney-shaped, and deeply concave to accommodate the large, convex articular surfaces of the occipital condyles. There is, however, wide variation in the size and shape of these facets. The inferior zygapophyseal facets are slightly convex and are directed inferiorly for articulation with the superior zygapophyseal facets of the axis (C2). The atlas also possesses a facet on the internal surface of the anterior arch for articulation with the dens **(odontoid process)** of the axis (see Fig. 4–22A).

FIGURE 4-22

The atlas is a markedly atypical vertebra. **A.** In this superior view, it is evident that the atlas is shaped like a ring and lacks a body and a spinous process. The transverse processes each have a transverse foramen common to all cervical vertebrae. **B.** The lateral view demonstrates the lateral mass. **C.** The zygapophyseal facets.



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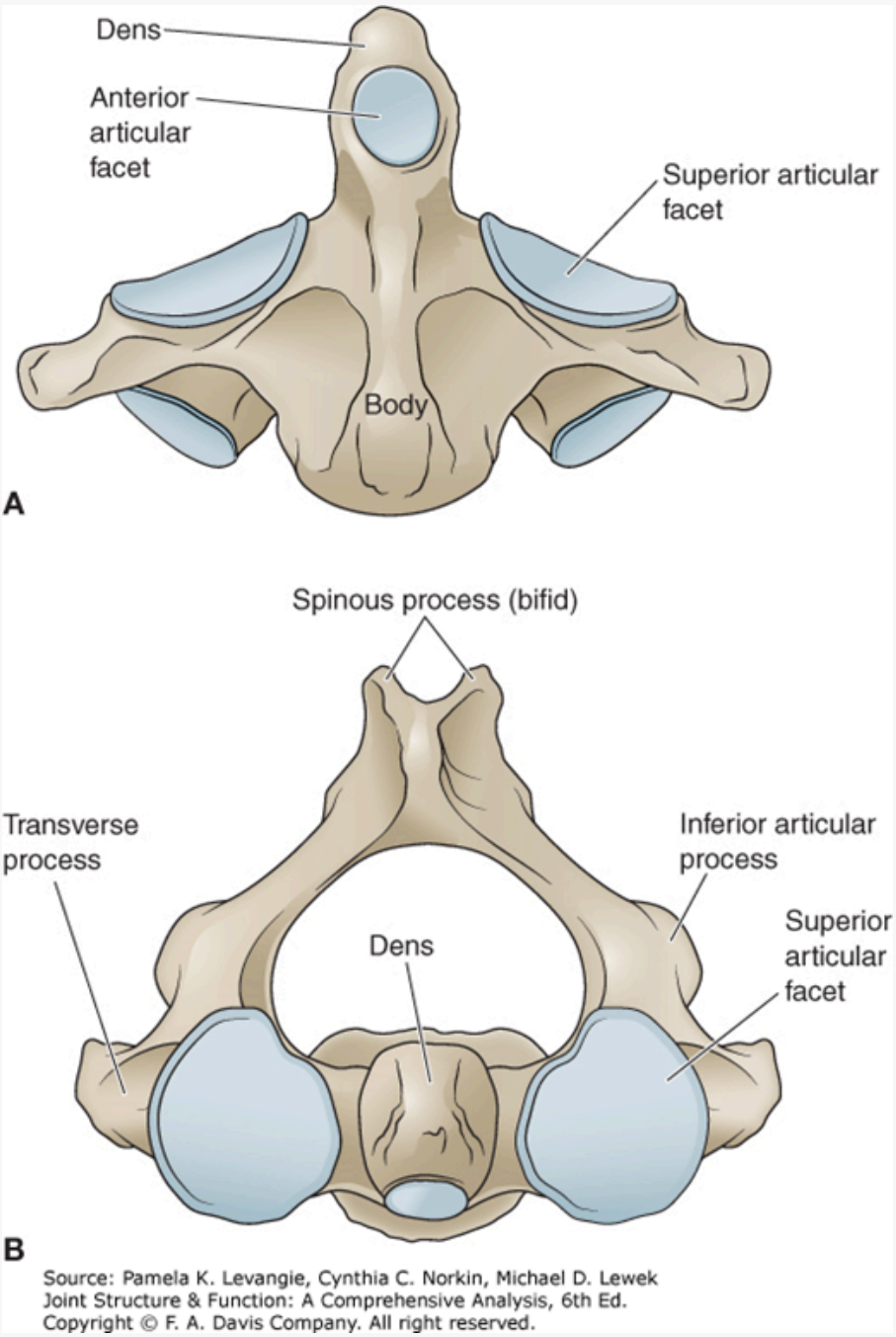
AXIS

The primary functions of the axis are to transmit the combined load of the head and atlas to the remainder of the cervical spine and to provide axial rotation of the head and

atlas.[5] The axis (Fig. 4–23) is atypical in that the anterior portion of the body extends inferiorly and a vertical projection called the **dens** arises from the superior surface of the body. The dens has an anterior facet for articulation with the anterior arch of the atlas (see Fig. 4–23A) and a posterior groove for articulation with the **transverse ligament.** The arch of the axis has superior and inferior zygapophyseal facets for articulation with atlas and C3, respectively. The spinous process of the axis is large and elongated, with a bifid (split into two portions) tip (see Fig. 4–23B). The superior zygapophyseal facets of the axis face upward and laterally. The inferior zygapophyseal facets face anteriorly.[29]

FIGURE 4-23

The dens (odontoid process) arises from the anterior portion of the body of the axis. The superior zygapophyseal facets are located on either side of the dens. **A.** An anterior view shows the anterior facet on the dens for the articulation of the dens with the anterior atlas and the inclination of the superior zygapophyseal facets. **B.** A superior view shows the bifid spinous process and other elements of the axis.



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Articulations

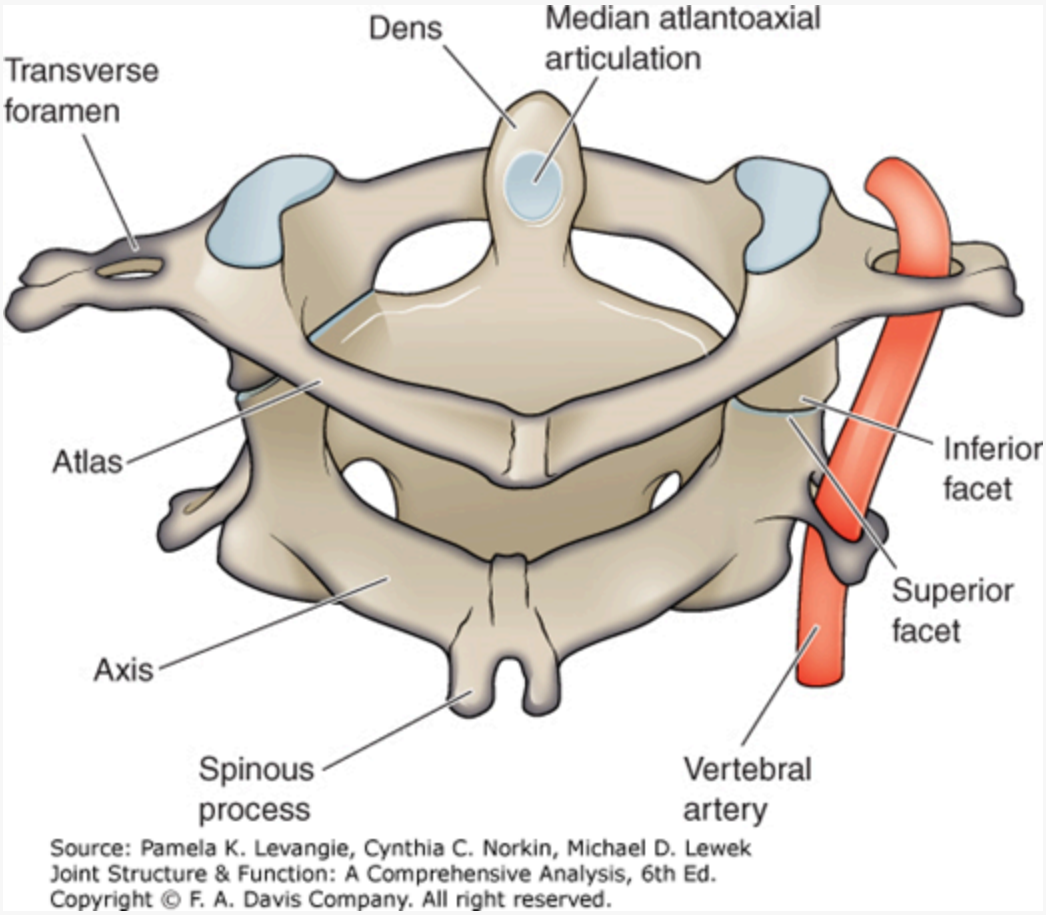
There are two **atlantooccipital joints** that consist of the two concave superior zygapophyseal facets of the atlas articulating with the two convex occipital condyles of the skull. These joints are true synovial joints with intra-articular fibroadipose

meniscoids, which lie close to the horizontal plane.

There are three synovial joints that compose the **atlantoaxial joints:** the median atlantoaxial joint between the dens and the anterior arch of atlas, and two lateral joints between the superior zygapophyseal facets of the axis and the inferior zygapophyseal facets of the atlas (Fig. 4–24). The median joint is a synovial pivot joint in which the dens of the axis rotates within an osteoligamentous ring formed by the anterior arch of the atlas and the transverse atlantal ligament posteriorly. The two lateral joints lie in the horizontal plane and, on the basis of bony structure, appear to be plane synovial joints; however, the articular cartilages of both the atlantal and axial facets are convex, rendering the zygapophyseal facet joints biconvex.[30] Joint spaces that occur as a result of the incongruence of the biconvex structure are filled with meniscoids.

FIGURE 4-24

Atlantoaxial articulations. The median atlantoaxial articulation is depicted with the posterior portion (transverse atlantal ligament) removed to show the dens and the anterior arch of the atlas. The two lateral atlantoaxial joints consisting of the superior facets of the axis and the inferior facets of the atlas lie on either side of the median atlantoaxial joint. This view also shows the right vertebral artery as it passes through the transverse foramens.



Craniovertebral Ligaments

Besides the ligaments mentioned earlier in this chapter that are found across regions of the spine, a number of other ligaments are specific to the cervical region. Many of these ligaments attach to the axis, atlas, or occiput and reinforce the articulations of the upper two vertebrae. Four of the ligaments are continuations of the longitudinal ligamentous system already described; the four remaining ligaments are specific to the cervical area.

As shown in Figure 4–25, the **posterior atlantooccipital** and **posterior atlantoaxial membranes** are fibroelastic membranes that are superior extensions of the ligamentum

flavum (Fig. 4–25A). Their structure, however, varies from the ligamentum flavum in that they are less elastic and therefore permit a greater ROM, especially into rotation.[31] The **anterior atlantooccipital** and anterior **atlantoaxial membranes** are superior extensions of the anterior longitudinal ligament (Fig. 4–25B). The **tectorial membrane** is the superior extension of the posterior longitudinal ligament in the upper two segments and is a broad, strong membrane that originates from the posterior vertebral body of the axis, covers the dens and its cruciate ligament, and inserts at the basilar groove of the occiput near the anterior rim of the foramen magnum (Fig. 4–26).[32]

FIGURE 4-25

A. Posterior atlanto-occipital and posterior atlantoaxial membranes.

B. Anterior atlanto-occipital and anterior atlantoaxial membranes.

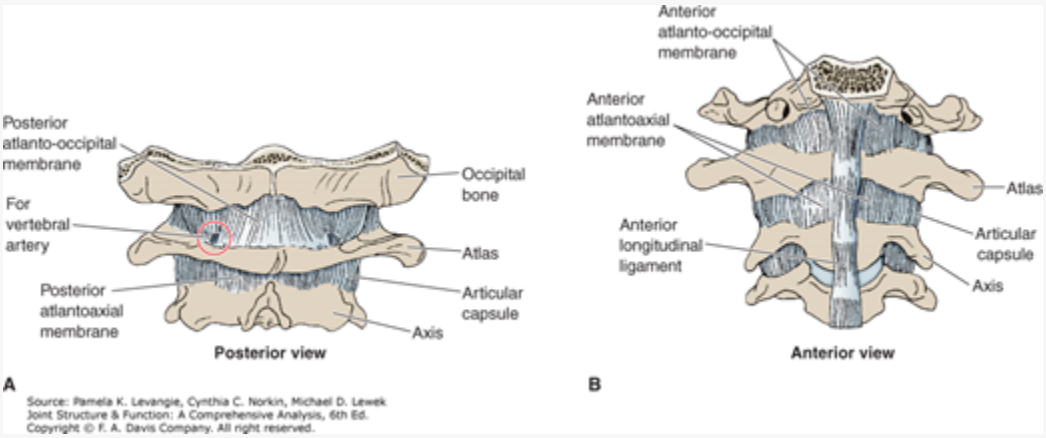
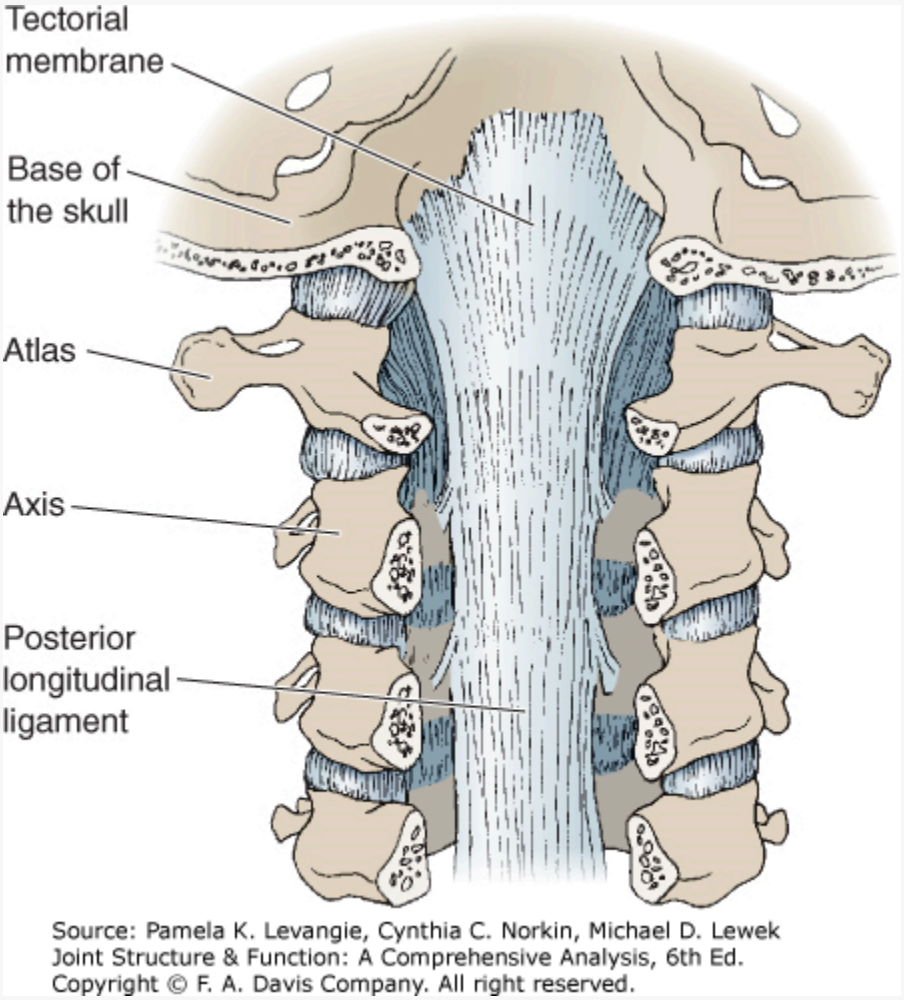


FIGURE 4-26

In this posterior view, the posterior vertebral arches are removed to show the tectorial membrane. The tectorial membrane is a continuation of the posterior longitudinal ligament in the

craniovertebral region.



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Descriptions of the **ligamentum nuchae** (see Fig. 4–21) vary. Some describe it as an evolution of the supraspinous ligament with a thick, triangular, sheet-like structure—the base of which is attached to the skull from the external occipital protuberance and foramen magnum and the apex of which is attached to the tip of the C7 spinous process.[15,17] Its function is sometimes described as resisting flexion of the head and serving as a site for muscle attachments.[33,34] Mercer and Bogduk,[34] however, describe the ligamentum nuchae as composed of two distinct parts: a dorsal raphe and a ventral midline septum. They describe the dorsal raphe as a narrow strip of

collagenous tissue interlacing with the tendons of the upper trapezius, splenius capitis, and the rhomboid minor muscles. The dorsal raphe is firmly attached to the external occipital protuberance and the tip of the C7 spinous process, but is unattached and mobile between these attachment sites. The ventral midline septum is composed of dense connective tissue running ventrally from the dorsal raphe and the external occipital crest to the tips of the cervical spinous processes.[34] It is continuous with the interspinous ligaments, the atlantoaxial and atlantooccipital membranes, and the fascia of the semispinalis capitis and splenius capitis muscles.[34] Mercer and Bogduk's description would appear to contradict the notion that the ligamentum nuchae is a strong ligament securely attached to all cervical vertebrae and capable of playing a primary role in resisting flexion of the head.[34] Johnson et al. found segmental variations in structure and fiber orientation that support either description depending upon the mobile segment and suggest that the ligamentum nuchae is most effective in the lower cervical region where it has well-defined attachments at C6 and C7.[35]

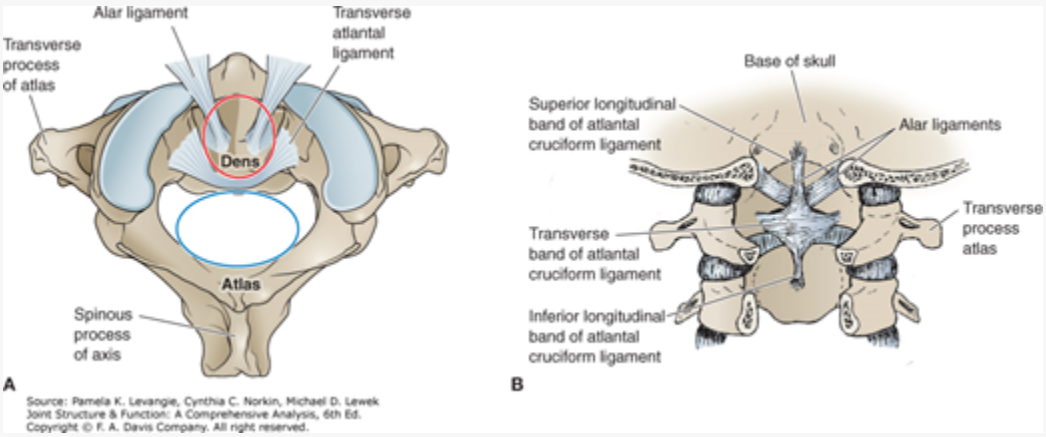
TRANSVERSE LIGAMENT

As shown in Figure 4–27, the **transverse atlantal ligament** stretches across the ring of the atlas and divides it into a large posterior ring for the spinal cord and a small anterior ring for the dens (Fig. 4–27A). The transverse ligament has a thin layer of articular cartilage on its anterior surface for articulation with the dens. The transverse ligament has superior fibers that extend upward to attach to the occipital bone and inferior fibers extending downward to the posterior portion of the axis. The transverse ligament and its longitudinal bands are sometimes referred to as the **atlantal cruciform ligament** (Fig. 4–27B). The transverse portion of the ligament holds the dens in close approximation against the anterior ring of the atlas and prevents anterior displacement of C1 on C2.

This ligament is critical in maintaining stability at the C1–C2 segment. The transverse atlantal ligament restrains craniovertebral flexion and ensures the pivot motion of the dens during axial rotation.[32] The transverse atlantal ligament is very thick and strong, and often the dens will fracture before the ligament will tear.[17] Integrity of the transverse ligament can be compromised, however, particularly with diseases such as rheumatoid arthritis and other disorders that compromise the connective tissues, such as Down syndrome.

FIGURE 4-27

The transverse atlantal (also known as the atlantal cruciform) ligament and the alar ligaments. **A.** In a superior view, it can be seen that the transversal atlantal ligament divides the ring of the atlas into a smaller anterior ring and a larger posterior ring. The alar ligaments attach axis (via the dens) to the occiput. **B.** A posterior view with the posterior portion of the vertebrae (spinous processes and portion of the arches removed) shows bands of the atlantal cruciform ligament and the attachment of the alar ligaments to the base of the skull.



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ALAR LIGAMENTS

The two alar ligaments are also specific to the cervical region (see Figs. 4–27A and B). These paired ligaments arise from the axis on either side of the posterior dens and extend laterally and sometimes slightly superiorly to attach to the occipital condyles.[32,36] The ligaments consist primarily of collagen fibers. These ligaments are relaxed with the head in neutral position and taut in craniocervical flexion. Axial rotation of the head and neck tightens both alar ligaments.[32] The **apical ligament** of the dens, also known as the superior longitudinal band of the atlantal cruciform ligament, connects the axis and the interposed atlas to the occipital bone of the skull. It runs in a fan-shaped arrangement from the apex of the dens to the anterior margin of the foramen magnum of the skull.[17]

The Lower Cervical Region

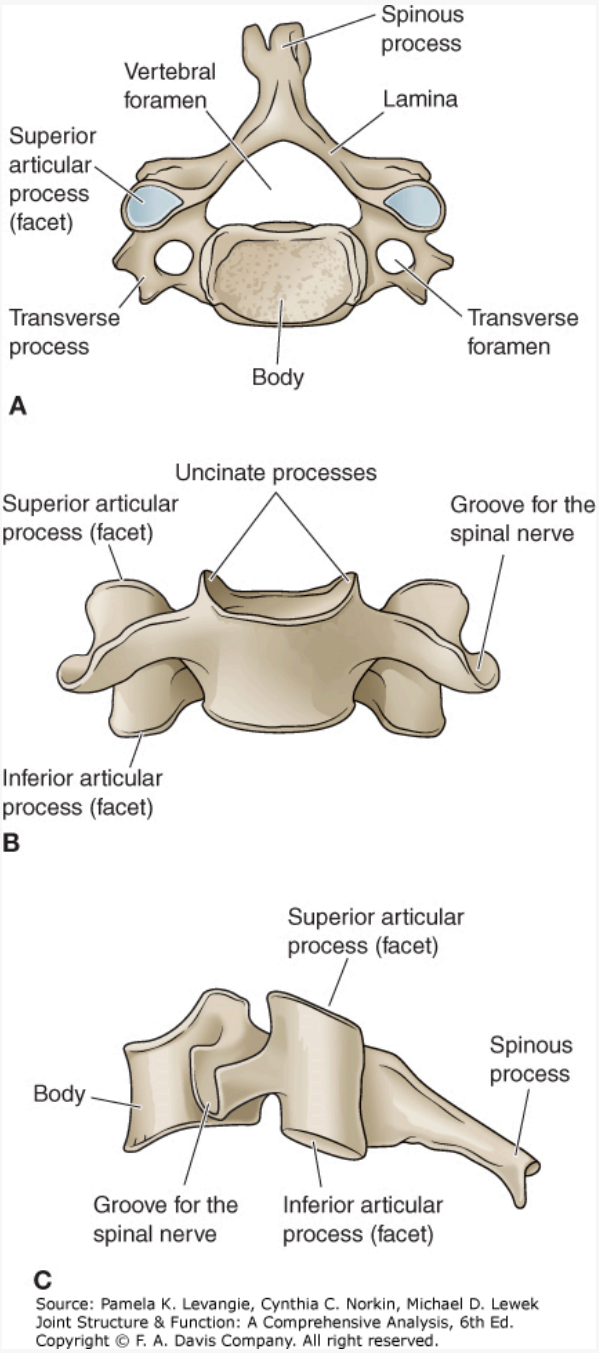
TYPICAL CERVICAL VERTEBRAE

BODY

As shown in Figure 4–28, the body of the cervical vertebra is small, with a transverse diameter greater than its anteroposterior diameter and height (Fig. 4–28A). The size of the body increases slightly at each level from C2 to C7. The posterolateral margins of the upper surfaces of the vertebral bodies from C3 to C7 support **uncinate processes** (Fig. 4–28B) that give the upper surfaces of these vertebrae a concave shape in the frontal plane. The uncinate processes are present prenatally, gradually enlarging between 9 to 14 years of age.[37] The anterior inferior border of the vertebral body forms a lip that hangs down toward the vertebral body below (Fig. 4–28C), which produces a concave shape of the inferior surface of the vertebra in the sagittal plane.

FIGURE 4-28

The typical cervical vertebra. **A.** The body and posterior elements of a typical cervical vertebra are evident in this superior view **B.** In this posterior view, uncinate processes on the posterolateral superior and inferior surfaces of the body create a concavity in the frontal plane. **C.** A lateral view shows the inferior projection of the anterior body that results in a concavity of the body in the sagittal plane.



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ARCHES

PEDICLES. The pedicles project posterolaterally and are located halfway between the superior and inferior surfaces of the vertebral body.

LAMINAE. The laminae are thin, slightly curved, and project posteromedially.

ZYGAPOPHYSEAL ARTICULAR PROCESSES (SUPERIOR AND INFERIOR). The cervical articular processes support paired superior and inferior facets. The superior facets are flat and oval and face superiorly and posteriorly. They lie between the transverse and frontal planes. The width and height of the superior zygapophyseal facets gradually increase from C3 to C7. The paired inferior facets face inferiorly and anteriorly.

TRANSVERSE PROCESSES. The transverse foramen is located in each of the transverse processes for the bilateral vertebral artery, vein, and venous plexus. Also, there is a groove for the spinal nerves that divides the cervical transverse processes into anterior and posterior portions or tubercles (see Fig. 4–28B and C).

SPINOUS PROCESSES. The cervical spinous processes are short and slender, extend horizontally, and have a bifid tip. The length of the spinous processes decreases slightly from C2 to C3, remains constant from C3 to C5, and undergoes a significant increase at C7.

VERTEBRAL FORAMEN. The vertebral foramen is relatively large and triangular to accommodate the large spinal cord.

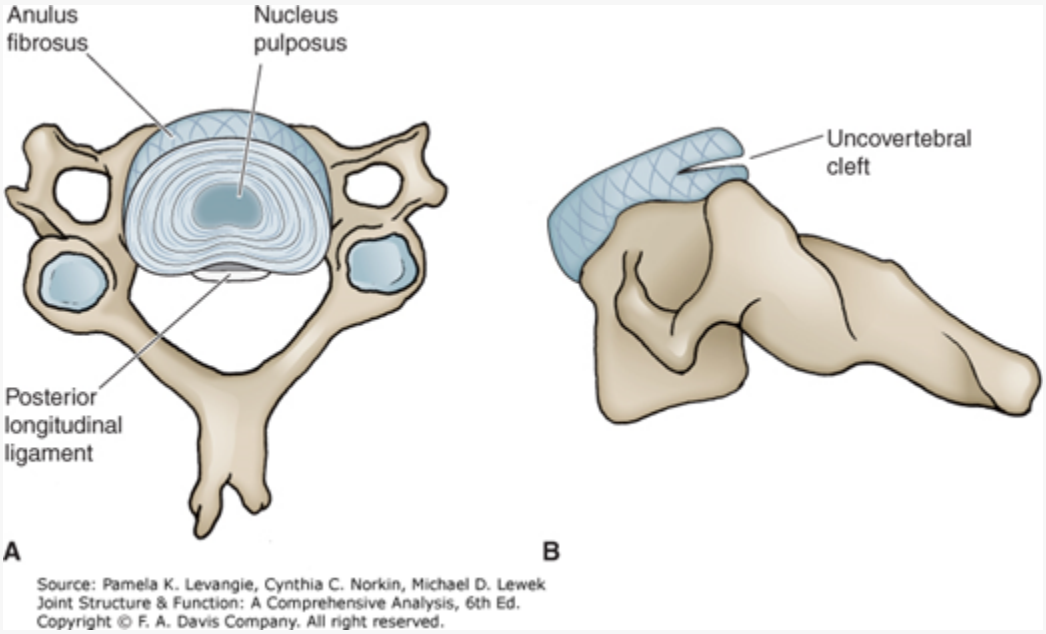
Intervertebral Disc

The structure of the intervertebral disc in the cervical region is different from that in the lumbar region (Fig. 4–29). Mercer and Bogduk state that in the cervical region the fibers of the annulus fibrosus have a crescent shape when viewed from above. The cervical discs are thick anteriorly and taper laterally as the discs approach their respective

uncinate processes. The posterior fibers of the annulus form only a thin layer with far fewer concentric rings than those of the lumbar spine.[5,30] Mercer and Bogduk describe that there is no true annulus in the posterior aspect of the cervical disc. However, Pouders et al. did find annulus fibers posteriorly although they agree that the annulus is thicker on the anterior aspect of the cervical disc.[38]

FIGURE 4-29

Cervical intervertebral disc. **A.** This superior view shows the crescent-shaped annulus fibrosus. **B.** The lateral view shows the uncovertebral cleft, also known as the uncovertebral joint or joints of Luschka.



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Fissures in the disc develop along with development of the uncinate processes and become clefts by approximately 9 years of age (Fig. 4–29B). These clefts become the joint cavity of what has been known as the **uncovertebral joints** or **"joints of Luschka."**[37]

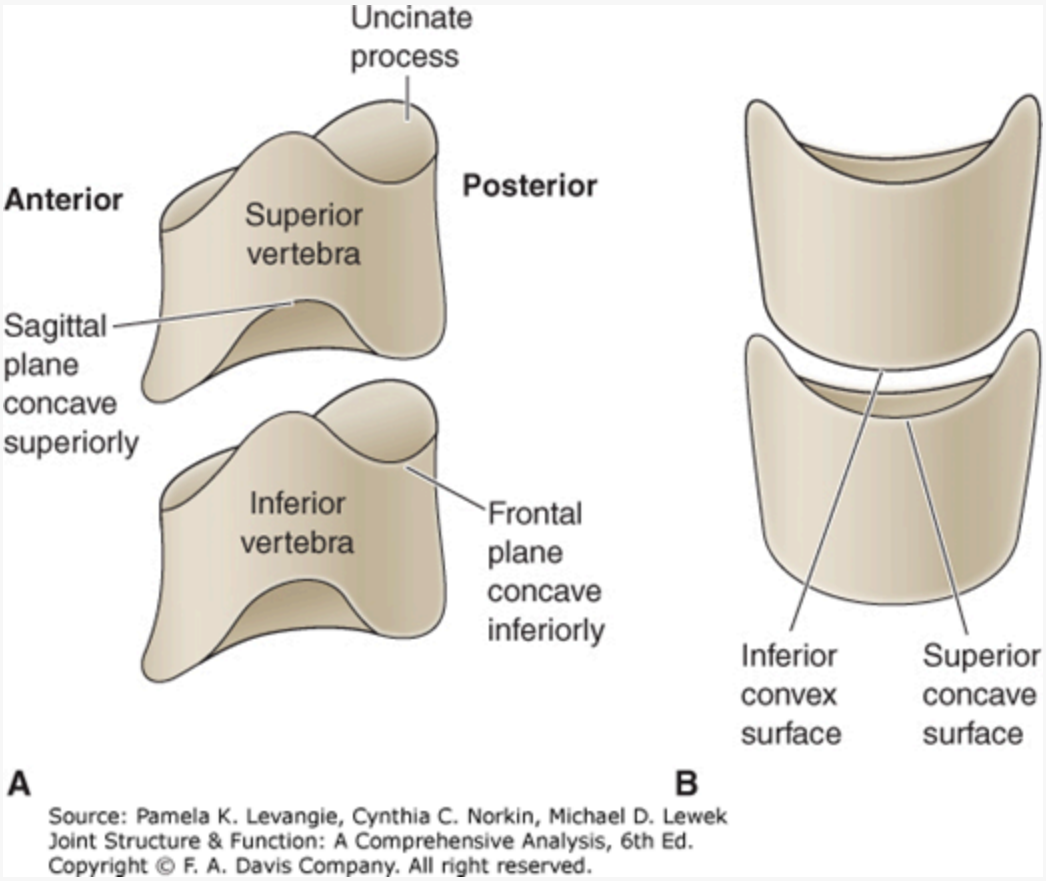
Interbody Joints of the Lower Cervical Region

The interbody joints of the lower cervical region (C3 to C7) are saddle joints (Fig. 4–30). In the sagittal plane, the inferior surface of the cranial vertebra is concave and the superior surface of the caudal vertebra is convex because of the uncinate processes (Fig 4–30A).[6] The motions that occur are predominantly tilting motions, although translatory motions remain available.[6,30] In the frontal plane (Fig. 4–30B), the inferior surface of the cranial vertebra is convex and sits in the concave surface of the caudal vertebra created by the uncinate processes.

FIGURE 4-30

A. Lateral view of an interbody saddle joint in the lower cervical spine.

B. Anterior view showing how the convex inferior surface of the superior vertebra fits into the concave superior surface of the inferior vertebra.



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Zygapophyseal Joints

The zygapophyseal joints in the cervical spine, as in other regions, are true synovial joints and contain fibroadipose meniscoids.[5,6,30] The joint capsules are lax to allow a large ROM; however, the capsules do restrict motion at the end of the available ranges. The joints are generally described as oriented approximately 45° from the frontal and horizontal planes; in other words, they lie midway between the two planes (see Fig. 4–12A). However, the orientation of the joints varies widely according to the individual, contributing to wide differences in ROM among individuals.[39]

Function of the Cervical Region

Although the cervical region demonstrates the most flexibility of any of the regions of

the vertebral column, stability of the cervical region—especially of the atlantooccipital and atlantoaxial joints—is essential for support of the head and protection of the brain stem, spinal cord, and vertebral arteries. The design of the atlas is such that it provides more free space for the spinal cord than does any other vertebra. The extra space helps to ensure that the spinal cord is not impinged during the large amount of motion that occurs there.

The differences in the two functional units of the craniocervical region and the lower cervical spine allow for different combinations of motions to occur between the head and neck in each region. Functionally, more combinations of motion are beneficial to allow optimal positioning of the sensory organs of the vestibular, visual, and auditory systems.

Kinematics

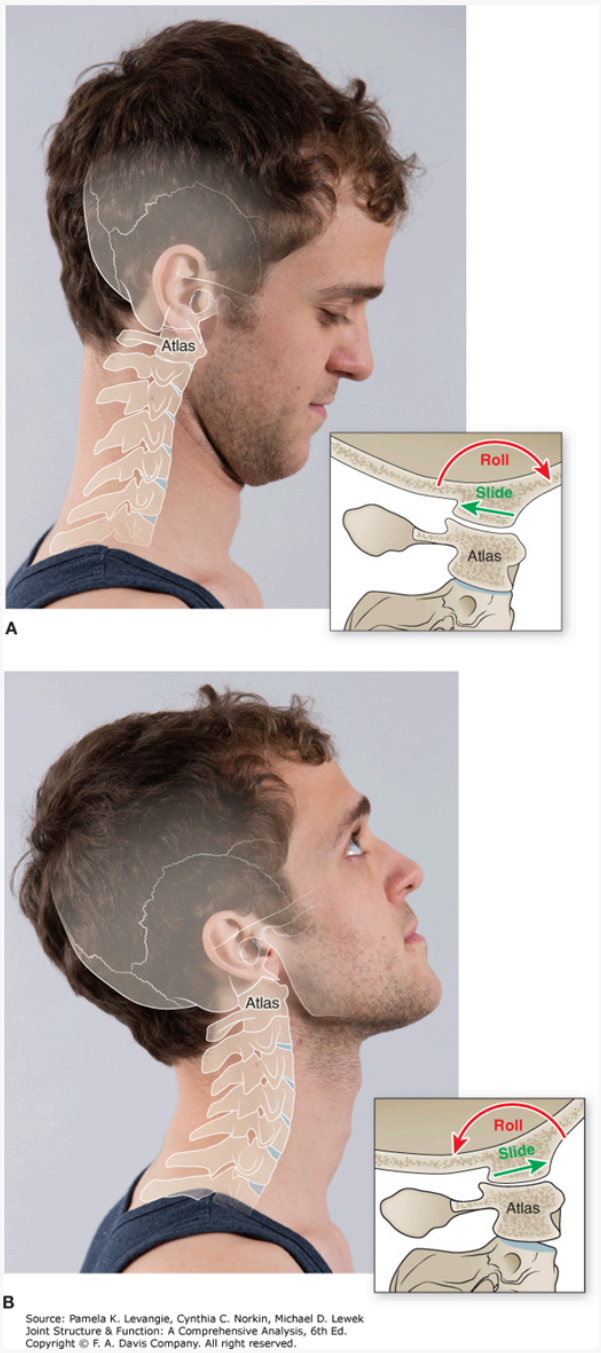
The cervical spine is designed for a relatively large amount of mobility. The motions of flexion and extension, lateral flexion, and rotation are permitted in the cervical region. These motions are accompanied by translations that increase in magnitude from C2 to C7. However, the predominant translation occurs in the sagittal plane during flexion and extension.[5,6]

The atlantooccipital joints primarily allow for nodding (flexion/extension) movements between the head and the atlas, although very small amounts of lateral flexion and rotation are possible (**Fig. 4–31**). The combined ROM for flexion/extension is approximately 15° with approximately 3° each of lateral flexion and axial rotation.[30] In flexion, the occipital condyles roll forward and slide backward (**Fig. 4–31A**). In extension, the occipital condyles roll backward and slide forward (**Fig. 4–31B**). The total motion

available in axial rotation and lateral flexion is extremely limited by tension in the joint capsules that occurs as the occipital condyles roll up the walls of the atlantal sockets on the contralateral side of either the rotation or lateral flexion.[40]

FIGURE 4-31

The primary motion available at the atlantooccipital joint is nodding (flexion/extension). **A.** In flexion, the condyles of the occiput roll forward and slide posteriorly. **B.** In extension, the condyles of the occiput roll backward and slide anteriorly.



Motions at the atlantoaxial joint include rotation, lateral flexion, flexion, and extension. Approximately 50% of the total rotation of the cervical region occurs at the atlantoaxial joints (Fig. 4–32). The atlas pivots approximately 45° to either side for a total of about 90°.[30] The alar ligaments limit rotation at the atlantoaxial joints. The remaining 40% of

total rotation available to the cervical spine is distributed evenly in the lower cervical joints.[40]

FIGURE 4-32

Atlantoaxial rotation takes place as motion of the occiput and atlas together on the axis below, typically resulting in 45° of rotation to either side.



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The shape of the zygapophyseal and interbody joints along with the ligaments and zygapophyseal joint capsules determine the motion at the lower cervical segments. The

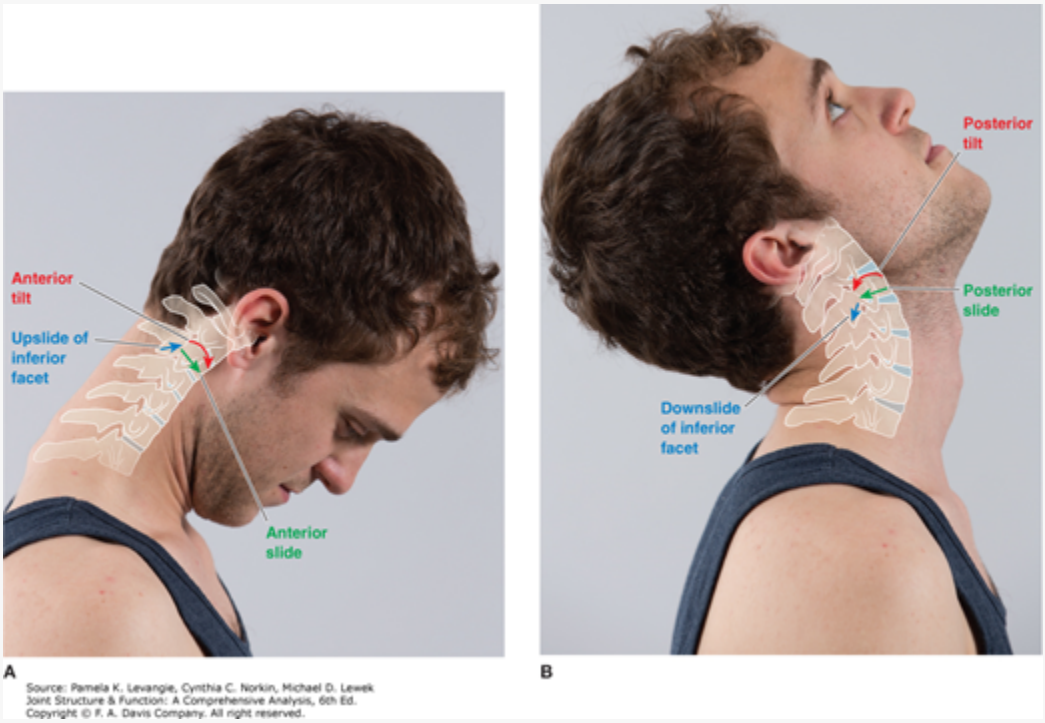
zygapophyseal joint capsules are generally lax in the cervical region, contributing to the large amount of motion available here. The height in relation to the diameter of the discs also plays an important role in determining the amount of motion available. The height of the cervical discs is large in comparison with the anteroposterior and transverse diameters. Therefore, a large amount of flexion, extension, and lateral flexion may occur at each segment, especially in young people. Average ROMs in the cervical spine across all age groups are as follows: total flexion/extension: 126° (standard deviation [SD] = 22°), total lateral flexion: 87° (SD = 22°), and total rotation: 144° (SD = 23°), with wide variation among age groups.[41] The disc at C5–C6 is subject to a greater amount of stress than other discs because C5–C6 has the greatest range of flexion/extension and is the area where the mechanical strain is greatest.[42]

In general, the range for flexion and extension increases from the C2–C3 mobile segment to the C5–C6 mobile segment and decreases again at the C6–C7 segment.[9] Flexion of these lower cervical segments includes anterior tilt of the superior vertebral body coupled with anterior slide at the interbody joints and upslide at the zygapophyseal joints (Fig. 4–33A). Extension includes posterior tilt of the cranial vertebral body coupled with posterior slide at the interbody joints and downslide at the zygapophyseal joints (Fig. 4–33B).

FIGURE 4-33

Flexion and extension of the lower cervical segments take place as movements of the cephalad vertebra (e.g., C3) on the adjacent vertebra below (e.g., C4). **A.** In flexion, the vertebral body tilts and

slides anteriorly while the inferior facet slides upward on the facet below. **B.** In extension, the vertebral body tilts and slides posteriorly while the inferior facet slides downward on the facet below.

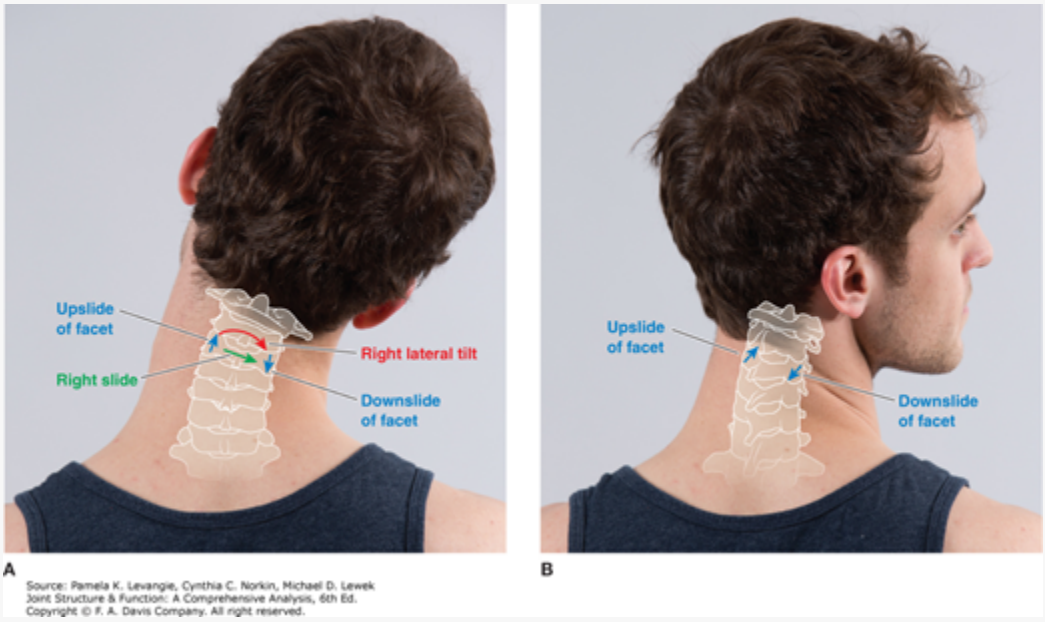


Rotation and lateral flexion are also coupled motions. Lateral flexion is coupled with ipsilateral rotation (Fig. 4–34A) and rotation is coupled with ipsilateral lateral flexion (Fig. 4–34B). These motions are also a combination of vertebral tilt and slide to the ipsilateral side at the interbody joints and ipsilateral downward slide and contralateral upslide at the zygapophyseal joints.[20,30]

FIGURE 4-34

Lateral flexion and rotation of the lower cervical segments are coupled movements of the cephalad vertebra (e.g., C3) on the

adjacent vertebra below (e.g., C4). **A.** In right lateral flexion, the vertebral body tilts and slides to the right while the right inferior facet slides down and the left inferior facet slides up on the facet below. The sliding of the facets produces slight rotation of the vertebral bodies to the right (ipsilaterally). **B.** In rotation to the right, the right inferior facet slides posteriorly while the left inferior facet slides anteriorly. Some right lateral tilt and slide of the vertebral body are also evident.



Mercer and Bogduk[5,30] suggested that the notion of lateral flexion and transverse plane rotation are an artificial construct. In their view, movement should be viewed as sliding that occurs in the plane of the zygapophyseal joints.

Kinetics

The cervical region is subjected to axial compression, tension, bending, torsion, and shear stresses, as is the remainder of the spinal column; however, there are some

regional differences. The cervical region differs from the thoracic and lumbar regions in that it bears less weight and is generally more mobile. The cervical spine is lordotic and therefore experiences anterior shear forces even in the ideal upright position.

No discs are present at either the atlantooccipital or atlantoaxial articulations. Therefore, the weight of the head (compressive load) must be transferred directly through the atlantooccipital joint to the articular facets of the axis. These forces are then transferred through the pedicles and laminae of the axis to the inferior surface of the body and to its two inferior zygapophyseal articular processes. Subsequently, the forces are transferred to the adjacent inferior disc. The laminae of the axis are large, with the structure reflecting the functional adaptation necessary to transmit the substantial compressive loads. The trabeculae show that the laminae of both the axis and C7 are heavily loaded, while the laminae of the intervening vertebrae are not. Loads diffuse into the laminae as they are transferred from superior to inferior articular facets.[43]

The loads imposed on the cervical region vary with the position of the head and body and are minimal in a well-supported reclining body posture. In the cervical region from C3 to C7, compressive forces are transmitted by three parallel columns: a central column formed by the vertebral bodies and discs, and two posterolateral columns composed of the left and right zygapophyseal joints. These posterolateral columns are often referred to as the articular pillars. The compressive forces are transmitted mainly by the bodies and discs, with approximately one-third transmitted by the two posterolateral columns.[29]

Foundational Concepts 4–3

Structure and Function of the Cervical Region

To summarize, goals of the cervical region are unique. The cervical vertebrae must achieve many combinations of motions to position the primary sensory organs optimally while still providing enough stability to protect the brain stem, spinal cord, and arteries that supply the brain. The organs associated with vision (eyes), equilibrium (vestibular apparatus), and hearing (ears) are all influenced by movement of the cervical spine. Ideally, these sensory organs will remain optimally oriented despite movement of our bodies. Having two distinct regions of the cervical spine that can function somewhat independently helps meet these different demands. The craniovertebral segments are able to move in opposite directions from the lower cervical spine as needed. Two common examples of these distinct and opposite motions within the cervical spine are found with **protraction** and **retraction** of the cervical region. Protraction involves craniovertebral extension combined with lower cervical flexion. Retraction involves craniovertebral flexion combined with lower cervical extension. When a person slouches while sitting, he or she will often assume a forward head posture, which is the protracted position of the cervical region. Physical therapists often teach exercises that involve retraction of the cervical region to help correct the problems that may arise from adopting a protracted cervical (forward head) position over a long period of time.

Structure of the Thoracic Region

Typical Thoracic Vertebrae

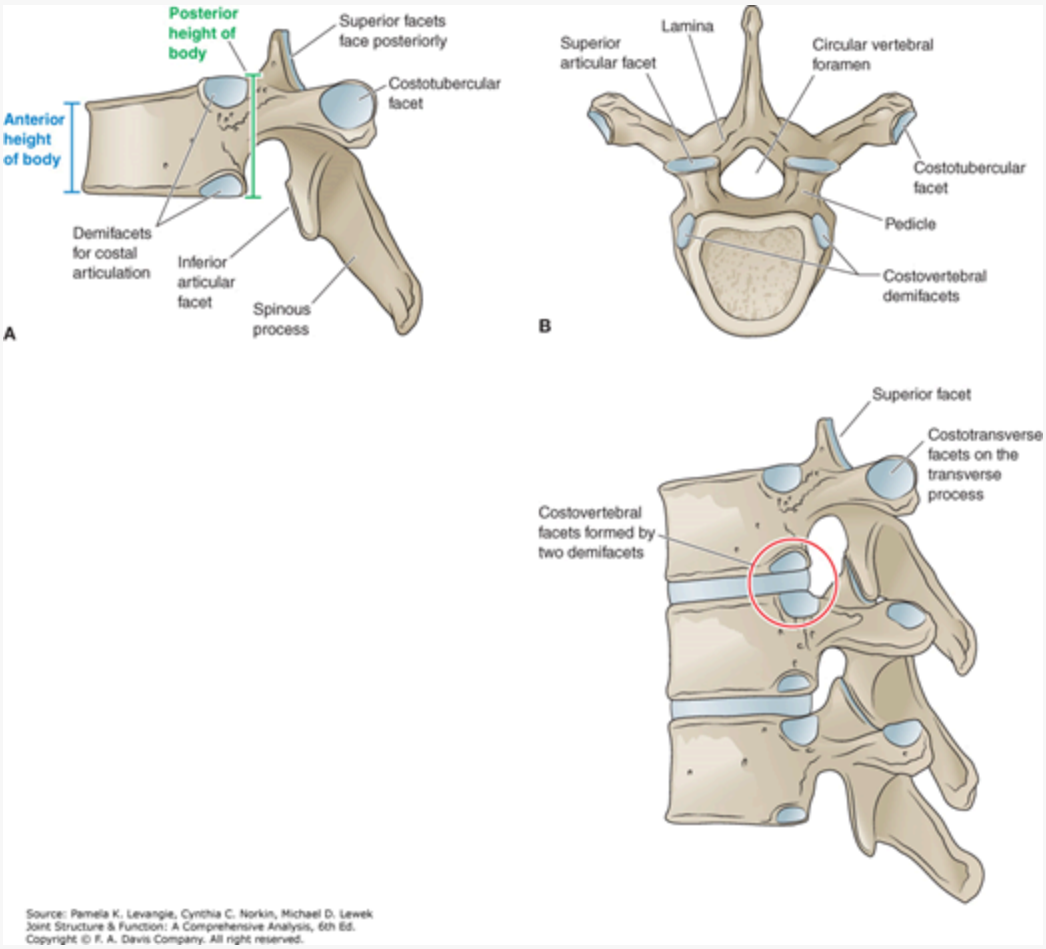
BODY

The features of a typical thoracic vertebra are shown in Figure 4–35. The body of a typical thoracic vertebra has equal transverse and anteroposterior diameters, which gives greater stability. The vertebral bodies are wedge-shaped, with posterior height greater than anterior height (Fig. 4–35A), with the peak anteroposterior height difference occurring at T7. This anterior wedging produces the normal kyphotic posture of the thoracic spine. **Demifacets** (or half facets) for articulation with the heads of the

ribs are located on the posterolateral corners of the vertebral plateaus (see Fig. 4–35).

FIGURE 4-35

Typical thoracic vertebra. **A.** A lateral view of a thoracic vertebra shows the superior and inferior facets of the zygapophyseal joints and the demifacets for articulation with the ribs. The posterior height of the body is greater than the anterior height. **B.** A superior view of a thoracic vertebra shows the small, circular shape of the vertebral foramen, the costotubercular facets on the transverse processes for articulation with the tubercles of the ribs, and the superior costovertebral facets on the vertebral body for articulation with the heads of the ribs, and the pedicles and laminae. **C.** In this lateral view, it can be appreciated that a costovertebral facet is formed by two adjacent demifacets and the intervening disc; the overlapping of spinous processes characteristic of the thoracic region is also evident.



ARCHES

PEDICLES

These generally face directly posteriorly with little to no lateral projection, creating a small vertebral canal (Fig. 4–35B).

LAMINAE

The laminae are short, thick, and broad.

ZYGAPOPHYSEAL ARTICULAR PROCESSES

The superior zygapophyseal facets are thin and almost flat and face posteriorly, superiorly, and laterally (Fig. 4–34A). The inferior zygapophyseal facets face anteriorly,

inferiorly, and medially. The facets lie nearly in the frontal plane, with their orientation changing at either T10 or T11 so that the superior facets face posterolaterally and the inferior facets face anterolaterally—oriented closer to the sagittal plane.

TRANSVERSE PROCESSES

The transverse processes have thickened ends that each support a large oval facet (**costotubercular facets**) for articulation with the tubercles of the ribs.

SPINOUS PROCESSES

The spinous processes slope inferiorly and, from T5 to T8, overlap the spinous process of the adjacent inferior vertebra. The spinous processes of T11 and T12 are triangular and project horizontally. For most of the thoracic spine, the tip of the spinous process lies at the level of the caudal vertebral body.

VERTEBRAL FORAMEN

The vertebral foramen is small and circular.

Intervertebral Discs

There has been little study of the structure of the thoracic intervertebral discs; however, the structure is generally held to be similar to that of discs in the lumbar region, with differences only in size and shape. Thoracic intervertebral discs are thinner than those in other regions, especially in the upper thoracic segments. Also, the ratio of disc size to vertebral body size is smallest in the thoracic region, resulting in less mobility and greater stability for this region. The endplates show a gradual increase in transverse and anteroposterior diameters from T1 to T12.

Articulations

INTERBODY JOINTS

The interbody joints of the thoracic spine involve flat vertebral surfaces that allow for translations in each direction to occur. The intervertebral discs allow for tilting of the vertebral bodies; however, relatively small disc size limits the total available motion.

ZYGAPOPHYSEAL JOINTS

The zygapophyseal joints are plane synovial joints with fibroadipose meniscoids present. These joints lie approximately 20° off the frontal plane, which allows greater ROM into lateral flexion and rotation and less ROM into flexion and extension (Fig. 4–16B). The joint capsules are tauter than those of the cervical and lumbar regions, which also contributes to less available motion.

Ligaments

The ligaments associated with the thoracic region are the same as the typical vertebral ligaments described at the beginning of the chapter, with the exception that the ligamentum flavum and anterior longitudinal ligaments are thicker in the thoracic region than in the cervical region.

Atypical Thoracic Vertebrae

The majority of the thoracic vertebrae adhere to the basic structural design of all vertebrae with some minor variations. The 1st and 12th thoracic vertebrae are transitional vertebrae, possessing characteristics of the cervical and lumbar vertebrae, respectively. The first thoracic vertebra has a typical cervical-shaped body with the transverse diameter of the body nearly twice the anteroposterior diameter. The spinous process of T1 is particularly long and prominent. The 12th thoracic vertebra has

thoracic-like superior zygapophyseal articular facets that face posterolaterally. The inferior zygapophyseal facets, however, are more lumbar-like and have convex surfaces that face anterolaterally to articulate with the vertical, concave, posteromedially facing superior zygapophyseal facets of the first lumbar vertebra. Additional differences in T1, T11, and T12 as compared with the other thoracic vertebrae include the presence of full costal facets on these three vertebral bodies rather than demifacets because ribs 1, 11, and 12 articulate only with their corresponding vertebral bodies (rather than with a body above and below).

Function of the Thoracic Region

The thoracic region is less flexible and more stable than the cervical region because of the limitations imposed by structural elements such as the rib cage, spinous processes, taut zygapophyseal joint capsules, the thicker ligamentum flavum, and the dimensions of the discs and the vertebral bodies. Each thoracic vertebra articulates with a set of paired ribs by way of two joints: the **costovertebral** and the **costotransverse joints.** The vertebral components of the costovertebral joints are the demifacets located on most of the vertebral bodies. The vertebral components of the costotransverse joints are the oval facets on the transverse processes. These joints are discussed in detail in Chapter 5.

Kinematics

All motions are possible in the thoracic region, but the range of flexion and extension is extremely limited in the upper thoracic region (T1–T6) because of the rigidity of the rib cage and the zygapophyseal facet orientation in the frontal plane. In the lower part of the thoracic region (T9–T12), the zygapophyseal facets lie closer to the sagittal plane,

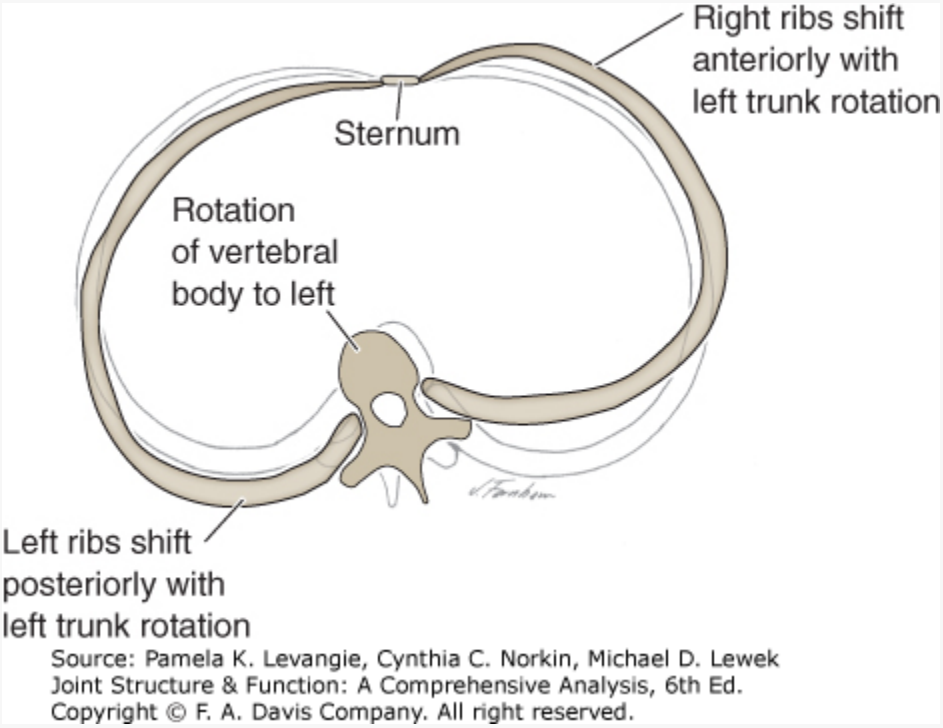
allowing an increasing amount of flexion and extension. Lateral flexion and rotation are free in the upper thoracic region. The ROM in lateral flexion is coupled with some axial rotation. The amount of accompanying axial rotation decreases in the lower part of the region because of the change in orientation of the zygapophyseal facets at T10 or T11. The direction of coupled rotation may vary widely among individuals.[9]

Flexion in the thoracic region is limited by tension in the posterior longitudinal ligament, the ligamentum flavum, the interspinous ligaments, and the capsules of the zygapophyseal joints. Extension of the thoracic region is limited by contact of the spinous processes, laminae, and zygapophyseal facets and by tension in the anterior longitudinal ligament, zygapophyseal joint capsules, and abdominal muscles. Lateral flexion is restricted by impact of the zygapophyseal facets on the concavity of the lateral flexion curve and by limitations imposed by the rib cage. Rotation in the thoracic region also is limited by the rib cage. When a thoracic vertebra rotates, the motion is accompanied by distortion of the associated rib pair (Fig. 4–36). The posterior portion of the rib on the side to which the vertebral body rotates becomes more convex as the anterior portion of the rib becomes flattened. The amount of rotation that is possible depends on the ability of the ribs to undergo distortion and the amount of motion available in the costovertebral and costotransverse joints. As a person ages, the costal cartilages ossify and allow less rib cage distortion. This results in a reduction with aging in the amount of thoracic rotation that is available.

FIGURE 4-36

Rotation of a thoracic vertebral body to the left (rotation of the trunk

to the left) produces a distortion of the associated rib pair that increases the convexity of the ribs posteriorly on the left and anteriorly on the right.



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Kinetics

The thoracic region is subjected to increased compression forces in comparison with the cervical region because of the greater amount of body weight that needs to be supported and the region's kyphotic shape. The line of gravity falls anterior to the thoracic spine. This produces a flexion moment on the thoracic spine that is counteracted by the posterior ligaments and the spinal extensors. The greatest flexion moment is at the peak of the kyphotic curvature as a result of the increased moment arm of the line of gravity.[9]

Foundational Concepts 4–4

Structure and Function of the Thoracic Region

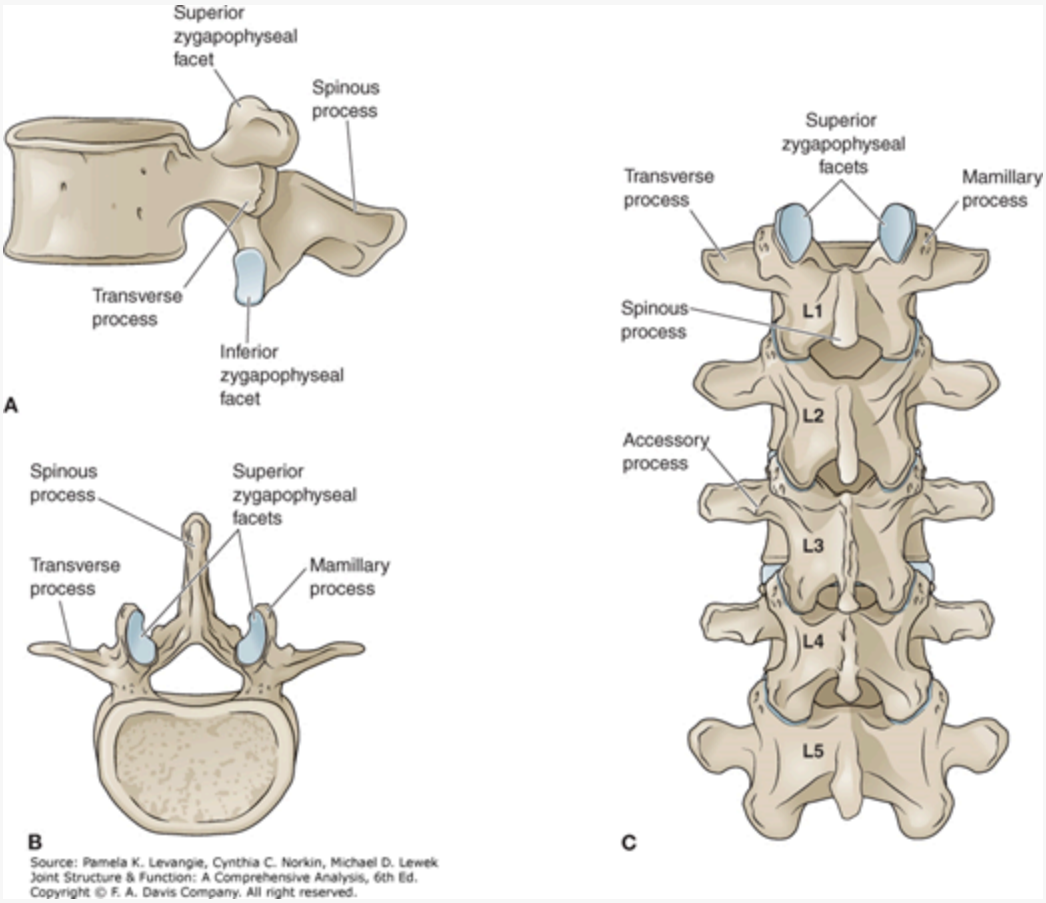
To summarize, the primary goal of the structures of the thorax (thoracic spine and rib cage) is to protect the vital organs and the spinal cord while preserving total trunk ROM. Clinically, diminished segmental mobility of the thoracic spine occurs frequently due to prolonged sitting positions that involve a slouched posture. This may contribute to problems in adjacent spinal regions. Given that the total available motion in the thoracic segments is less than that of the cervical and lumbar regions, loss of even a small amount of thoracic motion may have a functional impact. Retaining functional mobility when there is reduced thoracic mobility may place increased mobility demands on cervical or lumbar segments. This can increase the loads on structures of those regions, contributing to neck or low back pain.

Structure of the Lumbar Region

The first four lumbar vertebrae are similar in structure to each other and share the elements common to all the vertebrae (Fig. 4–37). The fifth lumbar vertebra has structural adaptations for articulation with the sacrum.

FIGURE 4-37

Typical lumbar vertebra. **A.** The lateral view of a typical lumbar vertebra shows the large body as well as the sagittal plane orientation of the superior and inferior zygapophyseal facets. **B.** A superior view shows the triangularly shaped vertebral foramen as well as the transverse and spinous processes. The mamillary processes appear as small, smooth bumps on the posterior edges of each superior facet. **C.** A posterior view of the five lumbar vertebrae.



Typical Lumbar Vertebrae

BODY

The body of the typical lumbar vertebra is massive, with a transverse diameter that is greater than the anteroposterior diameter and greater than the height of the body (Figs. 4–37A and B). The size and shape reflect the need to support great compressive loads caused by body weight, ground reaction forces, and muscle contraction.

ARCHES

PEDICLES

The pedicles are short and thick and project posterolaterally.

LAMINAE

The laminae are short and broad.

ZYGAPOPHYSEAL ARTICULAR PROCESSES

The superior and inferior zygapophyseal facets vary considerably in shape and orientation among individuals. **Mamillary processes,** which appear as small bumps, are located on the posterior edge of each superior zygapophyseal facet (Fig. 4–37B). The mamillary processes serve as attachment sites for the multifidus and medial intertransverse muscles. The inferior zygapophyseal facets are vertical and convex and face slightly anteriorly and laterally.

TRANSVERSE PROCESS

The transverse process is long and slender and extends horizontally (Fig. 4–37B).

SPINOUS PROCESS

The spinous process is broad, thick, rectangular in shape, and extends horizontally (Fig. 4–37A).

VERTEBRAL FORAMEN

The vertebral foramen is triangular and larger than the thoracic vertebral foramen, but smaller than the cervical vertebral foramen.

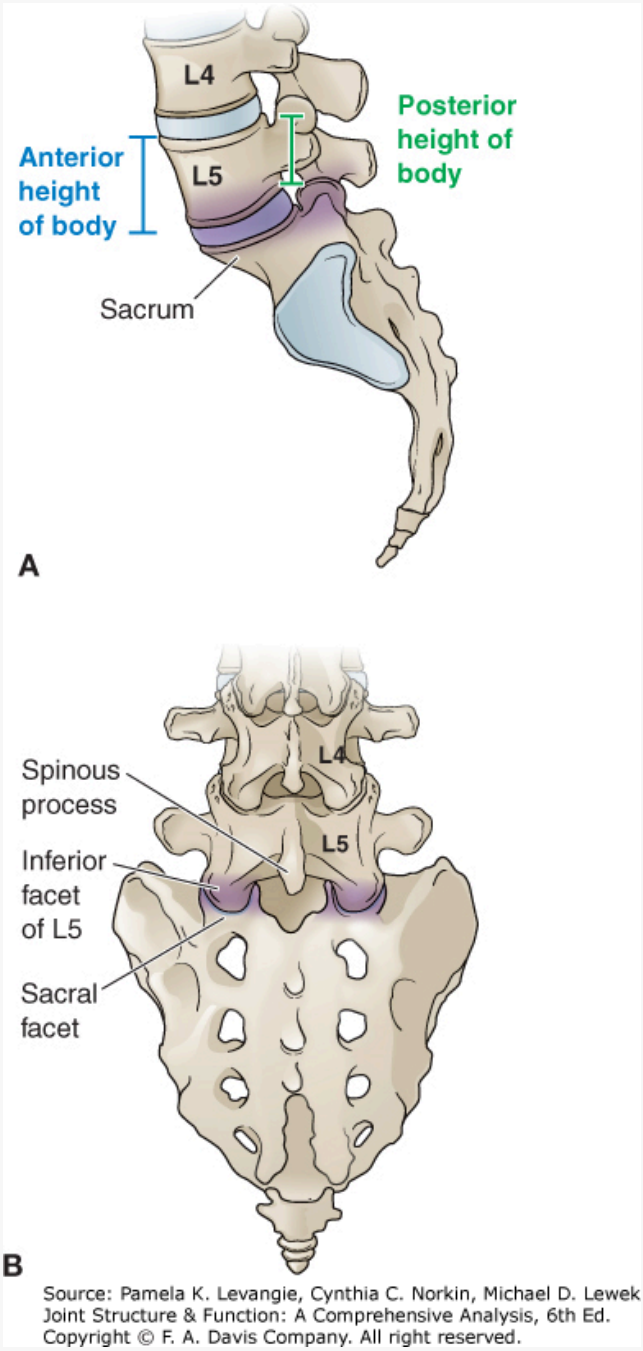
Atypical Lumbar Vertebra

The fifth lumbar vertebra (**Fig. 4–38**) is a transitional vertebra and differs from the rest of the lumbar vertebrae in that it has a wedge-shaped body whose anterior portion is of greater height than the posterior portion (**Fig. 4–38A**). The spinous process is smaller than other lumbar spinous processes, and the transverse processes are large and directed superiorly and posteriorly. The inferior zygapophyseal facets are larger and

wider apart than the other lumbar vertebrae (see Fig. 4–37C), as well as oriented more anteriorly to accommodate the large posteriorly facing sacral facets (Fig. 4–38B).

FIGURE 4-38

The fifth lumbar vertebra is a transitional vertebra. **A.** In this lateral view, it can be seen that the body of L5 is larger anteriorly than posteriorly, making it more wedge-shaped than the other lumbar vertebrae. **B.** A posterior view shows that the spinous process of L5 is shorter than that of the other lumbar vertebrae, and its inferior facets are oriented more anteriorly to articulate with the posteriorly-facing sacral facets.

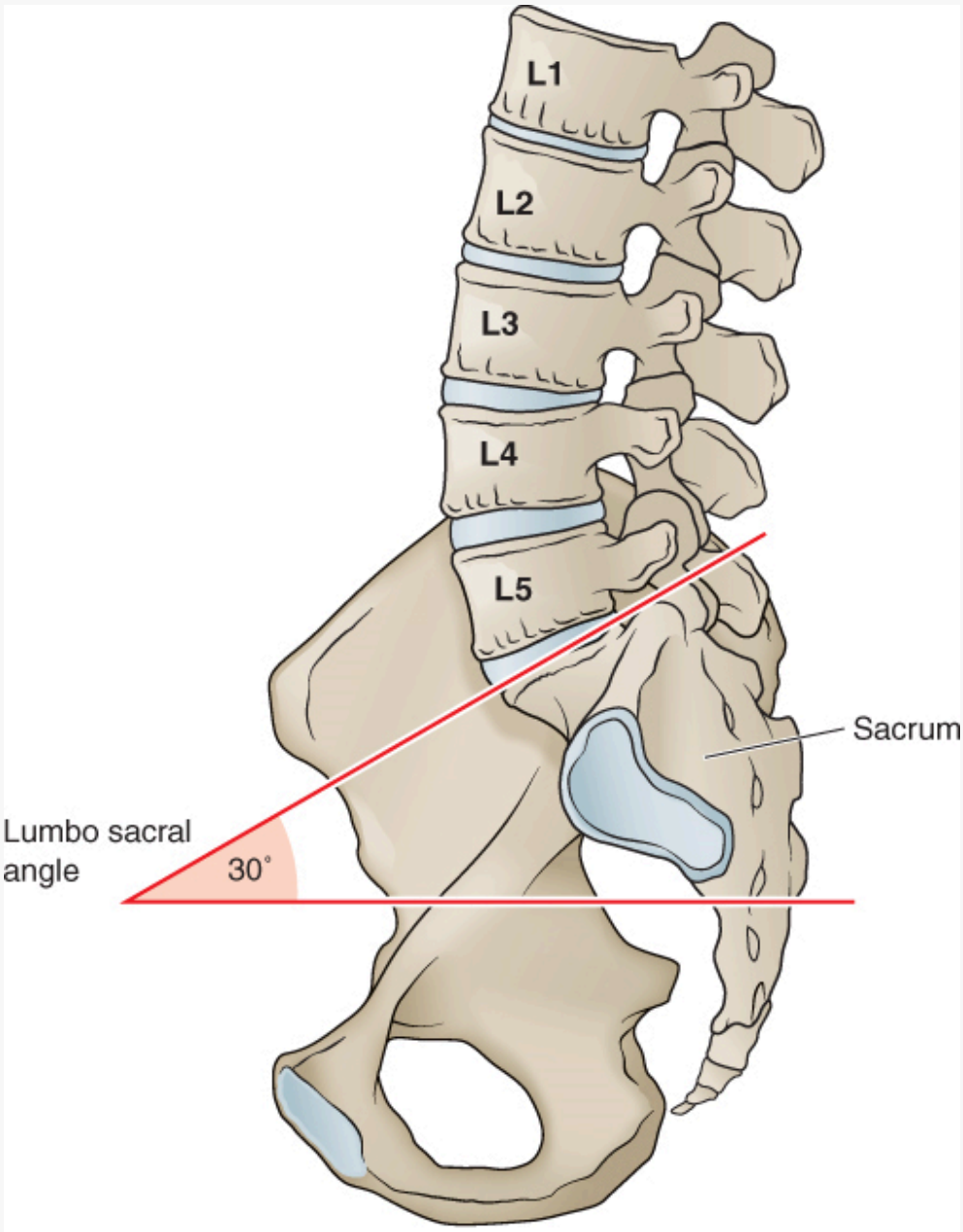


The **lumbosacral articulation** (see Fig. 4–38B) is formed by the fifth lumbar vertebra and first sacral segment, which is inclined anteriorly and inferiorly, forming an angle with the horizontal plane called the **lumbosacral angle** (Fig. 4–39). The size of the angle varies with the position of the pelvis and affects the superimposed lumbar curvature.

An increase in this angle will result in an increase in lordosis of the lumbar curve and will increase the amount of shear stress at the lumbosacral joint (Fig. 4–40).

FIGURE 4-39

The lumbosacral angle is determined by measuring the angle formed by a line drawn parallel to the superior aspect of the sacrum and a horizontal line. The right innominate has been removed to expose the sacrum in this lateral view.

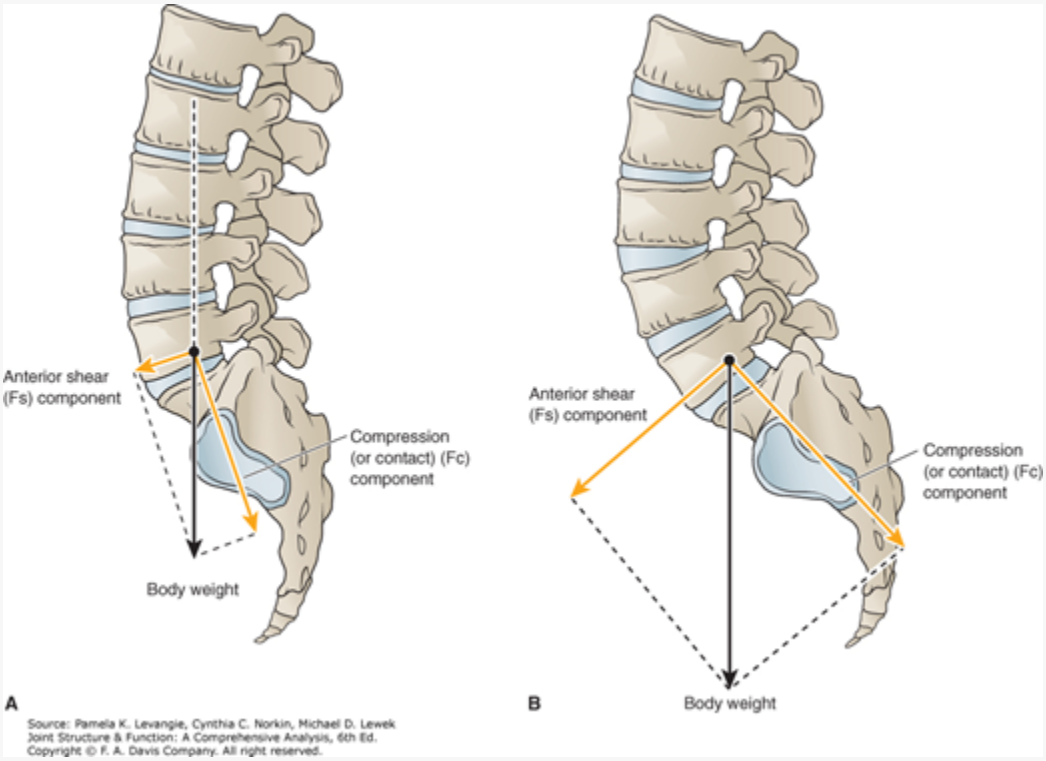


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FIGURE 4-40

Shear stresses at the lumbosacral joint. **A.** Anterior shear typical of a lumbosacral angle of 30°. Body weight acting on L5 results in L5

subjected to both a compressive force (Fc) and an anterior shear force (Fs) in relation to the inclined surface of S1. B. With an increased lumbosacral angle of 45°, the force of body weight acting on L5 results in a shear force (Fs) that is equal to or greater than the compressive force (Fc) in relation to the inclined surface of S1.



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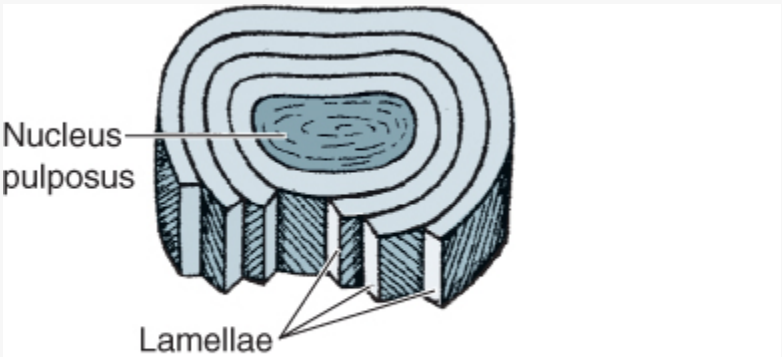
Intervertebral Discs

Specific regional variations occur in the intervertebral discs of the lumbar region, which differ from the discs of the cervical region in that the collagen fibers of the annulus fibrosus are arranged in sheets called **lamellae** (Fig. 4–41). The lamellae are arranged in concentric rings that surround the nucleus. Collagen fibers in adjacent rings are oriented in opposite directions at 120° to each other.[7] The advantage of the varying fiber orientation by layer is that the annulus fibrosus is able to resist tensile forces in

nearly all directions. The lumbar intervertebral discs are the largest in the body (as are the lumbar vertebral bodies). The shape of each disc is not purely elliptical but is concave posteriorly (Fig. 4–42). This provides a greater cross-sectional area of annulus fibrosus posteriorly and thus increases the ability to resist the tension that occurs here with forward bending.

FIGURE 4-41

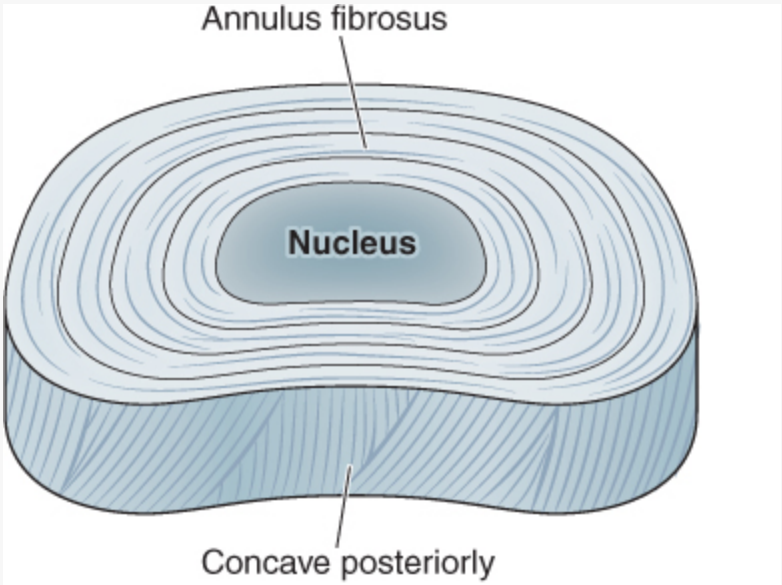
Lamellae (layers) in the annulus fibrosus of lumbar intervertebral discs shift fiber orientations from layer to layer.



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FIGURE 4-42

Lumbar intervertebral discs are concave posteriorly, providing a greater cross-section posteriorly. This means more annulus fibrosus is available to resist the posterior stretch that occurs in lumbar flexion.



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Articulations

INTERBODY JOINTS

The interbody joints of the lumbar region are capable of translations and tilts in all directions.

ZYGAPOPHYSEAL JOINTS

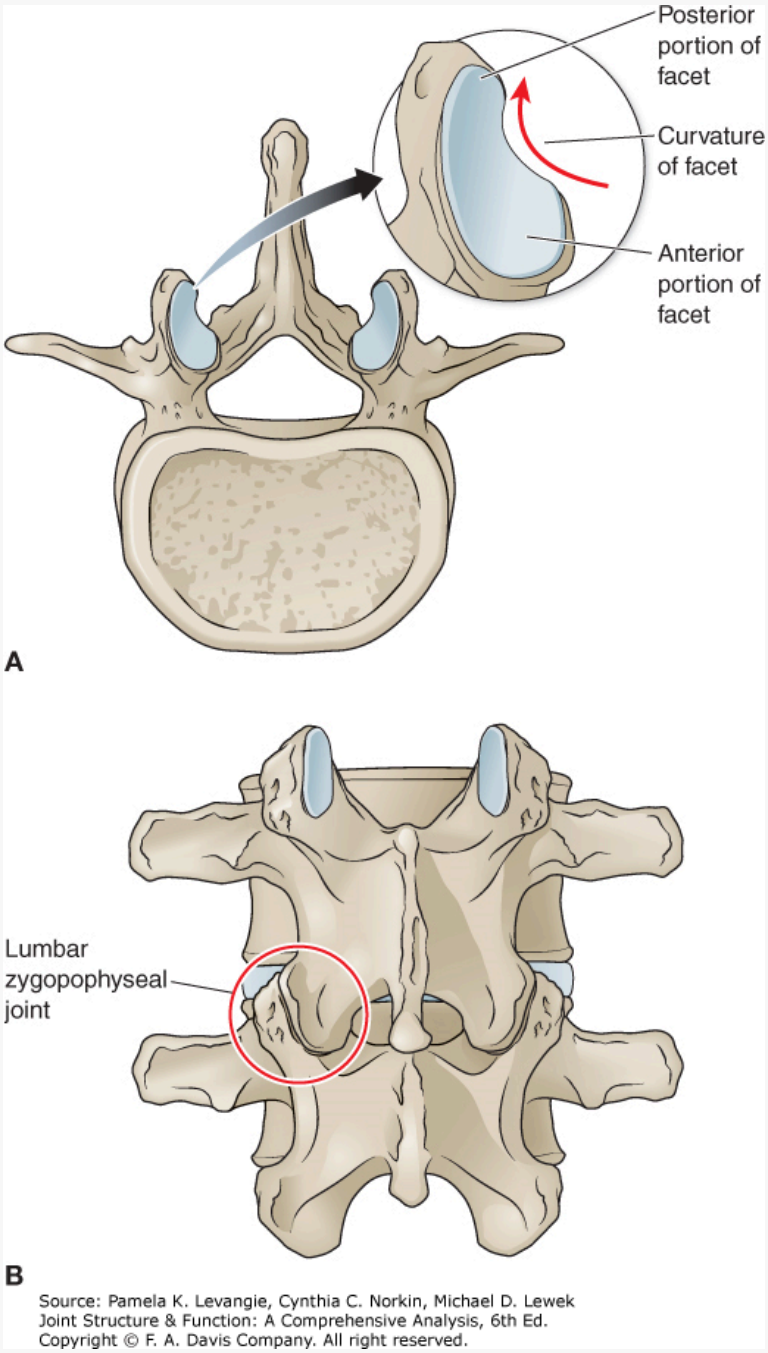
The zygapophyseal joints of the lumbar region, as in other regions, are true synovial joints and contain fibroadipose meniscoid structures. The joint capsules are more lax than those in the thoracic region but tauter than those in the cervical region. The posterior capsule has been demonstrated to be fibrocartilaginous, which suggests that this portion of the capsule is subject to compressive as well as tensile forces.[44]

In a newborn, the zygapophyseal joints in the lumbar region lie predominantly in the frontal plane with a lumbar kyphosis. As the child develops and assumes an upright posture, the curve of the lumbar region changes to lordosis and the orientation of the

zygapophyseal joints changes as well. The orientations of the adult lumbar zygapophyseal joints demonstrate great variability both between individuals and within individuals; however, the majority of the lumbar zygapophyseal joints have a curved structure that is biplanar in orientation (Fig. 4–43).[2] The anterior aspect of each joint remains in the frontal plane, whereas the posterior aspect is oriented close to or in the sagittal plane (Fig. 4–43A). The degrees to which this happens vary. The frontal plane orientation of the anterior portion of the facets provides resistance to the anterior shear that is present in the lordotic lumbar region. The sagittal plane orientation of the posterior portion of the facet is evident in a posterior view of the lumbar spine (Fig. 4–43B), allowing a great range of flexion and extension motion and providing resistance to rotation.

FIGURE 4-43

A. In this superior view, the biplanar orientation of the lumbar zygapophyseal joints is evident. The anterior portion of the facet is oriented more in the frontal plane whereas the posterior portion is oriented in the sagittal plan. **B.** A posterior view of the lumbar vertebrae that shows only the posterior aspect of the zygapophyseal joints appears to show an exclusively sagittal plane orientation to the joints.



Ligaments and Fascia

The majority of the ligaments associated with the lumbar region are the same ligaments described previously (ligamentum flavum, posterior longitudinal ligament, anterior longitudinal ligament, interspinous and supraspinous ligaments, and joint

capsules). However, a few of these ligaments have variations specific to the lumbar region and need to be mentioned here before structures unique to the lumbar region are introduced.

The supraspinous ligament is well developed only in the upper lumbar region and may terminate at L3, although the most common termination site appears to be at L4. The supraspinous ligament is almost always absent at S1, and its deep layer is reinforced by tendinous fibers of the multifidus muscle. The middle fibers of the supraspinous ligament blend with the dorsal layer of the thoracolumbar fascia. The intertransverse ligaments are not true ligaments in the lumbar area and are replaced by the iliolumbar ligament at L5. The posterior longitudinal ligament is only a thin ribbon in the lumbar region, whereas the ligamentum flavum is thickened here. The anterior longitudinal ligament is strong and well developed in the lumbar region.

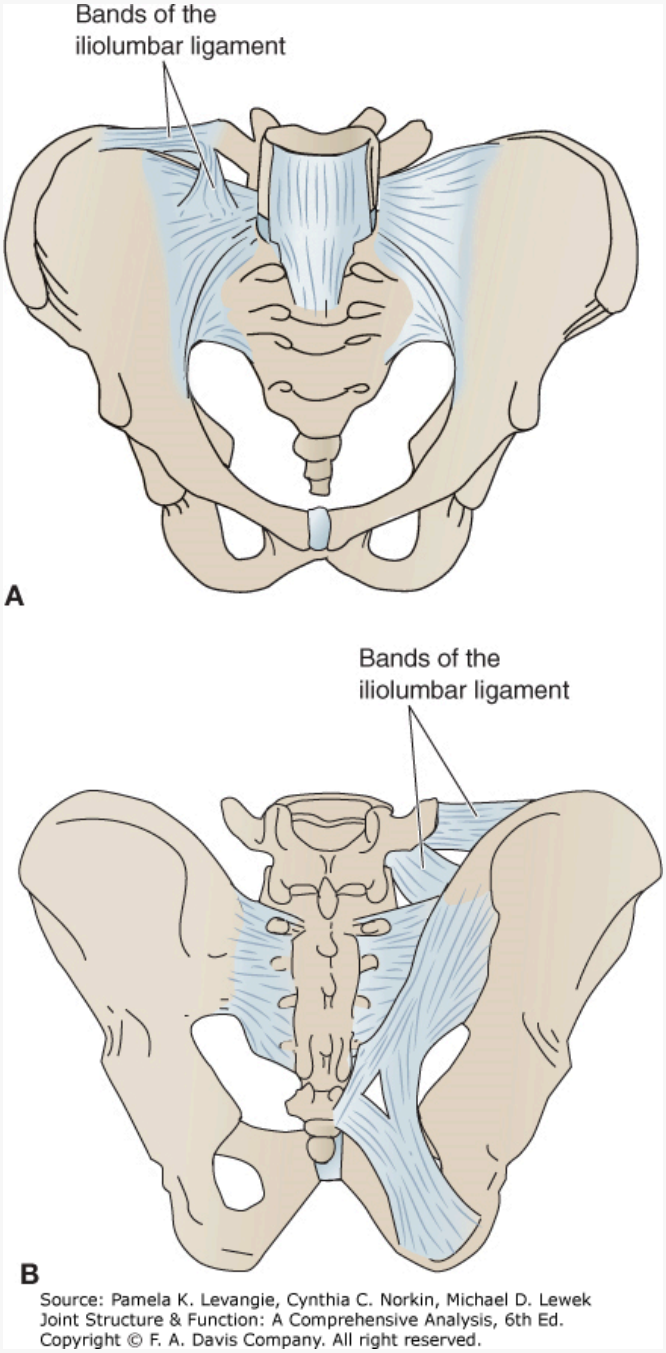
ILIOLUMBAR LIGAMENTS

The **iliolumbar ligaments** (Fig. 4–44) consist of two bands that run from the L5 transverse process to the iliac tuberosity with connections to the quadratus lumborum and sacroiliac ligaments.[45] The iliolumbar ligaments as a whole are very strong and play a significant role in stabilizing the fifth lumbar vertebra. They prevent anterior displacement on L5 that might result from anterior shear forces found here and resist flexion, extension, axial rotation, and lateral bending of L5 on S1. The iliolumbar ligaments are maximally loaded at the lumbosacral junction in full lumbar flexion without protection from spinal extensor muscle, for instance, when sitting in a relaxed, slouched posture. These ligaments can easily be a source of low back pain when subjected to creep in the slouched position.[46]

FIGURE 4-44

The two bands of the bilateral iliolumbar ligaments. **A.** Anterior view.

B. Posterior view.



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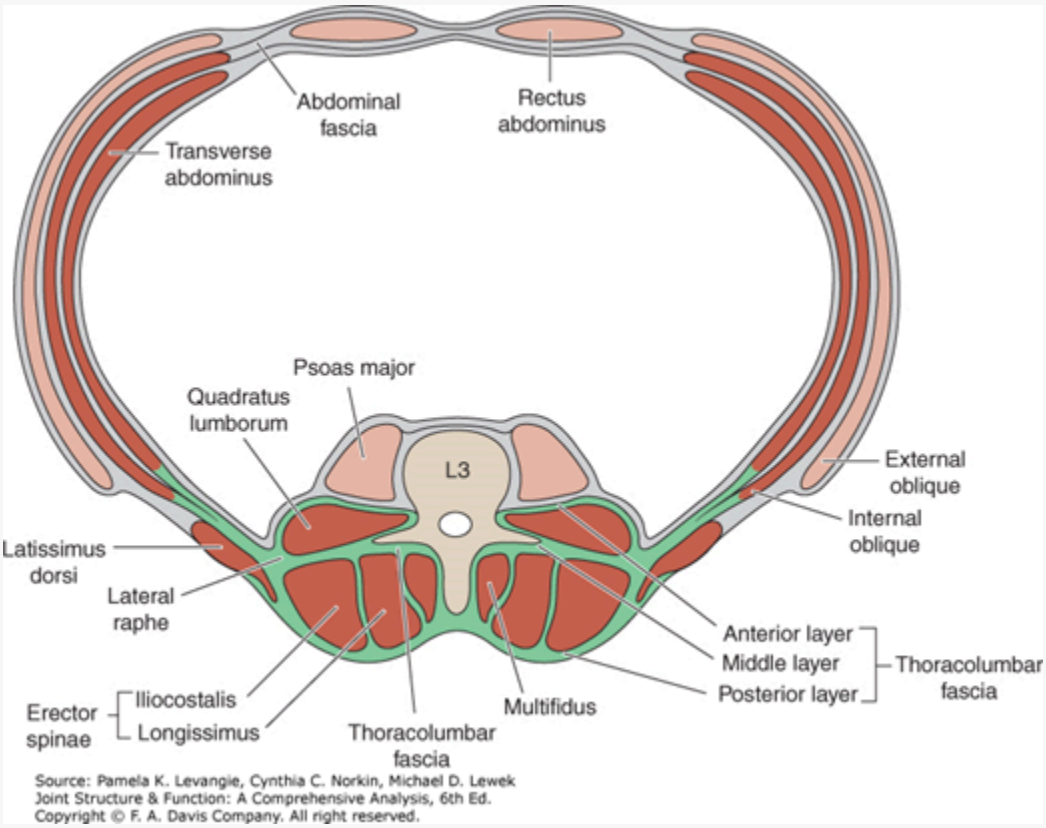
THORACOLUMBAR FASCIA

The thoracolumbar fascia (also called the lumbodorsal fascia) consists of posterior, middle, and anterior layers (Fig. 4–45). The posterior layer is large, thick, and fibrous and arises from the spinous processes and supraspinous ligaments of the thoracic, lumbar, and sacral spines. The posterior layer gives rise to the superiorly located latissimus dorsi and is continuous with some fibers of the splenius capitis and cervicis.[47] The posterior layer travels caudally to the sacrum and ilium and blends with the fascia of the gluteus maximus. Deep fibers are continuous with the sacrotuberous ligament and are connected to the posterior superior iliac spines, iliac crests, and long posterior sacroiliac ligament.[47] The posterior layer travels laterally over the erector spinae muscles and forms the lateral raphe at the lateral aspect of the erector spinae. The internal abdominal oblique and the transversus abdominal muscles arise from the lateral raphe. The posterior layer becomes the middle layer as it travels medially along the anterior surface of the erector spinae and attaches back to the transverse processes and intertransverse ligaments of the lumbar spine. The middle and posterior layers completely surround the lumbar extensor muscle group. The anterior layer of the thoracolumbar fascia is derived from the fascia of the quadratus lumborum muscle, where it joins the middle layer, inserts into the transverse processes of the lumbar spine, and blends with the intertransverse ligaments.[47]

FIGURE 4-45

A superior view of a cross-section to identify the abdominal hoop. The anterior, middle, and posterior layers of the thoracolumbar fascia, along with the abdominal fascia, are the passive parts. The muscles

are the active parts that pull on the fascia to tighten the hoop.



The thoracolumbar fascia, when combined with its indirect connections to the abdominal fascia, form a hoop around the abdomen (as shown in Fig. 4–45) that adds stiffness to the lumbopelvic region.[48] Muscles that attach to the fascia can increase fascial tension with an active contraction, helping to further increase spinal stiffness and stabilize the lumbar spine. For example, contraction of the gluteus maximus and contralateral latissimus dorsi tense the superficial layer of the thoracolumbar fascia and provide a pathway for the mechanical transmission of forces between the pelvis and trunk.[47]

Function of the Lumbar Region

Kinematics

The lumbar region is capable of movement in flexion, extension, lateral flexion, and rotation. The lumbar zygapophyseal facets favor flexion and extension because of the predominant sagittal plane orientation (Fig. 4–43B). The amount of flexion varies at each interspace, but the greatest amount of flexion takes place at the lumbosacral (L5–S1) joint. Lateral flexion and rotation are most free in the upper lumbar region and progressively diminish in the lower region.

As shown in Figure 4–46, lumbar flexion involves anterior tilt and anterior slide of the vertebral body above on the vertebral body below and upslide of the inferior zygapophyseal facet on the facet below (Fig. 4–46A). Lumbar extension involves posterior tilt and posterior slide at the interbody joints and downslide of the inferior zygapophyseal facet on the facet below (Fig. 4–46B). As shown in Figure 4–47, lumbar lateral flexion involves tilt and slide to the ipsilateral side of the bend at the interbody joints and ipsilateral downslide combined with contralateral upslide of the inferior zygapophyseal facets (Fig. 4–47A). Lumbar rotation, however, has a different motion in the lumbar region due to the shape of the zygapophyseal joints. The interbody joints tilt and translate to the ipsilateral side (Fig. 4–47B), but the zygapophyseal joints have less sliding motion than at other regions. In rotation, the ipsilateral zygapophyseal joint distracts and the contralateral zygapophyseal joint compresses.

FIGURE 4-46

Flexion and extension of the lumbar vertebrae take place as

movements of a cephalad vertebra on the vertebra below. **A.** In flexion, the vertebral body tilts and slides anteriorly while the inferior facets slide upwardly on the facets below. **B.** In extension, the vertebral body tilts and slides posteriorly while the inferior facets slide downwardly on the facets below.

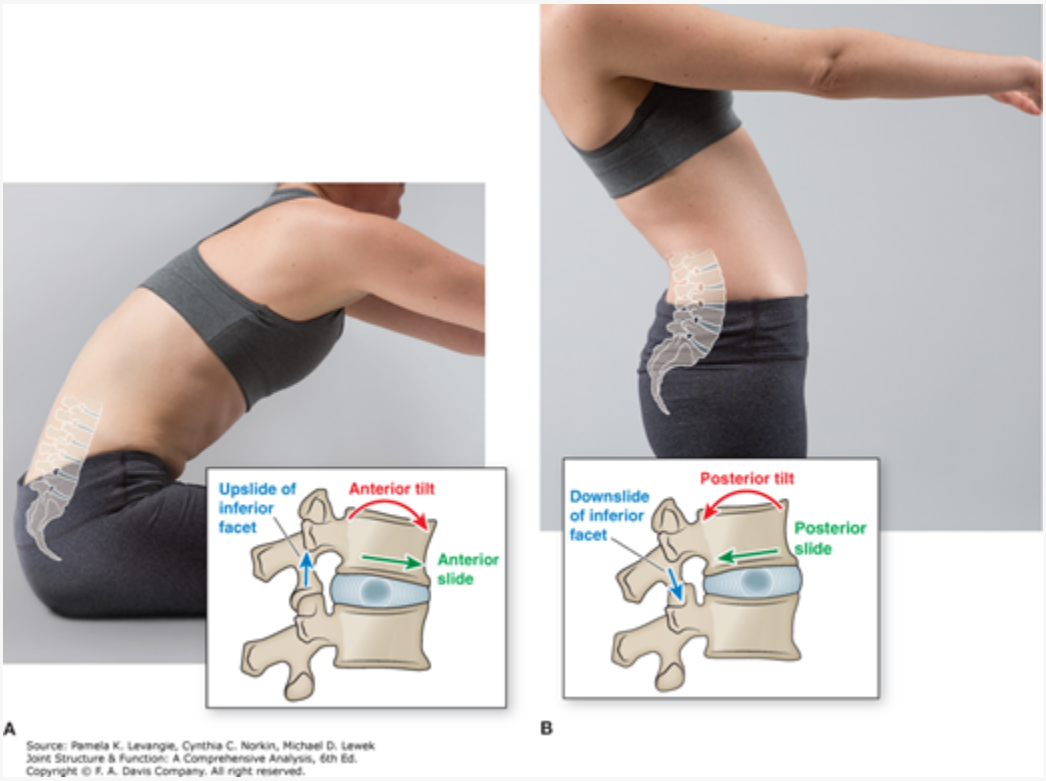
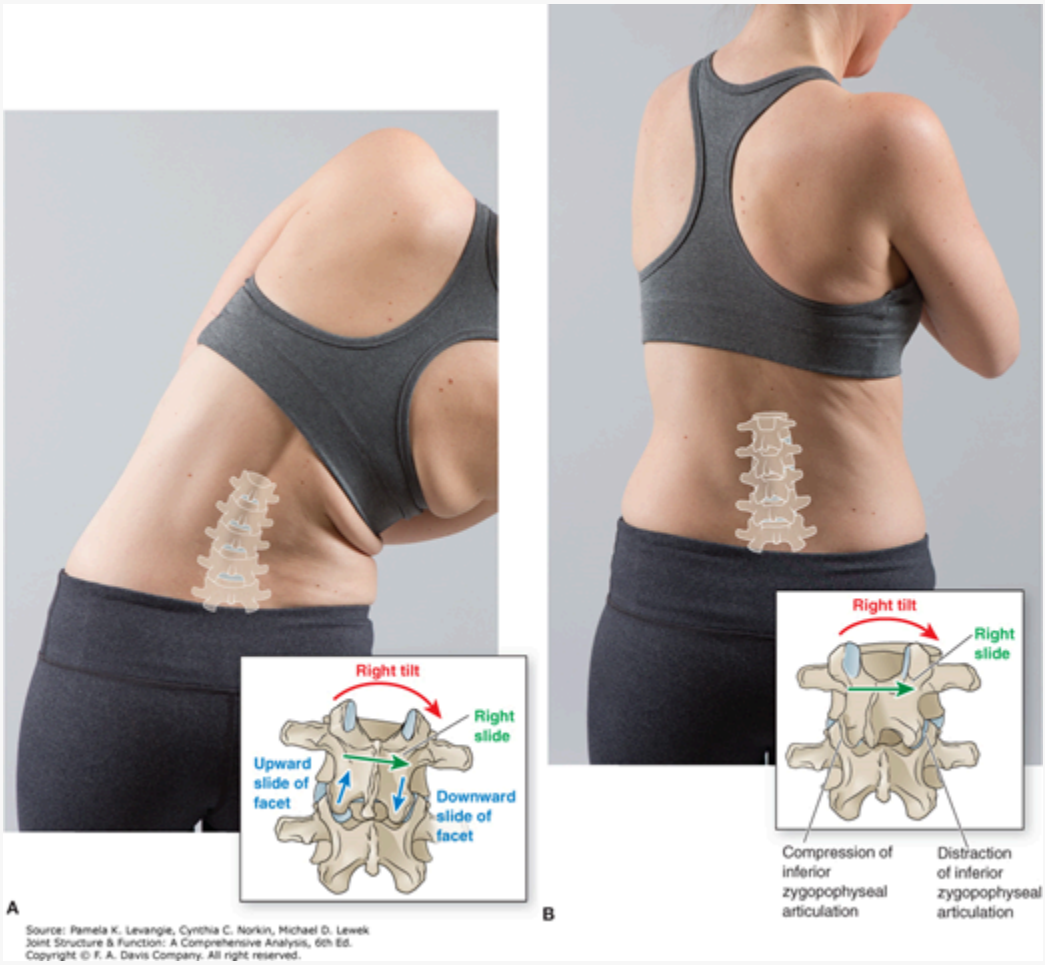


FIGURE 4-47

Lateral flexion and rotation of the lumbar vertebrae, although limited in range of motion, take place as movements of a cephalad vertebra on the adjacent vertebra below. **A.** In right lateral flexion, the vertebral body (and its associated posterior elements) tilts and slides to the

right while the left inferior facet slides upward and the right inferior facet slides downward on the facets below. **B.** In rotation of the lumbar spine to the right, the vertebral body tilts and slides to the right while the right inferior facet joint distracts (opens) and the left inferior facet joint compresses.



The amount of lumbar motion from a neutral lordotic position is reported to be as follows: flexion: 52° (SD = 9°); extension: 19° (SD = 9°); lateral flexion: 30° to each side (SD = 6°); and rotation: 32° to each side (+/–12°).[49]

In the lumbar region, coupled motions usually occur with lateral flexion and axial rotation. The pattern of coupled motion, however, appears to be inconsistent. Legaspi and Edmond performed a review of the literature to determine if a consistent pattern of coupling was demonstrated in published research. Of the 24 articles reviewed, there was little agreement about the specific characteristics of coupled motion in the lumbar spine.[21]

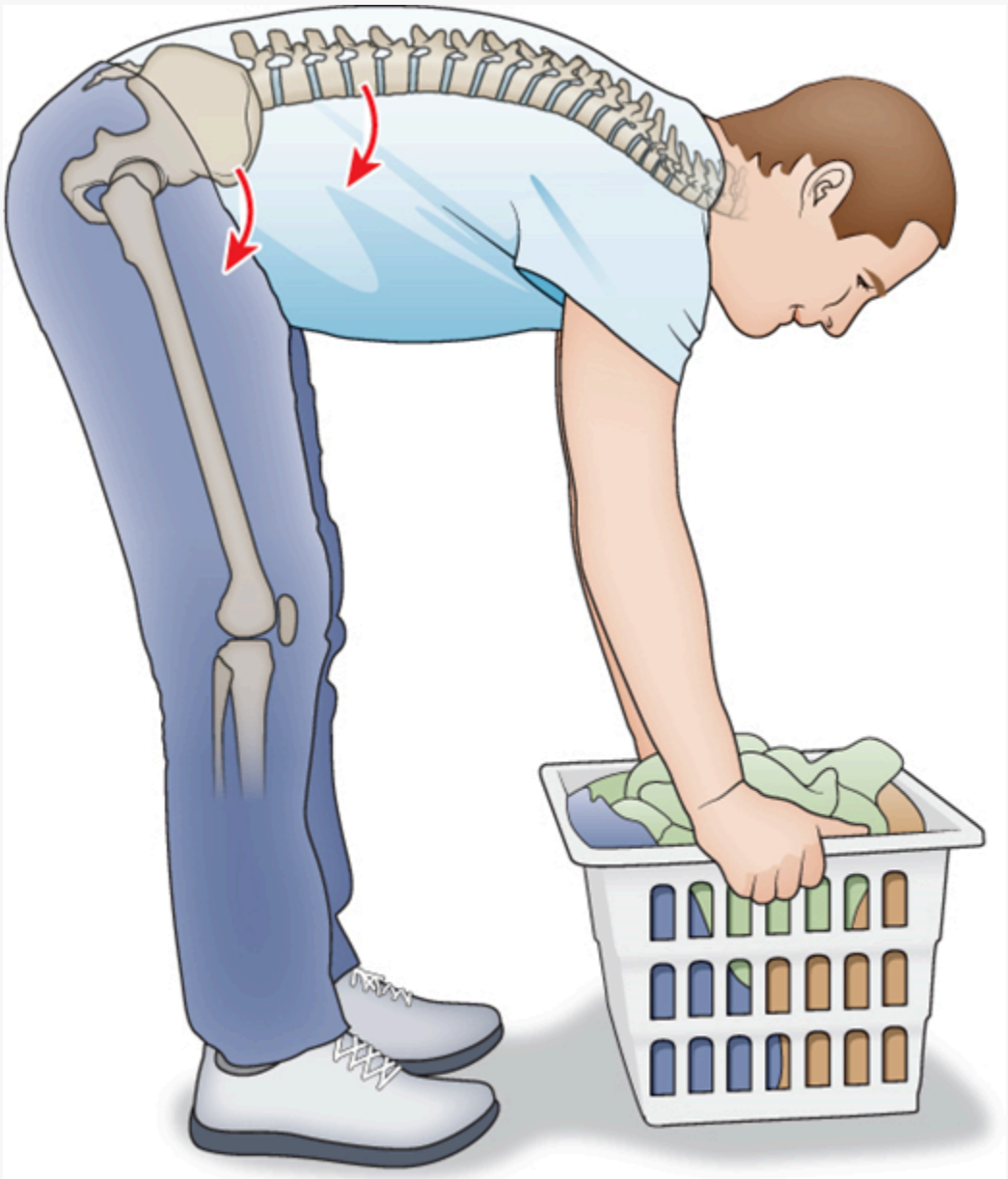
Lumbopelvic Rhythm

Full trunk motion, particularly in forward bending, is accomplished in part by a combination of motions at the lumbar spine and the pelvis (including the sacroiliac and hip joints; Fig. 4–48). The ratio of contribution from different areas is identified as **lumbopelvic rhythm.** In lumbopelvic rhythm, the goal is to maximize movement of the head and arms through space (open chain) rather than to keep the head upright and vertically oriented (closed chain). Lumbopelvic rhythm was first described by Calliet[50] as a *sequential* contribution from the lumbar spine first, then from the pelvis. This sequential pattern has since been questioned.[14] A recent study examined the lumbopelvic rhythm during trunk flexion and extension in asymptomatic individuals.[51] The motions of the lumbar and pelvis were found to be simultaneous rather than sequential. Further, it was determined that this simultaneous movement pattern yielded near optimal L5–S1 compression forces.

FIGURE 4-48

Lumbar/pelvic rhythm. The lumbar spine flexes (lordotic curve eliminated) and the pelvis rotates anteriorly in the sagittal plane to

increase the range of forward flexion.



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Kinetics

COMPRESSION

One of the primary functions of the lumbar region is to provide support for the weight

of the upper part of the body in static as well as dynamic situations. The increased size of the lumbar vertebral bodies and discs in comparison with their counterparts in the other spinal regions helps the lumbar structures support the additional weight. The lumbar region must also withstand the tremendous compressive loads produced by muscle contractions. The lumbar interbody joints bear approximately 80% of compressive forces while the lumbar zygapophyseal joints bear approximately 20%. This percentage can change with altered mechanics. For example, with increased lordosis or in the presence of intervertebral disc degeneration, the zygapophyseal joints will assume more of the compressive load.

Lumbosacral loads (ground reaction forces plus forces generated by erector spinae and rectus abdominis muscle groups) in the erect standing posture are about equal to one's body weight. However, compressive loads during walking on level ground are nearly twice the body's weight.[52] Changes in the position of the body will change the location of the body's line of gravity and thus change the forces acting on the lumbar spine.

SHEAR

In the upright standing position, the lumbar segments are subjected to anterior shear forces caused by the lordotic position, body weight, and ground reaction forces (see Fig. 4–40). This anterior shear or translation of the vertebra is resisted by the direct impact of the inferior zygapophyseal facets of the superior vertebra against the superior zygapophyseal facets of the subjacent (caudal) vertebra as well as by the intervertebral disc, posterior ligaments, and deep erector spinae muscles.

Shear loads at the L4–L5 mobile segment were examined in a variety of conditions to determine the load bearing responses by the disc and zygapophyseal joints.[53] With

lower shear loads, the zygapophyseal joints bear 65% of the load. With increasing shear loads, the disc plays a more significant role.



Expanded Concepts 4–2

Variations in Zygapophyseal Joints

The zygapophyseal joints, the zygapophyseal joint capsules, the fibers of the annulus fibrosus, and the iliolumbar ligaments (at L5-S1) provide structural resistance to anterior shear forces in the lumbar segments. Individual variation in joint structure, therefore, can be a contributing factor to pain in this region. If an individual has zygapophyseal joints oriented totally in the sagittal plane, there is little to no bony restraint to the anterior shear forces. In this case, the capsuloligamentous structures will be taxed and eventually the capsuloligamentous structures may become lengthened and a source of pain. Even if an individual has zygapophyseal joints with a biplanar orientation, excessive anterior shear forces can cause damage. In this case, in addition to the lengthened capsuloligamentous structures, the zygapophyseal joints themselves can experience excessive compression in the anterior regions and produce pain. Fortunately, the deep erector spinae muscles provide a dynamic restraint to anterior shear that will be discussed later in the chapter.



Foundational Concepts 4–5

Structure and Function of the Lumbar Region

To summarize, the primary goals of the lumbar region are to augment mobility at the hip joints to increase total mobility of the trunk and to effectively absorb and adjust body weight forces from head, arms, and trunk above and ground reaction forces below in weightbearing. Flexion and extension are the predominant motions available in the lumbar region, allowing us to reach our hands to the floor and overhead while also adjusting to keep the line of gravity of the body over our base of support.

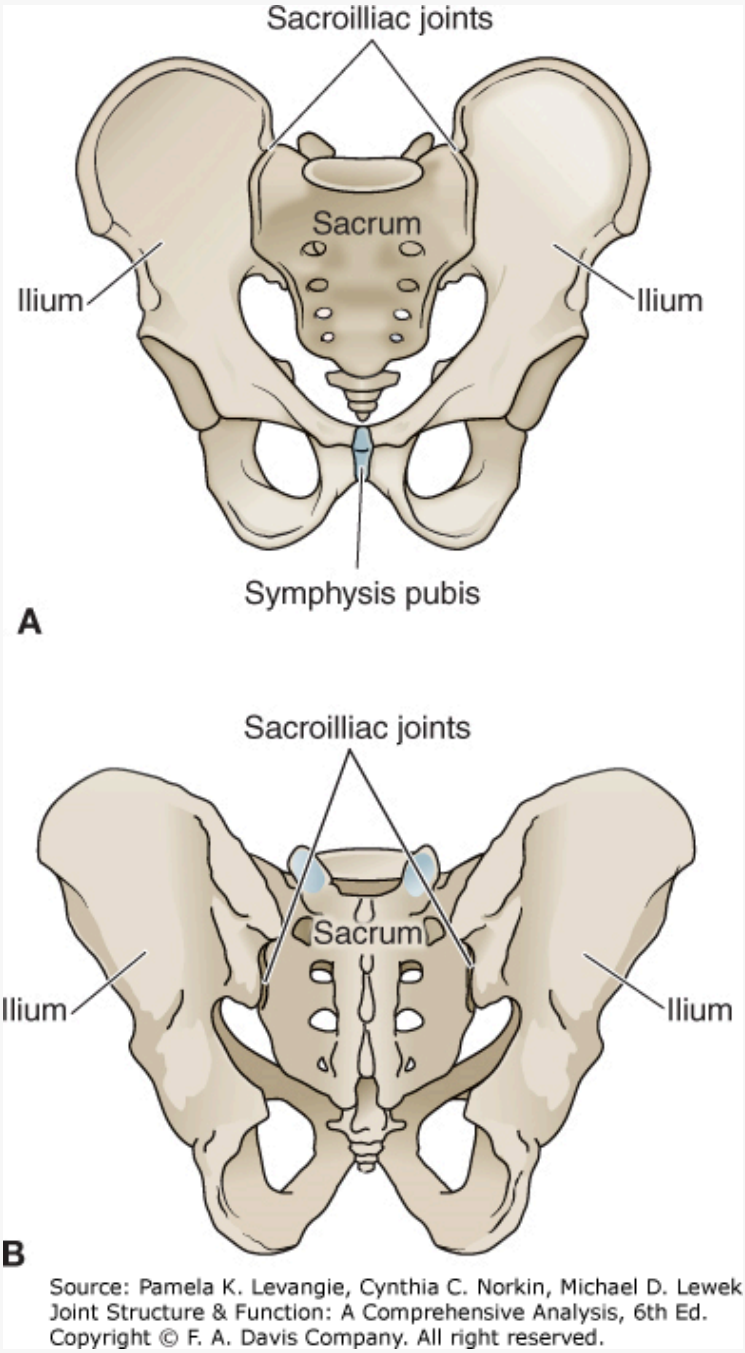
Structure of the Sacral Region

Five sacral vertebrae are fused to form the triangular or wedge-shaped sacrum (Fig.

4–49). The base of the inverted triangle is the first sacral vertebra and supports two zygapophyseal facets that face posteriorly for articulation with the inferior facets of the fifth lumbar vertebra. The apex of the triangle, formed by the fifth sacral vertebra, articulates with the coccyx. The two lateral parts of the sacrum, or ala, each bear an auricular (ear-shaped) surface that articulates with the corresponding iliac portion of the innominate bone (Fig. 4–50). The **innominate bone** (or innominate) refers to one side of the pelvis formed by the consolidation of the ilium, ischium, and pubis. Each ala of the sacrum also bears the sacral tuberosity that is posterior to the auricular surface and articulates with the iliac tuberosity.

FIGURE 4-49

The sacroiliac joints consist of the articulations between the first three sacral segments and the two ilia of the pelvis. **A.** Anterior view. **B.** Posterior view.

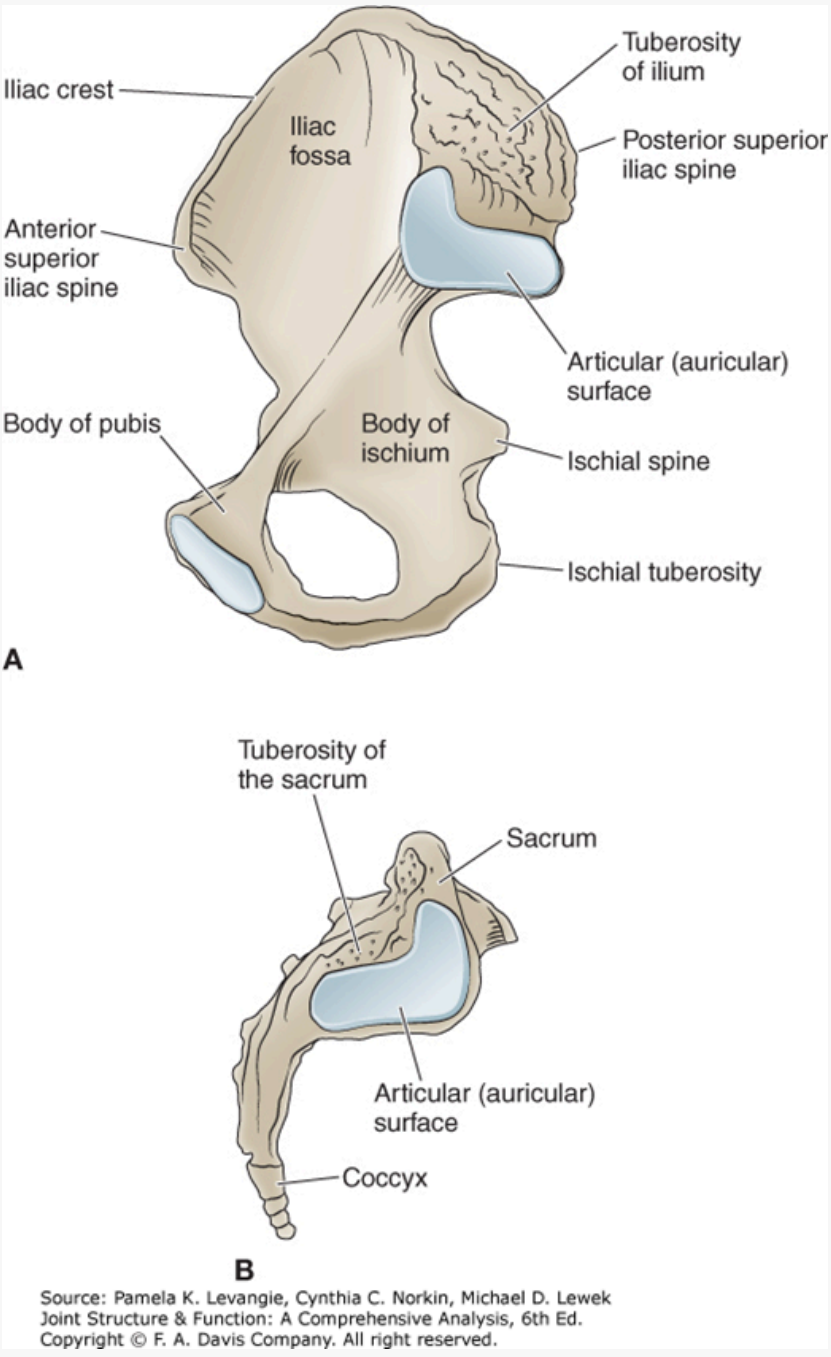


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FIGURE 4-50

Articulating surfaces of the sacroiliac joint. **A.** Lateral view of sacrum.

B. Medial view of the ilium.



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Sacroiliac Articulations

The **sacroiliac joints** are weightbearing compound joints consisting of an anterior synovial joint between the auricular surfaces of the sacrum and ilium and a posterior syndesmosis between the tuberosities of the same bones (Fig. 4–50). The role of the

sacroiliac joints appears to be to relieve the stress on the pelvic ring imparted by movement of the trunk and lower limbs, muscle contractions, the weight of the body, and ground reaction forces. The joints must allow enough movement to help dissipate forces that are ascending from the lower limb and ground and yet be stable enough to transmit the forces from the vertebral column descending to the lower limbs.[2]

ARTICULATING SURFACES ON THE ILIA

The auricular articulating surfaces on the ilia (**Fig. 4–50A**) are covered with hyaline cartilage, although the hyaline of the ilia contains a denser aggregate of collagen fibers than that of the sacrum and is less than 1 mm thick.[54,55] The articular surfaces of the ilia are multiplanar and curvy. The fetal and prepubertal articular faces of the ilia are flat and smooth, whereas the postpuberal surfaces develop grooves and depressions extending the length of the articulating surface that correspond to the grooves on the sacral articulating surfaces.[54] The tuberosities of the ilia are irregular bony surfaces posterior and superior to the auricular surfaces. The ilial tuberosities articulate with the tuberosities of the sacrum via the sacroiliac interosseous ligaments.

ARTICULATING SURFACES ON THE SACRUM

The auricular (ear-shaped) articulating surfaces on the sacrum are located on the sides of the fused sacral vertebrae lateral to the sacral foramina from S1 to S3 (**Fig. 4–50B**). These articulating surfaces, like those of the corresponding iliac surfaces, are multiplanar and curvy and show variations over time from flat and smooth in the young to quite rough past puberty.[54] The articular surfaces are covered with hyaline cartilage that is thicker on the sacral surfaces than the iliac surfaces. The tuberosities of the sacrum are, like their iliac counterparts, irregular bony surfaces posterior to the auricular surfaces that articulate with the tuberosities of the ilium via the sacroiliac

interosseous ligaments.

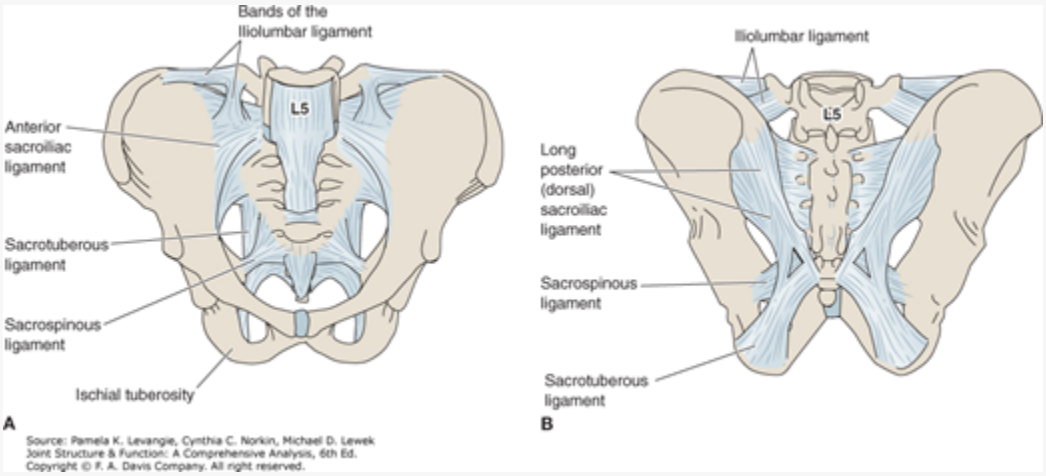
SACROILIAC LIGAMENTS

The anterior, interosseous, and long posterior sacroiliac ligaments are associated directly with the sacroiliac joints. The sacroiliac ligaments are reinforced by fibrous expansions from the quadratus lumborum, erector spinae, gluteus maximus, gluteus minimus, piriformis, and iliacus muscles, which contribute to the joints' stability. The fascial support is greater posteriorly than anteriorly because more muscles are located posteriorly. The sacroiliac ligaments are some of the strongest ligaments in the body.

The **anterior sacroiliac ligaments** are considered to be capsular ligaments because of their intimate connections to the anteroinferior margins of the joint capsules (Fig. 4–51). These ligaments cover the anterior aspects of the sacroiliac joints and join the ilia to the sacrum (Fig. 4–51A).

FIGURE 4-51

The sacroiliac ligaments. **A.** Anterior view. **B.** Posterior view.



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The **long posterior sacroiliac ligaments** (Fig. 4–51B) have superior attachments to the

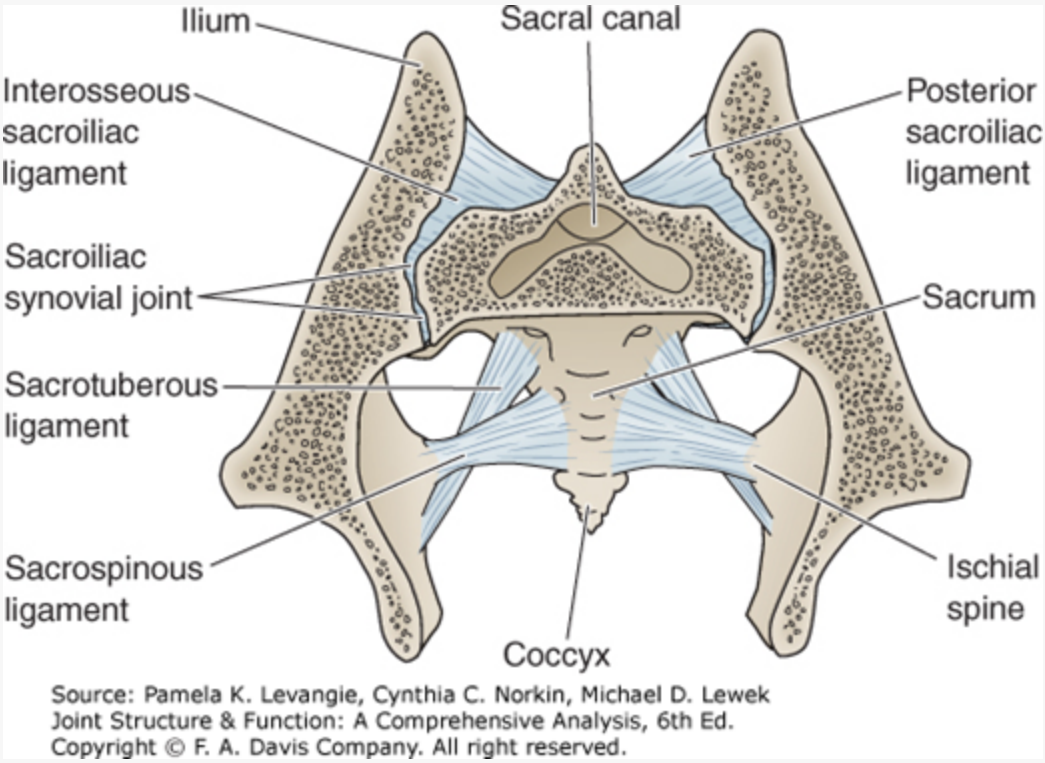
posterior superior iliac spines and adjacent parts of the ilium. Inferiorly, the ligaments are attached to the lateral crest of the third and fourth sacral segments. The medial fibers are connected to the deep lamina of the posterior layer of the thoracolumbar fascia and the aponeurosis of the erector spinae.[56]

The **sacrospinous ligaments** connect the ischial spines to the lateral borders of the sacrum and coccyx; the **sacrotuberous ligaments** connect the ischial tuberosities to the posterior spines at the ilia and the lateral sacrum and coccyx (Fig. 4–51B). The sacrospinous ligament forms the inferior border of the greater sciatic notch; the sacrotuberous ligament forms the inferior border of the lesser sciatic notch.

The **interosseous sacroiliac ligaments** (Fig. 4–52) that constitute the major bonds between the sacrum and the ilia are considered the most important ligaments directly associated with the sacroiliac joints. These ligaments create the more posteriorly located syndesmoses (fibrous union) between the tuberosities of the sacrum and ilia.

FIGURE 4-52

The sacroiliac ligaments in a superior view of a transverse section of the upper pelvis.



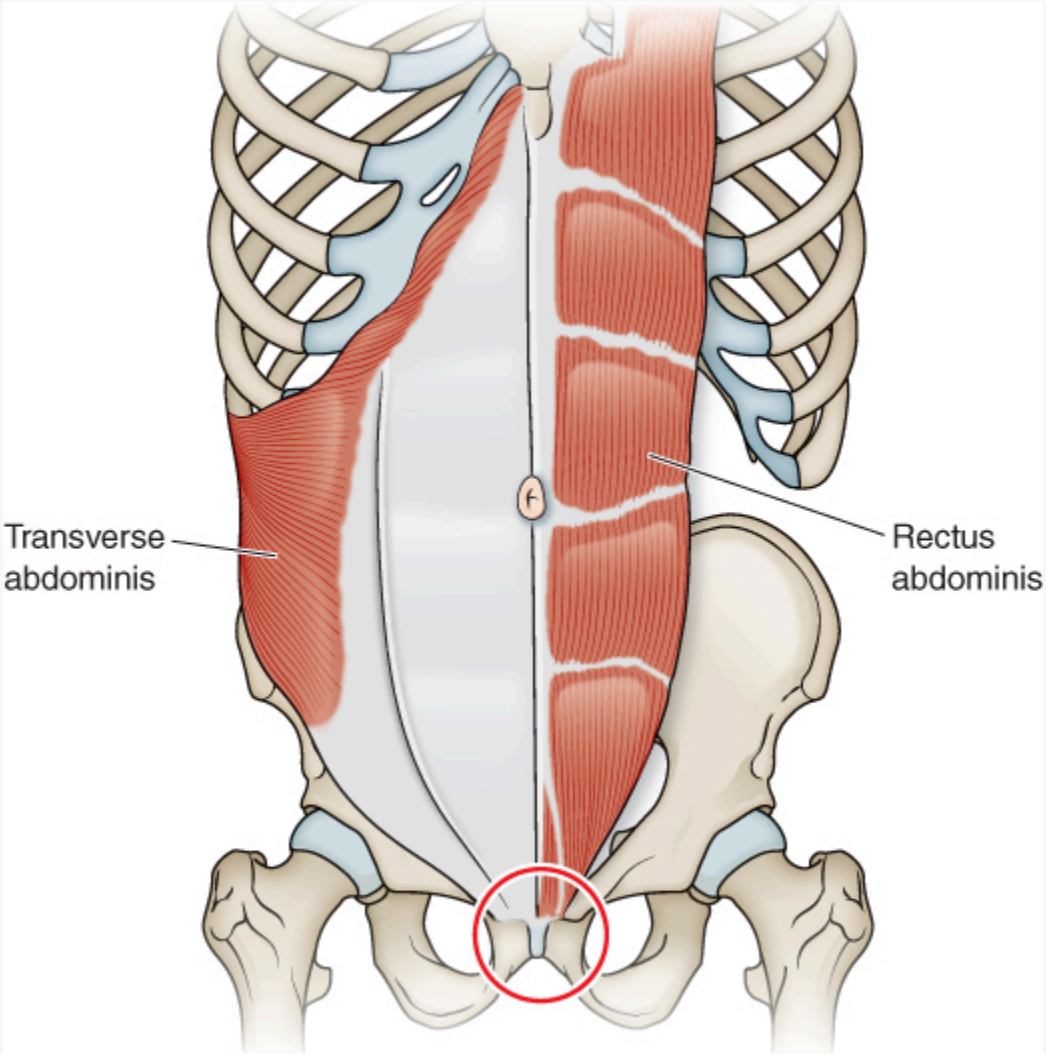
Symphysis Pubis Articulation

The symphysis pubis is a cartilaginous joint located between the two ends of the pubic bones (see Fig. 4–49). The end of each pubic bone is covered with a layer of articular cartilage, and the joint is formed by a fibrocartilaginous disc that joins the hyaline cartilage covered ends of the bones. The three ligaments that span the symphysis pubis are the **superior pubic ligament,** the **inferior pubic ligament,** and the **posterior pubic ligament.**[1] The superior ligament is a thick and dense fibrous band that attaches to the pubic crests and tubercles and helps support the superior aspect of the joint. The inferior ligament arches from the inferior rami on one side of the joint to the inferior portion of the rami on the other side and thus reinforces the inferior aspect of the joint. The posterior ligament consists of a fibrous membrane that is continuous with the periosteum of the pubic bones.[1] The anterior portion of the joint is reinforced by

aponeurotic expansions from a number of muscles, including the rectus abdominis and transverse abdominis (Fig. 4–53).

FIGURE 4-53

The aponeurotic expansions of several muscles cross the anterior aspect of the symphysis pubis, including the rectus abdominis and transverse abdominis.



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Function of the Sacral Region

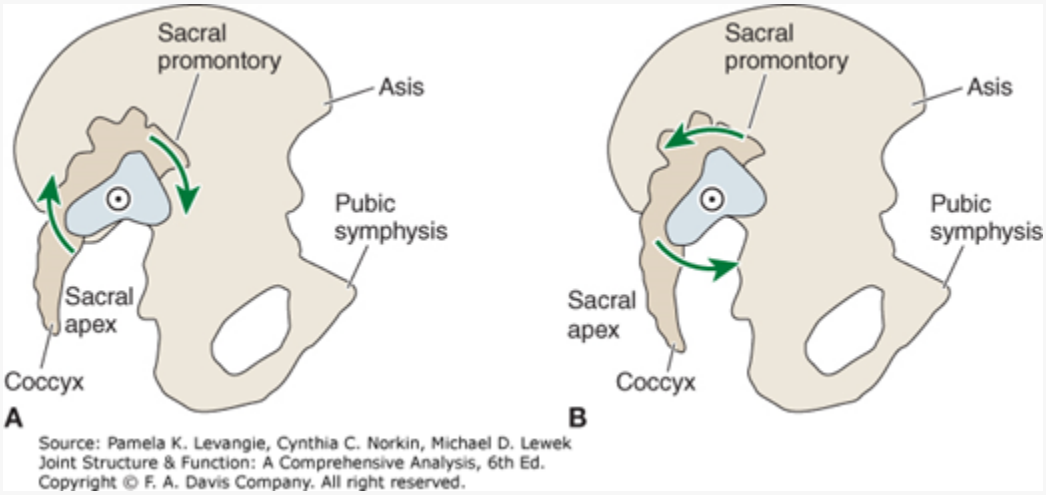
Kinematics

The kinematics of the sacroiliac joint are complex and the source of controversy, including the amount and type of motion available and the axes of motion. Three-dimensional analyses of motion using mathematical and computerized modeling have been used for measuring the small amounts of motion at these joints. Sturesson and colleagues found the mean amount of rotation at the sacroiliac joints to be between 1° and 3°, and the mean amount of translation to be approximately 1 mm when testing a variety of tasks.[57] This limited motion is supported by a systematic review of the literature that concluded: "motion at the sacroiliac joint is limited to minute amounts of rotation and of translation that may be sub-clinically detectable."[58] Although small in quantity, this motion is important for attenuating the forces transmitted via the pelvic ring to prevent sacral fracture.

Nutation is the term commonly used to refer to movement of the sacrum whereby the sacral base rotates or tips anteriorly on the relatively fixed innominates (**Fig. 4–54**). During the open-chain task of forward bending from a standing position, for example, the sacrum will nutate on the two innominate bones immediately following flexion at the lumbosacral junction and prior to flexion at the hip joints if, in fact, these happen sequentially (**Fig. 4–54A**). **Counternutation** refers to the opposite movement, in which the sacral base rotates or tips posteriorly on the relatively fixed innominates (**Fig. 4–54B**).

FIGURE 4-54

A. In sacral nutation the sacral promontory moves anteriorly and inferiorly while the inferior sacrum moves posteriorly and superiorly on the relatively fixed innominates. **B.** With counternutation, the sacral promontory moves posteriorly and superiorly while the sacral apex moves anteriorly on relatively fixed innominates.



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Motions of the sacroiliac joints are also described as **symmetrical** or **asymmetrical.** Symmetrical motion at the sacroiliac joints occurs when both innominates move simultaneously on the relatively fixed sacrum, although the motions are small in magnitude. In an **anterior pelvic tilt,** the innominates and sacrum move together as a relatively fixed unit in such a way that the anterior superior iliac spines rotate or tip anteriorly at the same time. In a **posterior pelvic tilt,** the two innominates and sacrum move together as a relatively fixed unit in such a way that the anterior superior iliac spines rotate or tip posteriorly at the same time. During both anterior and posterior pelvic tilt, the two innominates may move at a slightly different rate than the movement of the sacrum, resulting in slight sacroiliac joint movements during anterior

and posterior tilting. Anterior pelvic tilt is associated with hip flexion motion at the hip joints and posterior pelvic tilt is associated with extension motion at the hip joints.

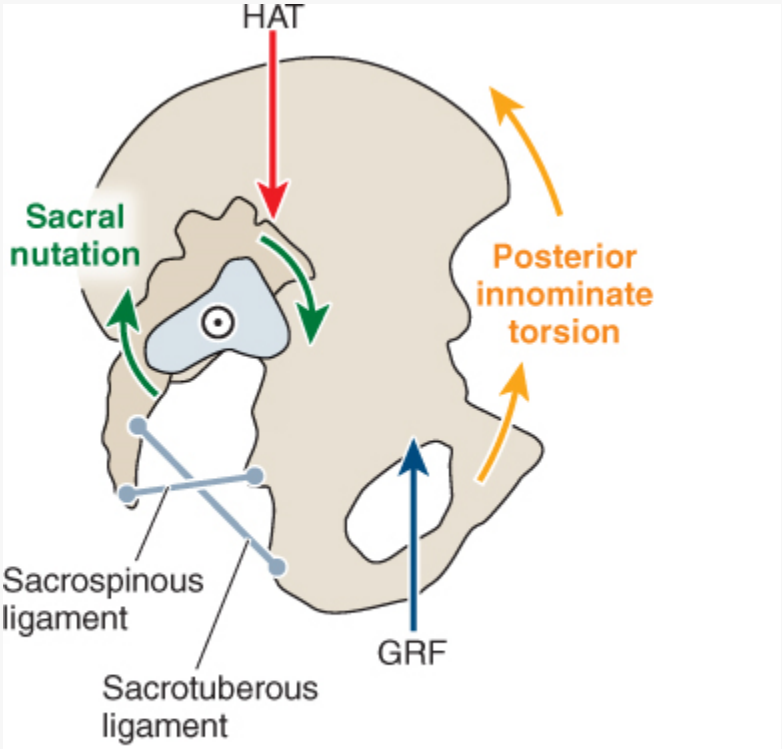
Asymmetrical sacroiliac motion is movement of the two innominate bones in opposite directions to each other. Because the pelvic ring consists of the sacrum and two innominates in a closed chain, any asymmetrical motion of the innominates at the sacroiliac joints results in motion at the pubic symphysis as well. Asymmetrical motion of the two innominates on the sacrum is described as **pelvic torsion.** This type of motion occurs during tasks such as ambulation, in which one hip is in a flexed position while the opposite hip is in an extended position. Although both motions are small in magnitude, the innominate on the side of the flexed hip will be torsioned in a posterior direction while the innominate on the side of the extended hip will be torsioned in an anterior direction.[59]

Kinetics

Stability of the sacroiliac joints is extremely important because these joints must support a large portion of the body weight. In normal erect posture, the weight of the head, arms, and trunk is transmitted through the fifth lumbar vertebra and lumbosacral disc to the first sacral segment. The force of the body weight creates a nutation torque on the sacrum; concomitantly, the ground reaction force creates a torque that causes posterior tilt of the innominates (Fig. 4–55). The counter torques of nutation of the sacrum and posterior tilt of the innominates are largely offset by the ligamentous tension and fibrous expansions from adjacent muscles that reinforce the joint capsules and blend with the ligaments.

FIGURE 4-55

In weightbearing, the weight of the superimposed head, arms, and trunk (HAT) creates a nutation torque of the sacrum whereas the ground reaction force (GRF) creates a posterior torque on the innominates via the femoral heads and acetabula. These counter-torques are offset by both muscles and ligaments, including the sacrospinous and sacrotuberous ligaments.



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Tension developed in the sacrotuberous, sacrospinous, and anterior sacroiliac ligaments counteracts the nutation torques of the sacrum. The interosseous ligaments compress the sacroiliac joint and bind the ilia to the sacrum. The long posterior sacroiliac ligament resists counternutation of the sacrum but is relaxed in nutation.[56]

The complementary ridges and depressions that develop in the cartilage of the sacral and ilial surfaces also contribute to the stability of the joint. Vertical loadbearing is facilitated by the development of these ridges and depressions, but motion is limited by them.



Foundational Concepts 4–6

Structure and Function of the Pelvic Ring

To summarize, the primary function of the pelvic ring is to transmit and dissipate forces from above and below. From above, the pelvic ring receives forces from the weight of the head, arms, and trunk as well as muscle contraction and any external loads that are being carried. From below, the pelvic ring receives ground reaction forces than can vary with activity. Although the sacroiliac joints and the pubic symphysis each have only very small amounts of movement, that movement is critical to allow the transmission and dissipation of all the descending and ascending forces without fracture of the pelvic ring. Further, the movement allows for torsion of the innominates, which is extremely important during ambulation and allows for counter rotation of the pelvis and trunk, which increases stride length.

Muscles of the Vertebral Column

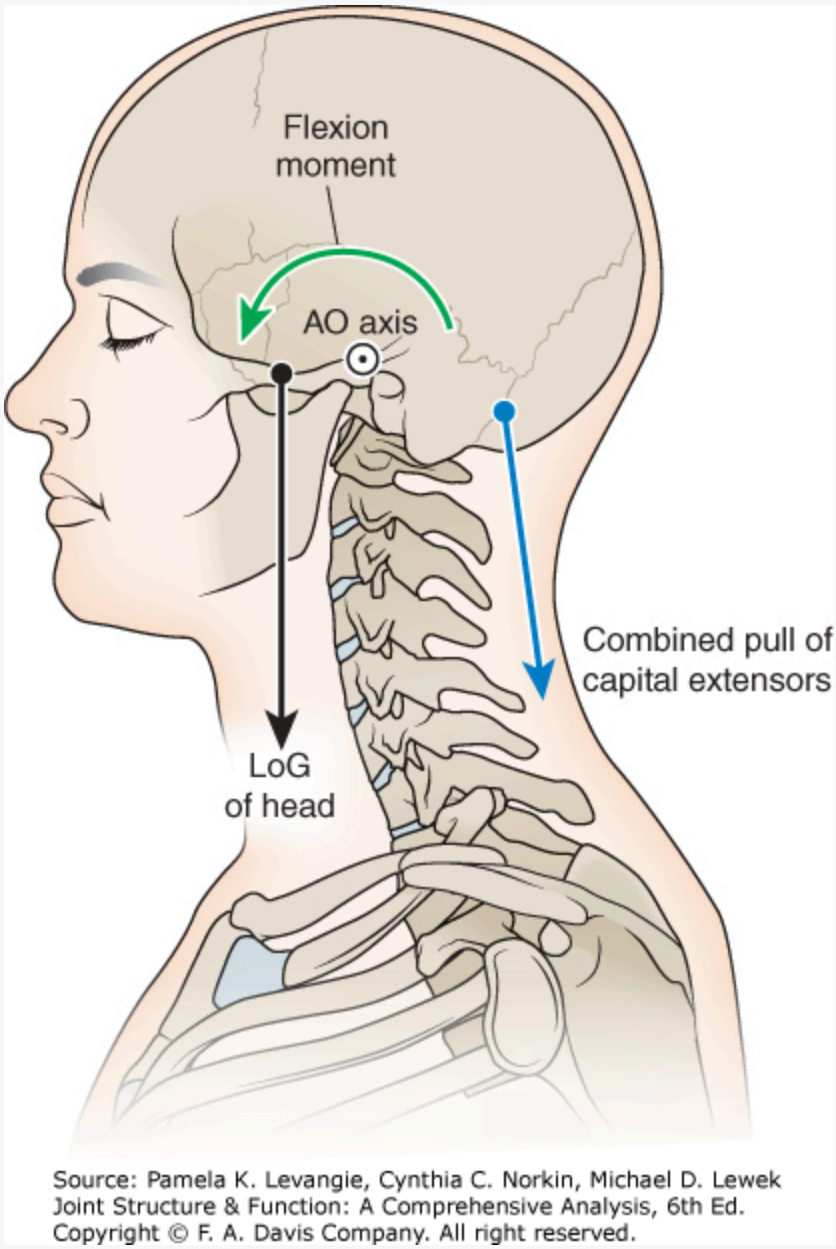
The Craniocervical/Upper Thoracic Regions

The muscles of the craniocervical region serve two primary roles: to hold the head upright against gravity and to position the head in space to optimally position the sensory organs. The muscles of the cervicothoracic region also position the head and neck in space along with the craniocervical muscles. An additional function of the cervicothoracic muscles is to stabilize the head and neck against the pull of muscles that produce or control movements of the scapula and upper extremity but pull on their direct or indirect vertebral attachments. The line of gravity in an upright standing

position passes anteriorly to the axis of rotation in the cervical region, producing a flexion moment (Fig. 4–56). The posterior muscles, along with the posterior ligamentous structures of this region, oppose this flexion moment. The need to position the head for the special sensory organs often includes rapid, coordinated movements, such as when a loud noise is heard and there is rapid turning of the head to locate the source of the sound. The muscular structure and function is complex to serve the demands for what can be both subtle and large amounts of motion, and yet provide sufficient stability to protect the spinal cord and allow for use of the upper extremities.

FIGURE 4-56

The line of gravity (LoG) in the cervical region passes anteriorly to the axes of rotation of the cervical vertebrae, producing a flexion moment. There must be tension in the extensor muscles to produce a counter moment.



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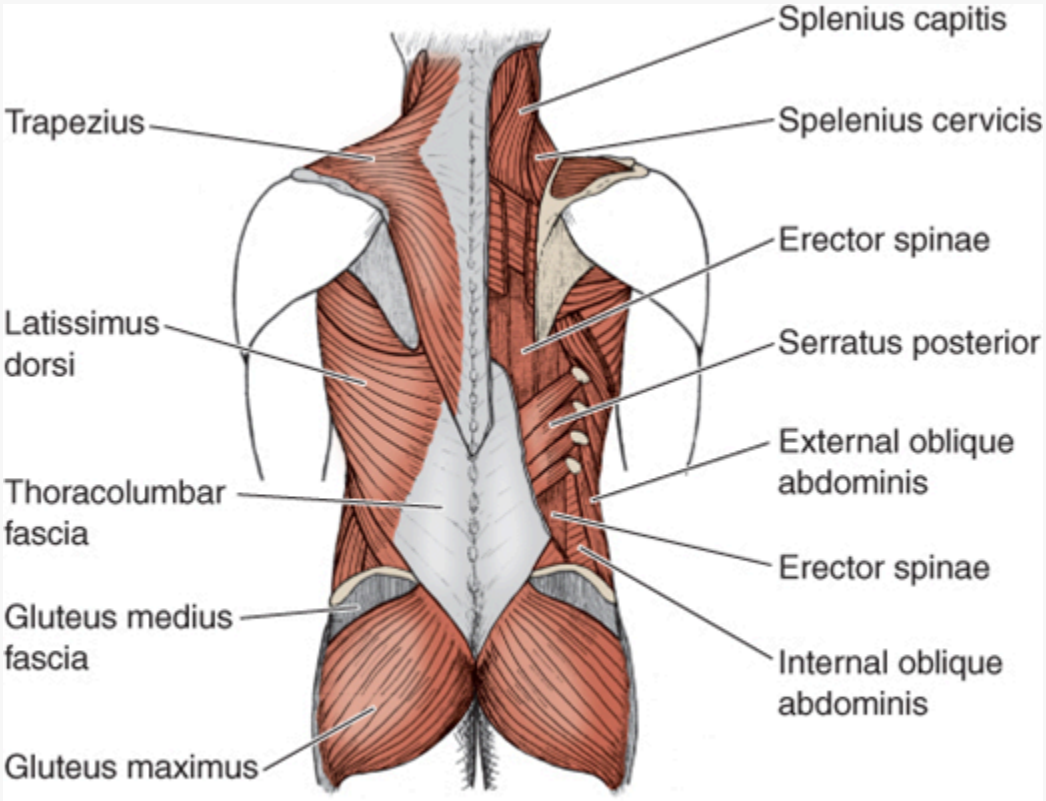
Posterior Muscles

We will examine the muscles from superficial to deep and begin with the posterior muscles (Figs. 4–57 and 4–58). The **trapezius muscle** is the most superficial of the posterior muscles. It spans from the occiput to the lower thoracic spine and contains a prominent tendinous region over the cervicothoracic junction. The trapezius functions

predominantly as a muscle of the shoulder region but can also produce extension of the head and neck. Acting unilaterally, the upper trapezius can produce ipsilateral lateral flexion and contralateral rotation of the head and neck.

FIGURE 4-57

Posterior back muscles. The superficial muscles have been removed on the right side to show the erector spinae. The anterior layer of the thoracolumbar fascia is intact on the left side of the back.

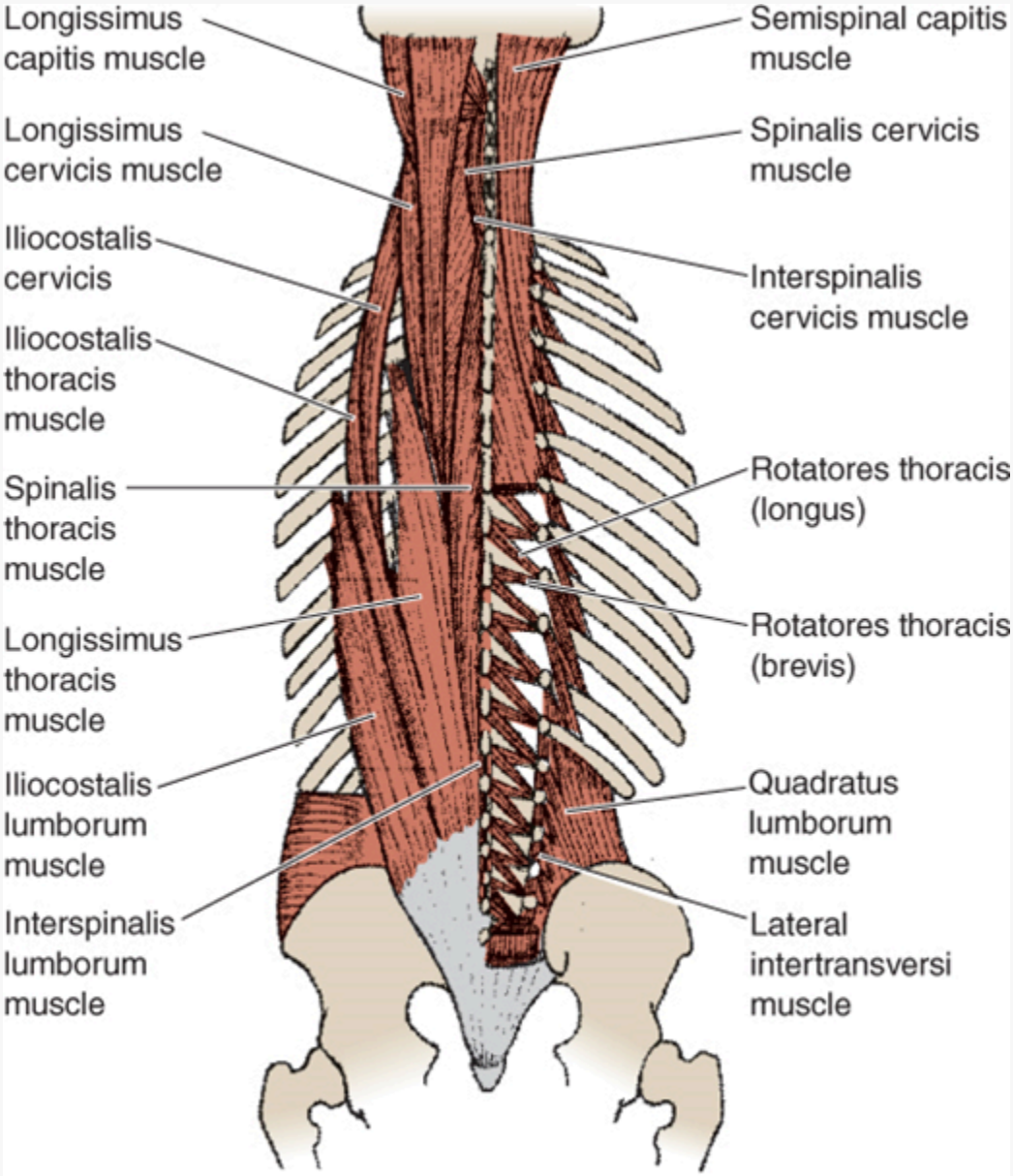


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FIGURE 4-58

Erector spinae and deep back muscles. The erector spinae muscles

have been removed from the right side of the neck to show the deep back muscles.



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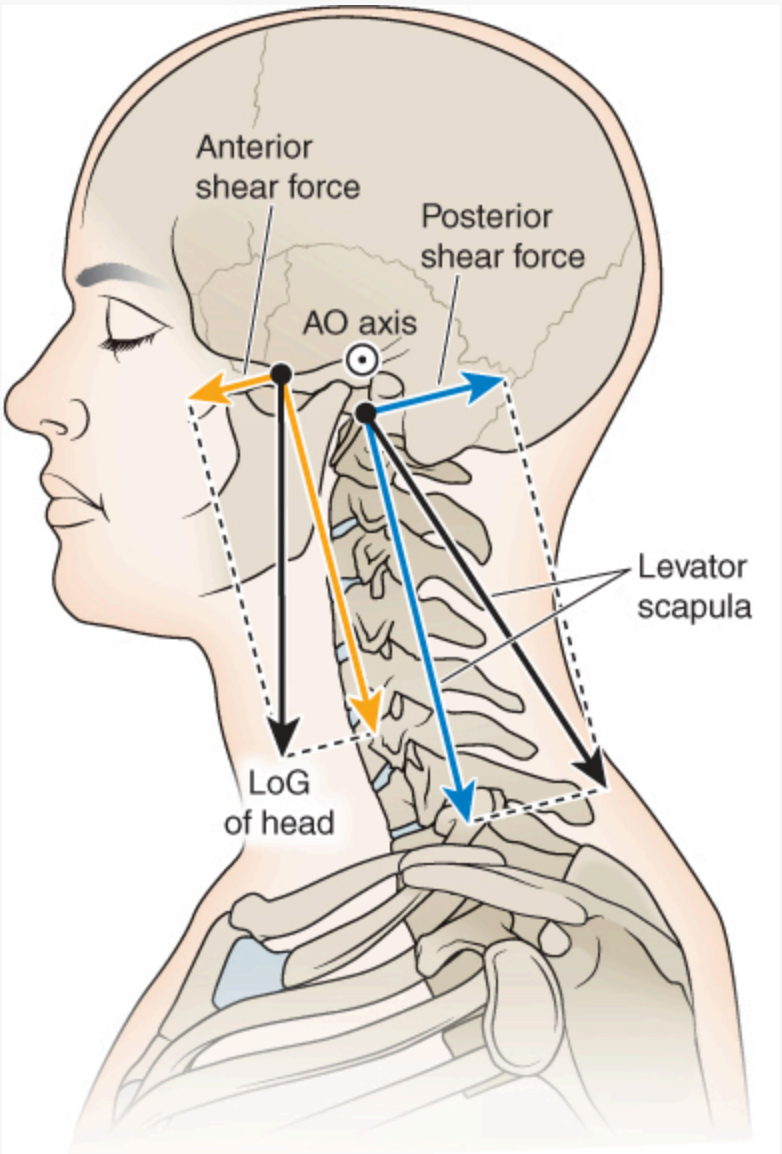
The **levator scapula** lies deep to the trapezius. It is attached proximally to the transverse processes of the first four cervical vertebrae and distally to the vertebral border of the

scapula between the root of the spine and the superior angle. This muscle has a large cross-sectional area. The levator scapula is an elevator and downward rotator of the scapula when the neck is stabilized, but can produce ipsilateral lateral flexion and rotation of the cervical spine if the scapula is stabilized.

The cervical spine is subjected to constant anterior shear forces caused by gravity and the lordotic position of the spine in this region. The levator scapulae help resist these forces by producing a counteracting posterior shear force (Fig. 4–59). An increase in the cervical lordosis or a greater flexion moment, as is often seen with an excessive forward head posture, will further increase the anterior shear forces on the cervical vertebrae and may cause excessive activity of the levator scapulae in resisting these more extreme anterior shear forces.

FIGURE 4-59

The cervical spine is subjected to anterior shear forces as a result of the lordotic curvature and anteriorly located line of gravity (LoG). The levator scapulae help resist the anterior shear forces by producing posterior shear.



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Patient Application 4–3

Sarah is a 23-year-old graduate student enrolled in a Doctor of Physical Therapy program. She spends approximately 15 hours per week sitting in lectures and also has to sit to study for many additional hours. Sarah describes her seated position as slouched with a forward head (cervical protraction), thoracic flexion, lumbar flexion, and posterior pelvic tilt. She also reports that she is frequently keyboarding on her laptop both during and outside of class. Since mid-semester, she has had right sided

neck pain over the insertion of the levator scapula that is now interfering with her ability to concentrate in class and type notes.

Over-activity of the levator scapulae in the presence of a forward head posture may be the reason that Sarah has pain and tenderness to palpation in the levator scapulae. In fact, the muscle is likely to be strained. Sarah states that she keeps trying to stretch that muscle but the pain is only worsening. Stretching the levator scapula in the presence of forward head posture may actually be worsening the situation and causing further irritation because it will decrease the muscle's ability to control the anterior shear forces produced from spending so much time in this position. Reduction or elimination of the excessive anterior shear forces is necessary for adequate treatment by changing Sarah's seated position into a more retracted cervical position. In addition, the levator scapulae may need strength and endurance training.

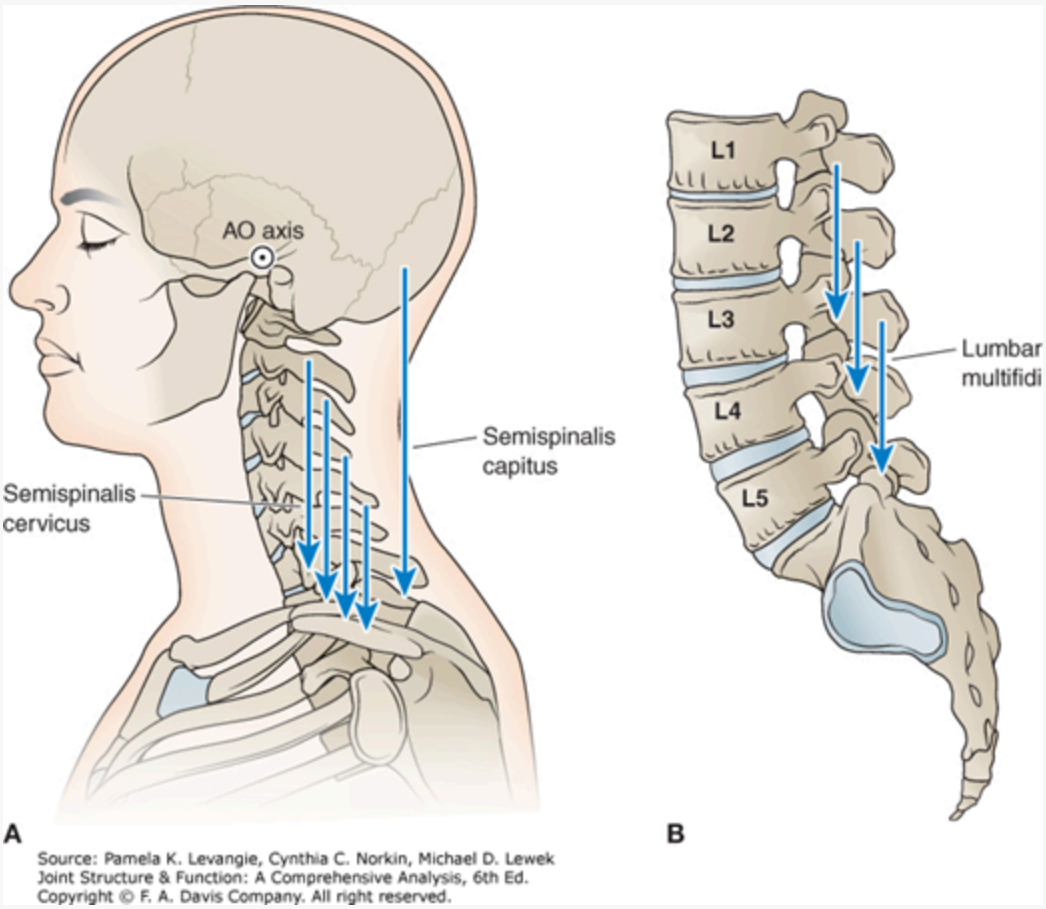
The splenius capitis and splenius cervicis muscles lie deeper than the levator scapulae. The splenius muscles are large, flat muscles with proximal attachments on the superior nuchal line, mastoid processes, and transverse processes of the upper first two or three cervical vertebrae and distal attachments on the spinous processes of the seventh cervical and first three to six thoracic vertebrae (see Fig. 4–57). These muscles serve as prime movers of the head and neck as a result of their large cross-sectional area and large moment arm. These muscles produce extension of the head and neck when working bilaterally and rotation to the ipsilateral side when working unilaterally. These muscles show little electromyographic activity in normal quiet stance.

The semispinalis capitis and semispinalis cervicis muscles are deeper than the splenius group. These muscles have an optimal line of pull and a large moment arm for producing extension of the head and neck and an increase in the cervical lordosis. The muscles run from the occiput to the cervical spinous processes (semispinalis capitis) and from the thoracic transverse processes to the articular processes of the lower

cervical spine (semispinalis cervicis) (see Fig. 4–58). These muscles together form the cord-like bundle of muscles that can be palpated lateral to the cervical spinous processes. Their function is often likened to that of the multifidi in the lumbar region. Like the lumbar multifidi, the semispinalis capitis and cervicis have an optimal alignment and moment arm for increasing the lordosis of the cervical spine (**Fig. 4–60**). It is important to note that the greater occipital nerve pierces the semispinalis capitis muscle on its way to innervate the skull. This can be a site of nerve irritation and entrapment when the semispinalis capitis is overactive or shortened, as in a forward head posture with craniocervical hyperextension. Occipital region headaches can result.

FIGURE 4-60

A. The semispinalis *capitis* and *cervicis* have optimal lines of pull to produce capital and cervical extension, respectively, which can increase the cervical lordosis. **B.** The lumbar multifidi have a similar line of pull that increases lumbar extension and the lumbar lordosis. *(B from Porter JA, DeRosa C: Mechanical Neck Pain. Saunders/Elsevier, 1995; with permission.)*



The longissimus capitis and longissimus cervicis are deeper than and lateral to the semispinalis group (see Fig. 4–58). Their deep position places them close to the axis of rotation for flexion and extension, rendering them less effective as extensors because of the small moment arm. They do, however, produce compression of the cervical segments. The lateral position allows them to produce ipsilateral lateral flexion when working unilaterally. When working bilaterally, the longissimus capitis and cervicis serve as frontal plane stabilizers of the cervical spine.

The suboccipital extensor muscles are the deepest posterior muscles and consist of the rectus capitis posterior major and minor, and the obliquus *capitis* superior and inferior.

These muscles run only between the occiput and C2, allowing independent movement of the craniovertebral region on the lower cervical spine. Together, they produce cranial (occipital) extension. Working unilaterally, they produce ipsilateral rotation and lateral flexion of the occiput or atlas. They also serve a proprioceptive role to fine-tune a craniocervical position. The suboccipital extensor muscles have been found to have density of muscle spindles that are 15 times greater than the density of the first lumbrical.[60] These muscles play an important somatosensory role in the cervical spine and are involved with integrated functions of the oculomotor and vestibular systems, particularly for high demand tasks such as head/eye coordination.

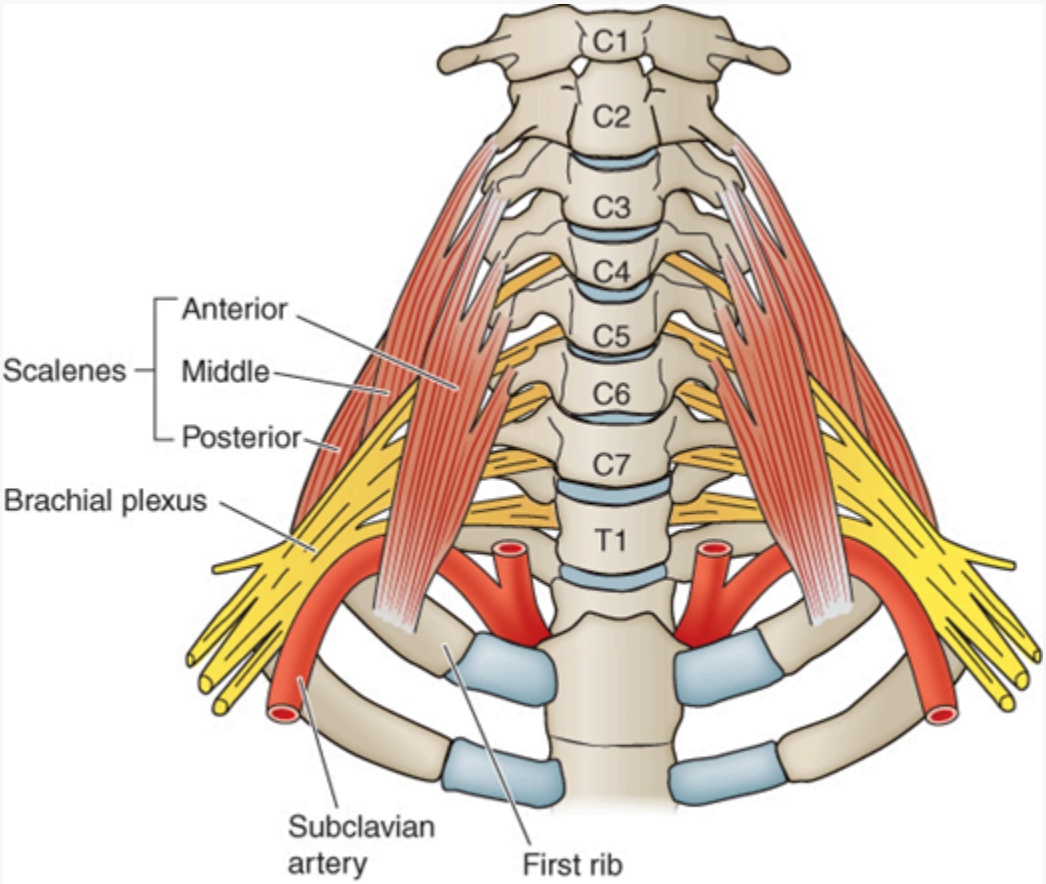
Lateral Muscles

The scalene muscles are located on the lateral aspect of the cervical spine (Fig. 4–61) and serve as frontal plane stabilizers when acting as a group with the posteriorly located longissimus muscles. The anterior scalene muscles run from the first rib to the anterior tubercles of the transverse processes of C3 to C6. The anterior scalenes work synergistically with the levator scapulae to provide cervical stabilization (Fig. 4–62). When working bilaterally, the anterior scalene will flex the cervical spine and produce an anterior shear. Unilaterally, the anterior scalene muscles will produce ipsilateral lateral flexion and rotation to the cervical spine to the opposite side. The middle scalene muscles run from the first rib to the posterior tubercles of the transverse processes of C3 to C7. The middle scalene muscles are placed more laterally than are the anterior scalene muscles, with lines of pull that make them excellent frontal plane stabilizers. The posterior scalene muscles run from the second rib to the posterior tubercles of the transverse processes of C3 to C7. The posterior scalene muscles predominantly laterally flex the neck. The role of the scalene muscles in breathing will

be discussed in Chapter 5. The anterior and middle scalene muscles form a triangle through which the brachial plexus and the subclavian artery and vein pass (see Fig. 4–61). This can be a site for compression of the neurovascular structures by the anterior scalene muscle that can produce pain, numbness, and tingling in the arm.

FIGURE 4-61

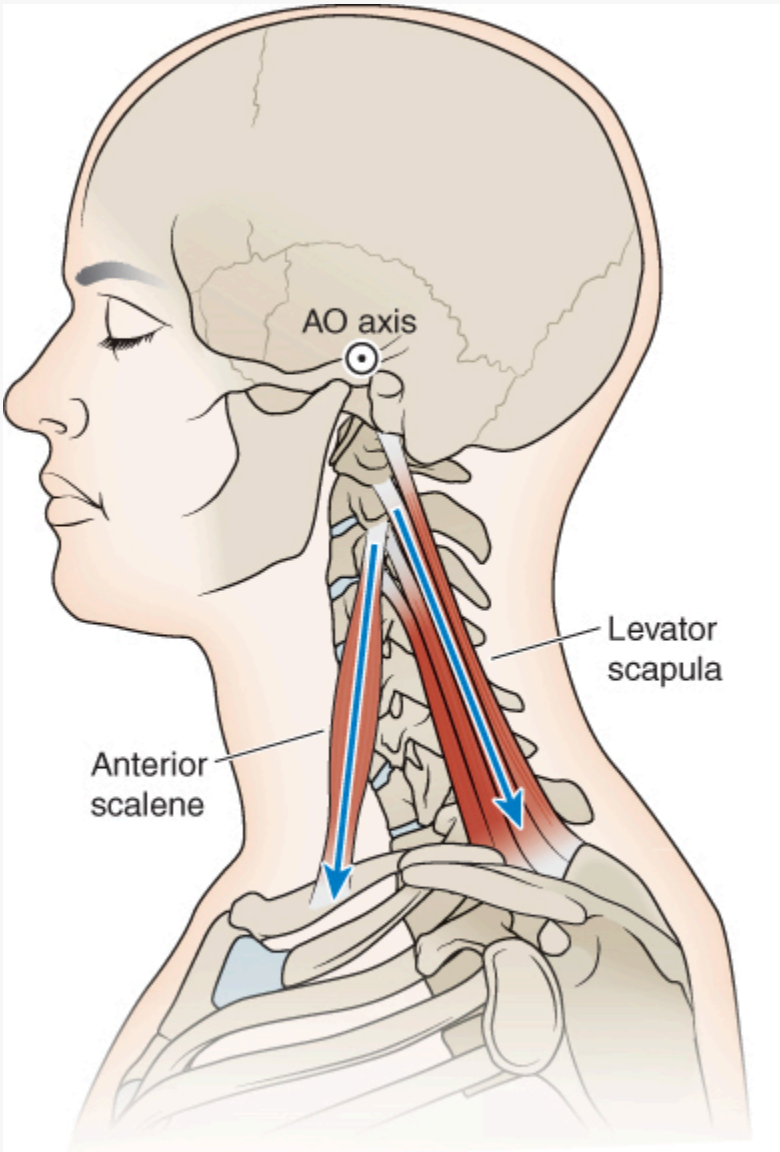
The anterior, middle, and posterior scalene. The brachial plexus and subclavian artery and vein pass between the anterior and middle scalene muscles.



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FIGURE 4-62

In the sagittal plane, the anterior scalene muscles work in synergy with the levator scapulae to provide stabilization of the cervical spine.



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The sternocleidomastoid muscle runs from the manubrium and medial clavicle to the mastoid process. The angle of pull of the muscle is posterior, medial, and superior. It is

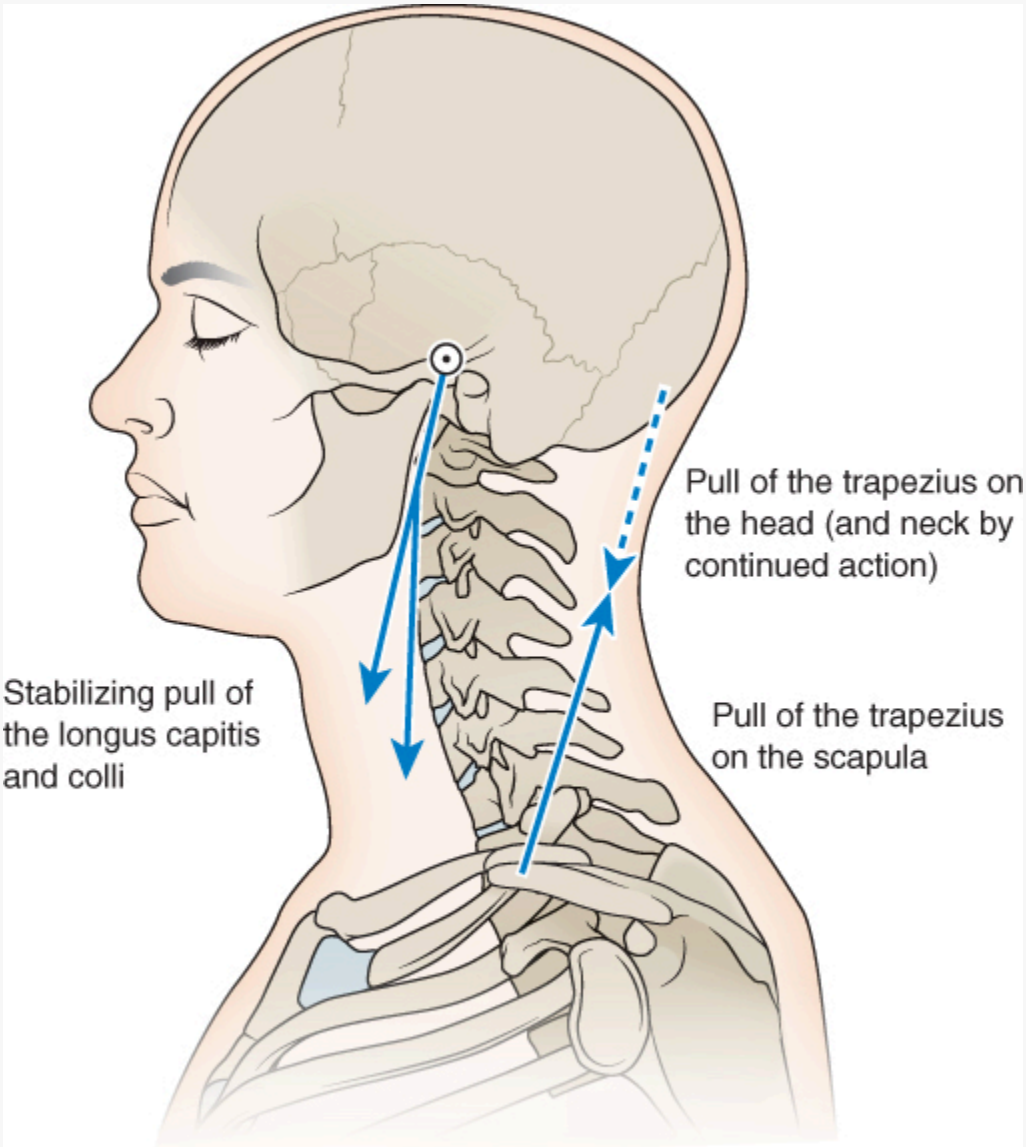
unique in that it lies anterior to the axis of rotation in the lower cervical spine but posterior to the atlantooccipital axis. Therefore, a bilateral contraction of the sternocleidomastoid muscles produces simultaneous flexion of the cervical spine and extension of the head on the neck. Acting unilaterally, the sternocleidomastoid muscle will produce ipsilateral lateral flexion and contralateral rotation of both the head and neck.

Anterior Muscles

The longus capitis runs from the anterior tubercles of the cervical transverse processes to the occiput. The longus colli runs from the thoracic vertebral bodies to the anterior tubercles of the cervical transverse processes and cranially from the anterior tubercles of the transverse processes to the atlas. These muscles lie close to the vertebral bodies and, therefore, are relatively close to the axis of rotation. Although they do have sufficient moment arm to produce flexion, they also produce a fair amount of cervical compression. The longus capitis and longus colli work synergistically with the trapezius to stabilize the head and neck so that the trapezius can act effectively on its distal attachment to produce upward rotation of the scapula[31] (Fig. 4–63). Given that muscles always pull on both ends during a contraction, the trapezius would extend the lighter head and neck rather than upwardly rotate the heavier scapulohumeral complex if it weren't for the counterforce on the neck of the longus capitis and colli. The longus colli has a high density of muscle spindles that serve as sensory input and highlight their role in fine-tuning segmental control of the cervical region.[61]

FIGURE 4-63

The longus capitis and longus colli work synergistically with the upper trapezius to stabilize the head so the upper trapezius can effectively pull on the scapula, to contribute to elevation of the heavier upper extremity.



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The small rectus capitis anterior and rectus capitis lateralis run between the occiput and the atlas (C2) and are flexors of the head as a result of their lines of pull. Like the suboccipital extensor muscles, the small cross-sectional area and moment arms for these paired muscles likely serve a greater proprioceptive function rather than acting as prime movers of the head.

Patient Application 4–4

Lisa is a 39-year-old woman who was stopped at a traffic light 1 week ago when she was rear-ended by another car going about 20 miles per hour. She was wearing her seatbelt when she was hit. Lisa is experiencing severe neck pain with activities, especially those that involve raising either arm. She is having trouble dressing, reaching overhead, and even holding her arms for typing, which is interfering with her job. She had radiographs that were normal and she cannot understand why moving her arms is causing so much pain when her arms were not injured in the accident.

Patients who have experienced a whiplash injury, as in a rear-end motor vehicle accident, often have extreme difficulty raising their arms. The longus capitis and longus colli can be overstretched and even torn in whiplash injuries and damage to either will not be evident on radiograph. If the longus capitis and colli are damaged, they will be unable to stabilize the head and neck against the pull of the trapezius on its proximal attachments. As a result, the trapezius acts on the head and neck rather than effectively producing the upward rotation of the scapula needed to fully elevate the shoulder complex. The paradoxical action of the trapezius on the head and neck rather than on the scapula explains Lisa's pain and problems raising her arms.

Lower Thoracic/Lumbopelvic Regions

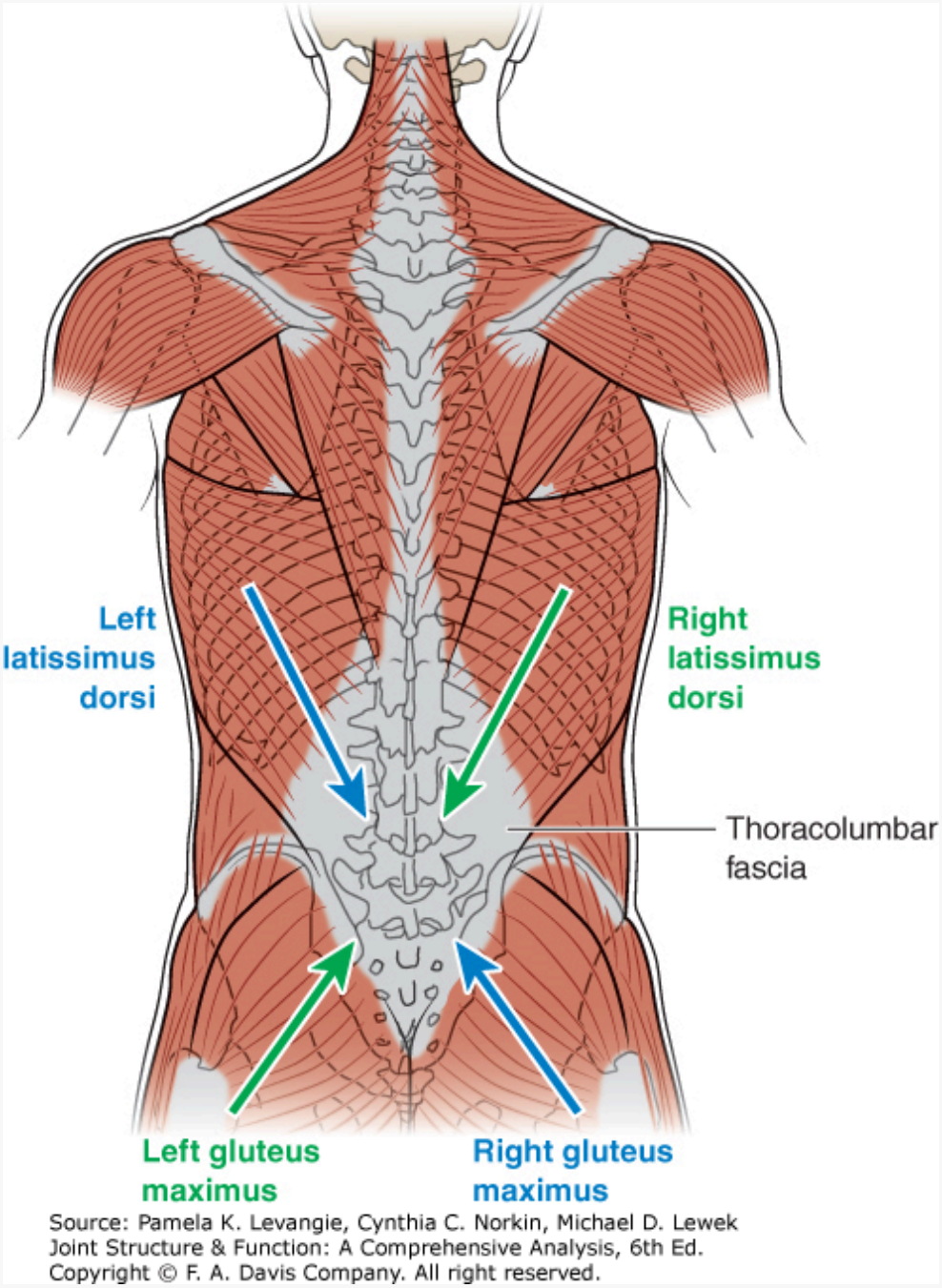
Muscles of the lower spine regions produce and control movement of the trunk and stabilize the trunk for motion of the lower extremities. They assist in attenuating the extensive forces that affect this area and are involved with anticipatory motor control of the extremities to stabilize the spine.

Posterior Muscles

The thoracolumbar fascia is the most superficial structure on the posterior aspect of the lower thoracic and lumbopelvic region (see Fig. 4–57) and is associated with several major muscle groups of this region. The thoracolumbar fascia blends with the latissimus dorsi, the gluteus maximus, the internal and external abdominal oblique, and the transversus abdominis (see Fig. 4–57). In addition, the fascia surrounds the erector spinae and the multifidus muscles of the lumbar region (see Fig. 4–45). These attachments are significant because tensile forces can be exerted on the thoracolumbar fascia through active and passive tension in these muscles. Tension on the thoracolumbar fascia will produce a force that compresses the abdominal contents. Like a contraction of the abdominal muscles, the abdominal compression produced through the thoracolumbar fascia is similar to that of an external corset. The coupled action of contralateral latissimus dorsi and gluteus maximus muscles, and tension through the thoracolumbar fascia together compress the lumbosacral region and make it stable (**Fig. 4–64**).[47]

FIGURE 4-64

Coupled action of the contralateral latissimus dorsi and gluteus maximus muscles, and tension through the thoracolumbar fascia will compress and stabilize the lumbosacral region.



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Also posteriorly located, the erector spinae consist of the longissimus and iliocostalis muscle groups (Table 4–5). In general, these muscles are identified as extensors of the trunk. Bogduk[2] examined the function of the longissimus thoracis and the iliocostalis lumborum; he further described these muscles as each having a lumbar portion (pars

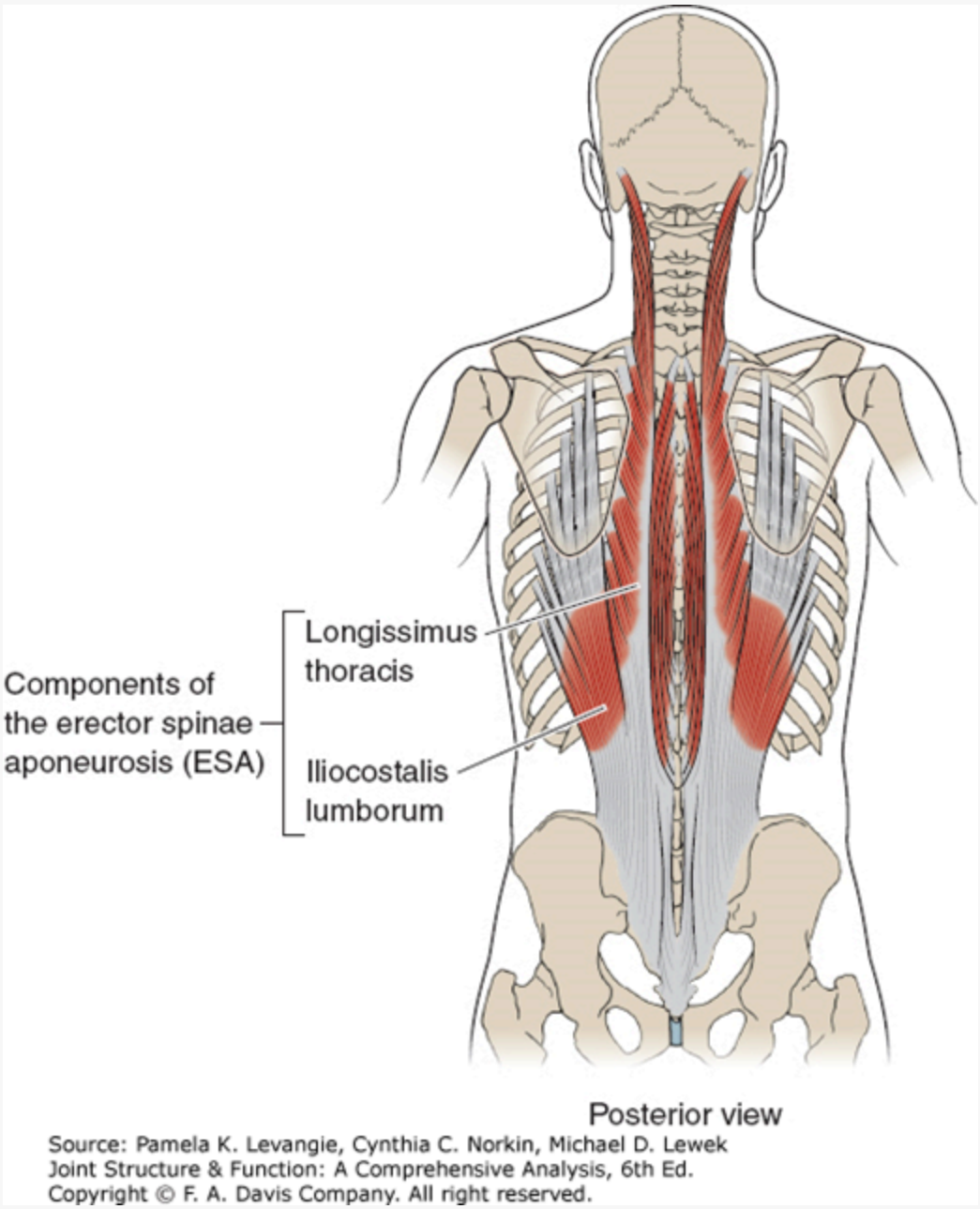
lumborum) and a thoracic portion (pars thoracis). Anatomically and functionally, it is easier to group these muscles together as the superficial and deep layers. The **superficial layer** (longissimus thoracis pars thoracis and the iliocostalis lumborum pars thoracis[2]) runs from the ribs and thoracic transverse processes to form muscle bellies that are laterally located in the thoracic region. The superficial muscles have long tendons that join together to form the **erector spinae aponeurosis** that inserts into the spinous processes of the lower lumbar spine, sacrum, and iliac crest (**Fig. 4–65**). This superficial layer, with its long moment arm and excellent line of pull, produces extension of the thoracic and lumbar regions when acting bilaterally (**Fig. 4–66**). These muscles are considered the primary extensors of the trunk. Acting unilaterally, the superficial erector spinae are able to laterally flex the trunk and contribute to rotation. During trunk flexion from a standing position, the erector spinae are responsible for contracting eccentrically to control the motion. The gravitational moment will produce forward flexion, but the amount and rate of flexion are controlled partially by eccentric contractions of the extensors with the passive restraint of the erector spinae aponeurosis and partially by the thoracolumbar fascia and posterior ligamentous system. The erector spinae act eccentrically until approximately two thirds of maximal flexion has been attained, at which point these muscles become electrically silent. This is called the **flexion-relaxation phenomenon.** Flexion-relaxation is presumed to occur when the stretched passive tissues are able to generate the required extension moment. However, the extensor muscles may be relaxed only in the electrical sense because they may also be generating passive force through passive stretching.[62]

Table 4-5

Location and Function of the Erector Spine

FIGURE 4-65

The superficial erector spinae with the erector spinae aponeurosis (ESA).

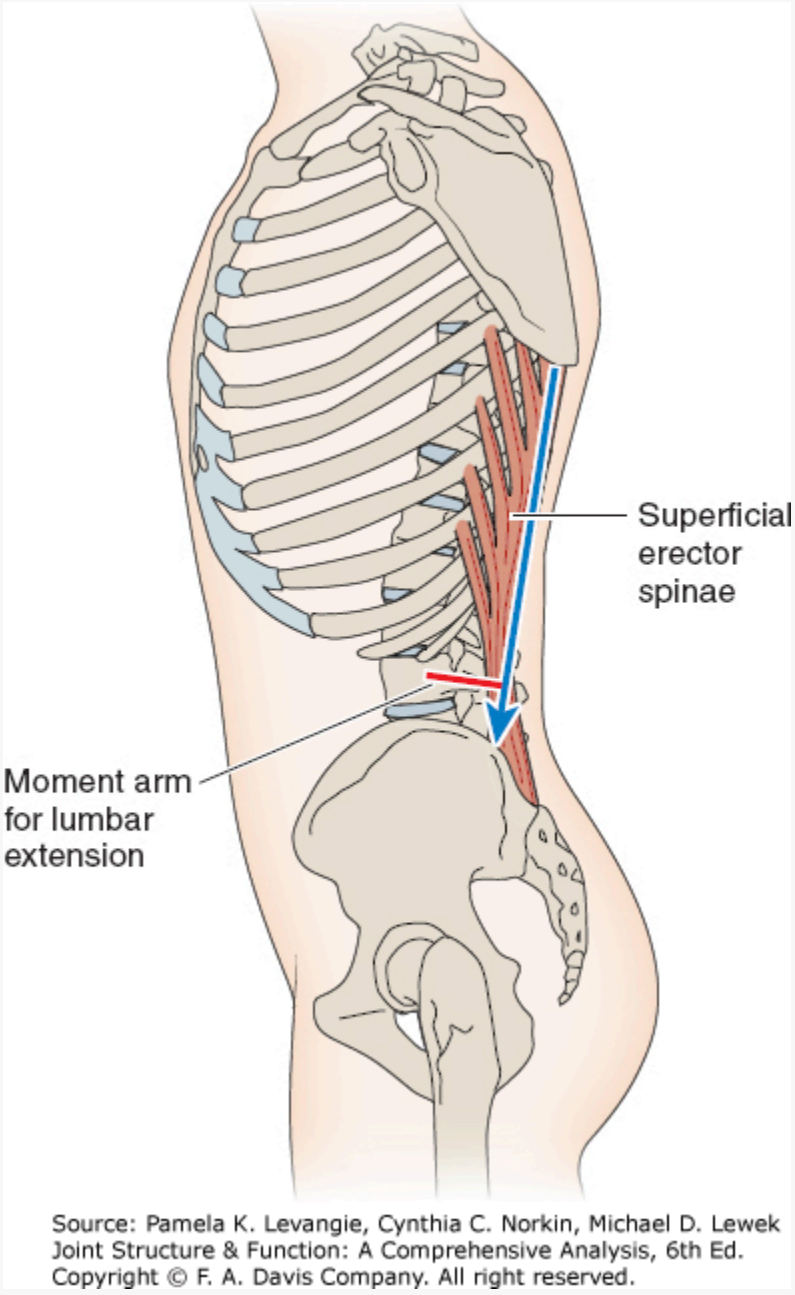


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FIGURE 4-66

Sagittal view of the force vectors of the superficial erector spinae, demonstrating the excellent line of pull and large moment arm for

extension.



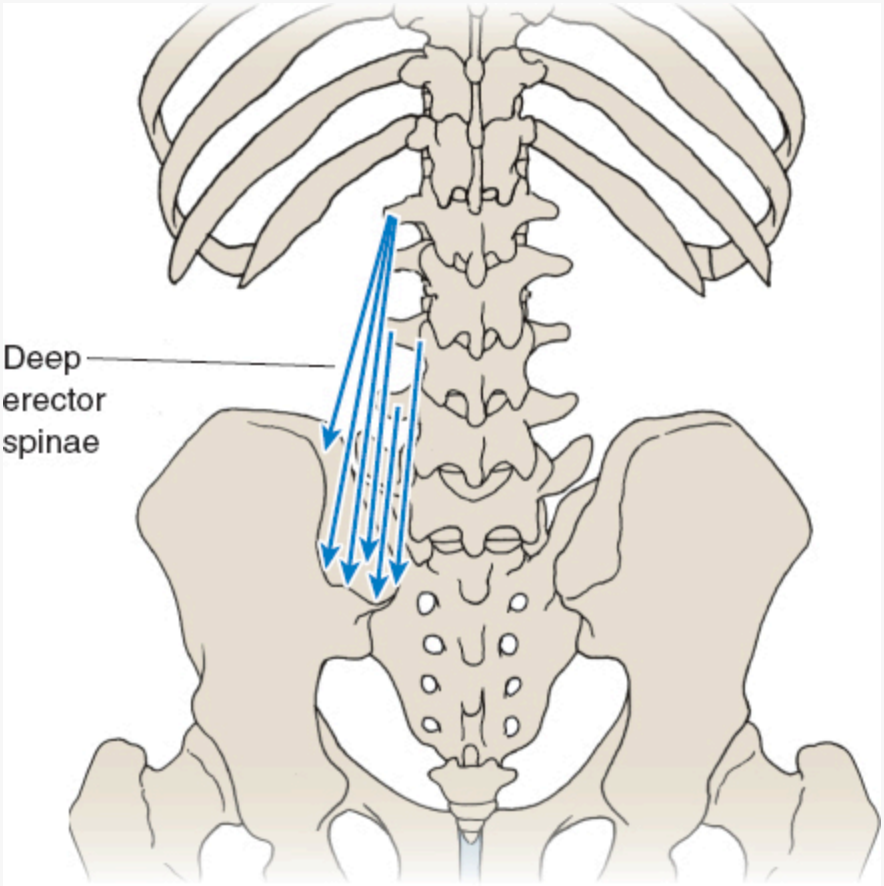
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The **deep layer** of the erector spinae (longissimus thoracis pars lumborum and the iliocostalis lumborum pars lumborum[2]) consists of individual fascicles with common tendinous insertions. The fascicles arise from the ilium at the posterior superior iliac

spine and the iliac crest and course superiorly, medially, and anteriorly to insert on the lumbar transverse processes (Fig. 4–67). There may also be some attachment to the erector spinae aponeurosis of the upper lumbar fascicles. Porterfield and DeRosa[63] described these deep erector spinae as similar in orientation and function to the levator scapulae in the cervical region. Both of these muscle groups lie close to the flexion/extension axis without a sufficient moment arm to be prime movers for extension. The oblique orientation, however, allows these muscles to exert both posterior shear and compressive forces on their vertebrae (Fig. 4–68). Because of their oblique line of action and ability to produce posterior shear, the deep erector spinae muscles provide an extremely important dynamic resistance to the constant anterior shear forces of the lumbar region caused by the lordotic position and the combined forces of gravity and the ground reaction. These muscles, then, have great clinical significance.

FIGURE 4-67

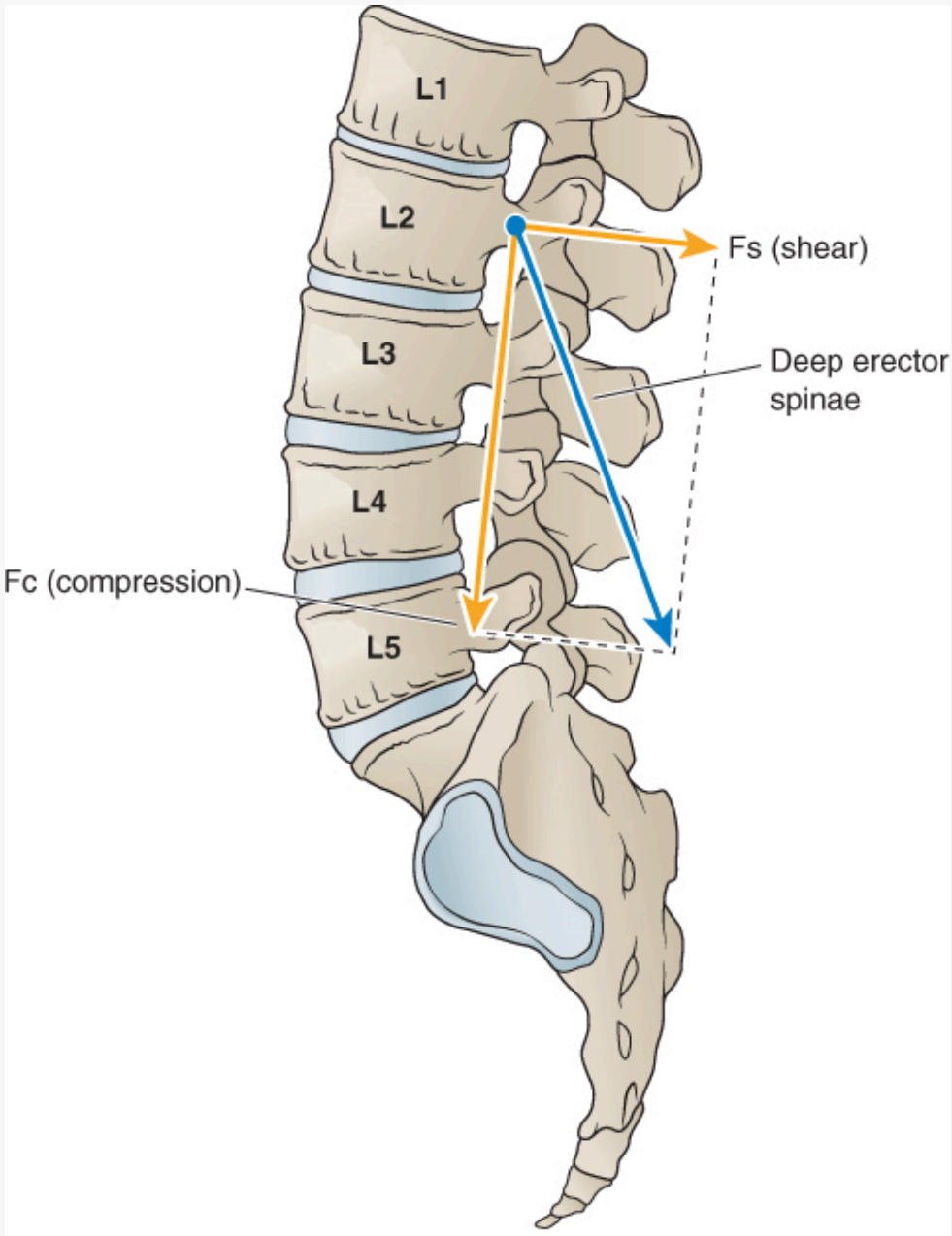
The fascicles of the deep erector spinae.



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FIGURE 4-68

Side view of the force vectors of the deep erector spinae, demonstrating the compression (Fc) and posterior shear (Fs) components of the muscular pull.



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Expanded Concepts 4–3

Negative Effects of Overstretching the Deep Erector Spinae

Like the levator scapulae in the cervical region, the deep erector spinae will become overworked, painful, and strained when subjected to excessive anterior shear forces.

Caution should be taken, however, in regard to stretching these muscles. Overstretching the deep erector spinae muscles can remove the dynamic restraint to excessive anterior shear forces and either further load the non-contractile structures or, if the non-contractile forces have already been damaged, remove the only remaining restraint to anterior shear. In either case, stretching the deep erector spinae is likely to worsen the symptoms. It is more likely that these muscles need greater endurance and strength training.

The multifidus muscles of the spine are complex and demonstrate segmental and regional differences. The multifidus muscle group is typically considered to be part of the **transversospinales** group that also includes the semispinalis and **rotatores** muscles that both attach to transverse processes below and spinous processes above. However, the lumbar multifidi run from the dorsal sacrum and the ilium in the region of the posterior superior iliac spine to the spinous processes of the lumbar vertebrae and have separate fascicles.[64–66] In this region, therefore, the multifidus muscles are not characteristic of the other muscles in the transversospinales group. The lumbar multifidus muscles are a thick mass that fills the area of the sacral sulcus and are easily palpable there (unlike the other transversospinales).[64,65]

The most superficial fibers of each fascicle of the lumbar multifidi cross as many as five segments and attach caudally to the sacrum and ilium. Their line of pull is vertically oriented, and they are distant from the flexion/extension axis. Therefore, the superficial fibers of the multifidi have an effective moment arm for extension by increasing the lumbar lordosis (see Fig. 4–60). The deep fibers of each fascicle attach from the lamina and spinous process and cross two segments to insert on the mamillary process and facet joint capsule. The deep fibers in the lumbar region have a small moment arm and, therefore, are not strong extensors. These deep fibers are ideally positioned to control

segmental shear and torsion by producing compression. The role of the lumbar multifidus muscles in trunk rotation is to work in synergy with the abdominal muscles by counteracting the flexion moment that the abdominal muscles produce. The lumbar multifidus muscles are involved in local segmental control in the lumbar region.

Expanded Concepts 4–4

The literature related to low back pain identifies the lumbar multifidi as having a unique role in anticipatory postural control. Most of the literature that discusses motor control of the lumbopelvic region includes the deep erector spinae fibers as part of the multifidi. Researchers have examined a variety of tasks that include upper extremity tasks, lower extremity tasks (both open and closed chain tasks), and ambulation. When examining anticipatory motor control, most muscle activity is task dependent. They have found a unique role, however, for the lumbar multifidi and the transversus abdominis. Regardless of the task tested, these muscles fire prior to the contraction of the prime mover of the task, that is, in an anticipatory manner. In the presence of low back pain (or a history of low back pain), these anticipatory contractions are delayed.[42,67]

In the thoracic region, the multifidus muscles are similar to other muscles of the transversospinales group because they are more laterally oriented with an oblique line of pull. They run from all of the transverse processes of the thoracic vertebrae to the spinous processes of the more cranial vertebrae, covering one to three segments. The thoracic multifidus muscles are activated with rotation but in a variable manner, suggesting that the thoracic multifidi, like the deep lumbar multifidi, have a role in segmental control of motion rather than production of motion.[64]

The posteriorly located **intertransversarii** and rotatores muscles are frequently described as producing lateral flexion and rotation, respectively. Because of their small cross-sectional areas and small moment arms, however, it appears likely that these

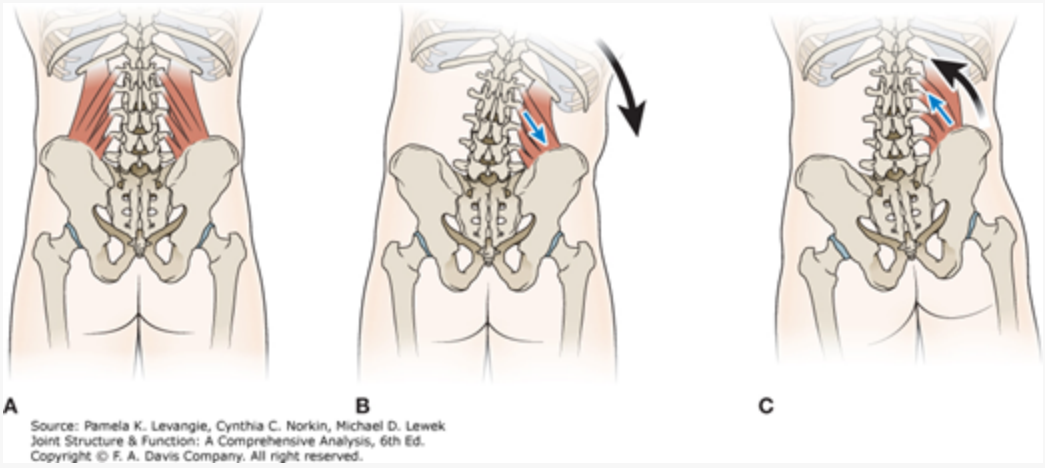
muscles serve more of a proprioceptive role.

Lateral Muscles

The quadratus lumborum is deeper than the erector spinae and multifidus muscles (Fig. 4–69). The quadratus lumborum, when acting bilaterally, serves as an important frontal plane stabilizer and is active in many tasks in this role (Fig. 4–69A).[14] When acting unilaterally, the quadratus lumborum can laterally flex the spine (Fig. 4–69B). If lateral flexion occurs from erect standing, the force of gravity will continue the motion, and the contralateral quadratus lumborum will control the movement eccentrically. The quadratus lumborum can also control rotational motion due to its attachments to the transverse processes of the lumbar vertebrae. If the pelvis is free to move, the quadratus lumborum will "hike the hip," or laterally tilt the pelvis in the frontal plane (Fig. 4–69C).

FIGURE 4-69

A. The attachments of the right and left quadratus lumborum muscles. **B.** A unilateral contraction of the right quadratus lumborum muscle when the pelvis and left leg are fixed will cause right trunk flexion. **C.** A unilateral contraction of the right quadratus lumborum muscle will lift and tilt the right side of the pelvis and "hike the hip" when the trunk is fixed and the pelvis and leg are free to move.



Anterior Muscles

The rectus abdominis is the prime flexor of the trunk. It is contained within the abdominal fascia, which separates the rectus abdominis into sections and attaches the rectus abdominis to the aponeurosis of the abdominal wall. The abdominal fascia also has attachment to the aponeurosis of the pectoralis major. These fascial connections are important as they transmit forces across the midline and around the trunk. Tension on this fascial system will provide stability around the trunk similar to that provided by a corset.

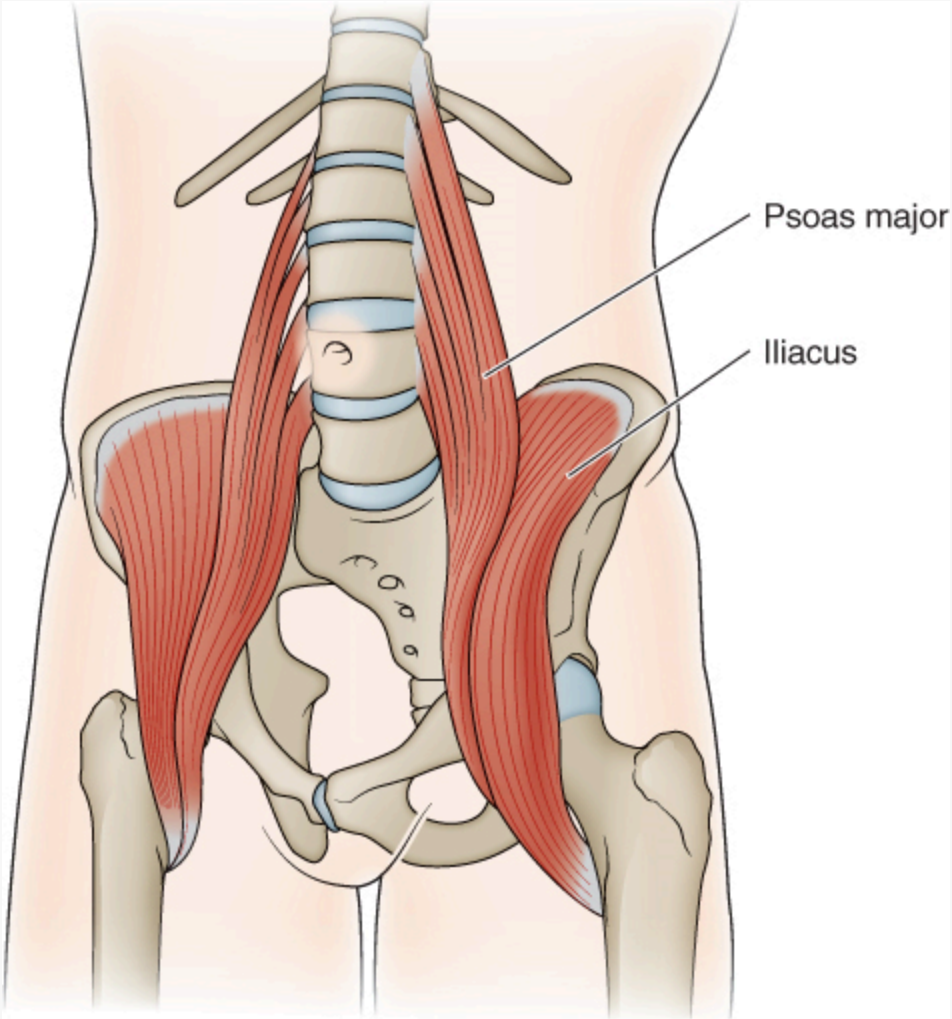
The abdominal wall consists of the external oblique, the internal oblique, and the transversus abdominis muscles. As described earlier in the discussion of the thoracolumbar fascia, these muscles form the abdominal hoop around the entire abdomen (see Fig. 4–45). The transversus abdominis has been shown to stabilize the sacroiliac joints via the application of compressive forces with contraction due to the transverse (perpendicular) orientation of the muscle to the joints.[68]

The **psoas major** may also be considered an anterior truck muscle. The psoas major

muscle runs from the lumbar transverse processes, the anterolateral vertebral bodies of T12 to L4, and the lumbar intervertebral discs to the lesser trochanter of the femur. The psoas major courses inferiorly and laterally, and the distal tendon merges with that of the **iliacus** muscle (Fig. 4–70). The primary role of the psoas major is flexion of the hip. The iliacus runs from the iliac crest over the pubic ramus to the lesser trochanter with the tendon of the psoas major. When active hip flexion is initiated, the iliac muscle exerts a pull on its respective innominate that, if unopposed, would create an anterior pelvic tilt and consequent lumbar extension (lordosis) if the goal was to remain upright. The psoas major, also active in hip flexion, creates an anterior pull on the lumbar vertebrae that resists the lumbar extension force secondary to the pull of the iliacus muscle. The psoas major also provides stability to the lumbar spine during hip flexion activities by providing lumbar compression during activation. Some anterior shear is also produced when it is activated.

FIGURE 4-70

The anteriorly located psoas major and iliacus muscles are primary hip flexors. However, the iliacus simultaneously creates an anterior pull on the pelvis that would result in lumbar lordosis if not offset by the simultaneous anterior pull of the active psoas major on the lumbar vertebrae.



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Foundational Concepts 4–7

Exercises for Low Back Pain

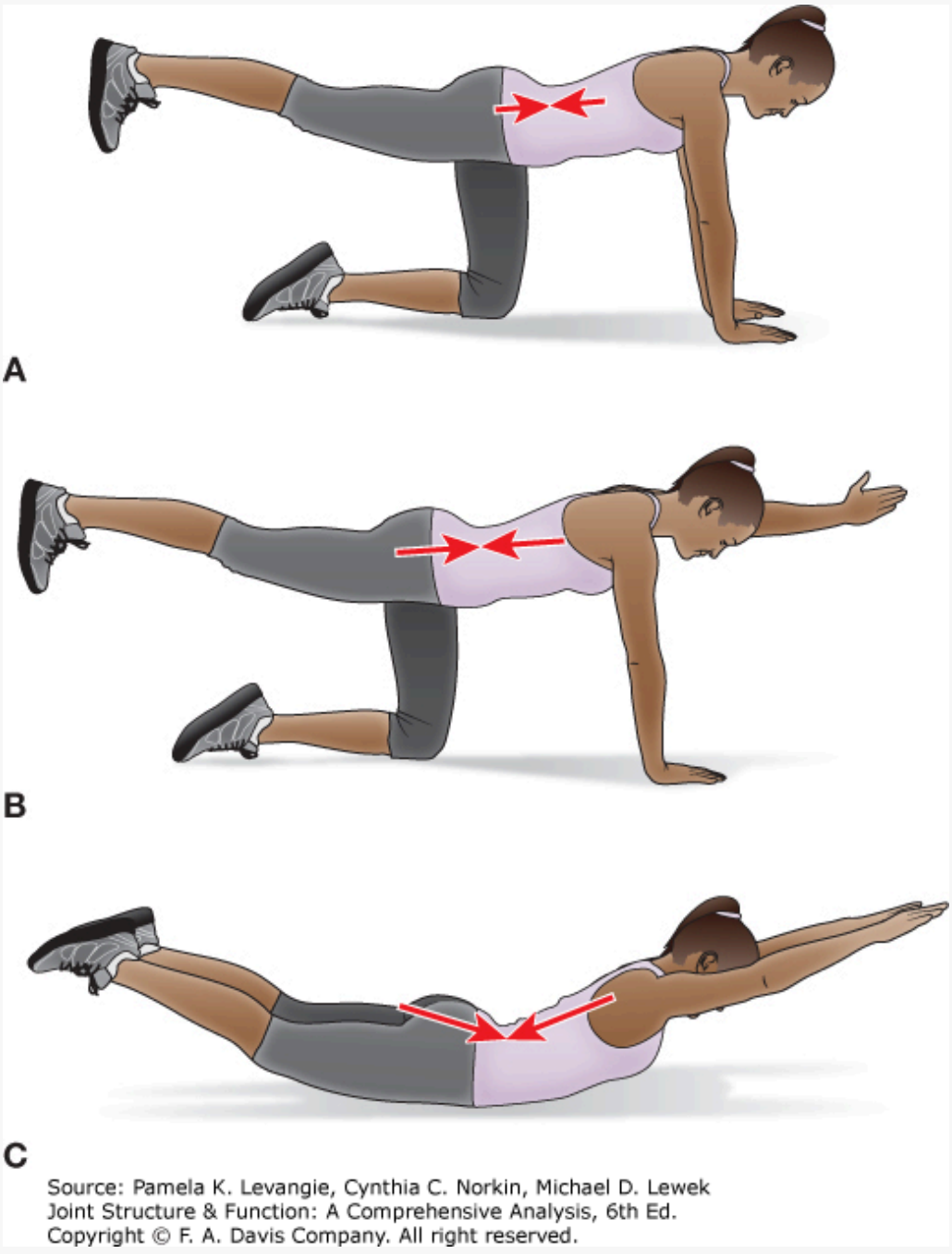
When developing therapeutic exercises for people with low back disorders, one should choose exercises that, while training the target muscle groups, also impose the lowest possible loads through this region, especially in the acutely painful or early healing stages.

Exercises to increase the strength of the back extensors cause substantially different compressive loads related to position of the joints and compressive forces from muscle contraction. For example, as shown in Figure 4–71, single-leg extension in the

quadruped (Fig. 4–71A) produces the lowest compressive forces (approximately 2,500 N). Raising an opposite arm and leg simultaneously (Fig. 4–71B) increases the compressive forces by 1,000 N and upper erector spinae muscle activity by 30%.[69] The right erector spinae and contralateral abdominal muscles were activated during single right-leg extension to maintain a neutral pelvis and spine posture and to balance internal moments and lateral shear forces. A prone exercise that involves raising upper and lower extremities off the support surface simultaneously (often called the Superman exercise) causes up to 6,000 N, which may cause injury (Fig. 4–71C).

FIGURE 4-71

A. Single leg extension in the quadruped position creates relatively low compression forces at the L4–L5 segment at an estimated 2,500 N. **B.** Raising the opposite leg and arm simultaneously increases compression forces at the L4–L5 segment by 1,000 N and upper erector spinae muscle activity by 30% in comparison with single leg raising. **C.** Raising all four limbs simultaneously can raise the compression at L4–L5 to an estimated 6000 N.



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Muscles of the Pelvic Floor

A comprehensive review of the structure and function of the pelvic floor is beyond the scope of this chapter. The role of the muscles of the pelvic floor in providing stability to the lumbopelvic ring, however, is now recognized as being important to understanding

low back pain. Dysfunction of the muscles of the pelvic floor has been identified as both a risk factor for as well as a result of low back pain.[70] Given the role of the pelvic floor musculature in stability of this region, its structure and function will be discussed here briefly.

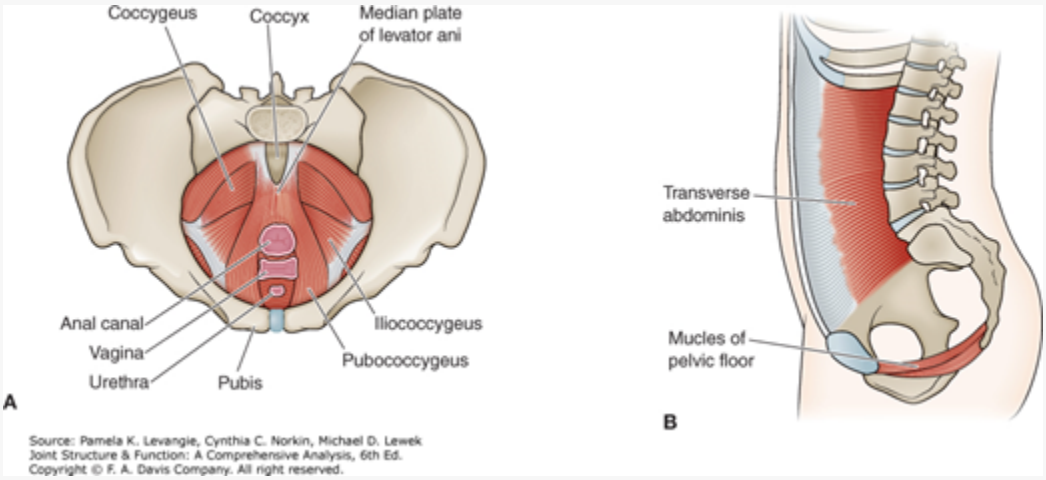
Structure

The muscles of the pelvic floor are the broad muscle sheets of the **levator ani** muscles and the coccygeus muscles, which together separate the pelvic cavity from the perineum (Fig. 4–72). The two distinct parts of the levator ani are the **iliococcygeus** and the **pubococcygeus muscles** (Fig. 4–72A). The pubococcygeal part of the levator ani muscle arises from the posterior aspect of the pubis and has attachments to the rectum in the anal canal, the urethra, and the walls of the vagina (in women). The iliococcygeal portion of the levator ani arises from the obturator fascia, blends with the fibers of the anococcygeal ligament to form the median plate (raphe) of the levator ani, and attaches to the last two coccygeal segments. The coccygeus muscle arises from the spine of the ischium and attaches to the coccyx and lower portion of the sacrum. The gluteal surface of the coccygeus muscle blends with the sacrospinous ligament.

FIGURE 4-72

Muscles of the pelvic floor. **A.** The two components of the levator ani muscles (pubococcygeus and iliococcygeus muscles) and the coccygeus muscles form the floor of the pelvis referred to as the pelvic diaphragm. **B.** The pelvic diaphragm and the transverse abdominis work together actively or passively to increase support (stiffness) to

the lumbopelvic region.



Function

In addition to their roles in controlling the sphincters for micturition and defecation, the levator ani and the coccygeus muscles form a muscular (pelvic) diaphragm that supports the pelvic viscera (**Fig. 4–72B**) while also generating, maintaining, and increasing intra-abdominal pressure in functional tasks.[71,2] Increased intra-abdominal pressure created by tension in the muscles of the pelvic diaphragm increases stiffness to the lumbopelvic area, especially during co-contraction with the transverses abdominis muscle,[73] thus contributing to lumbar spine stiffness and support. In addition, the coccygeus muscle crosses the sacroiliac joint and contributes to compression of these joints in the female, which increases the stiffness of the pelvic ring.[72]

Expanded Concepts 4–5

Pelvic floor muscle dysfunction has been related to the development of low back pain. Hodges and colleagues have shown that the muscles of the pelvic floor contract as part of a feed forward (anticipatory) mechanism of postural control, similar to the

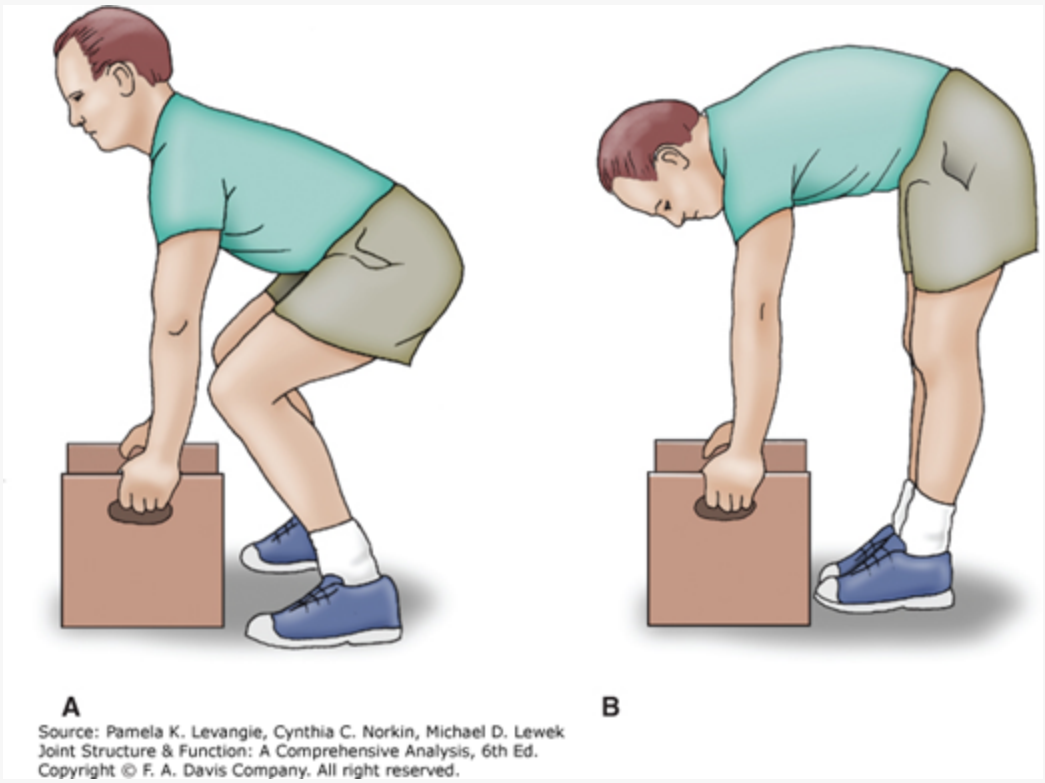
lumbar multifidi discussed earlier, and that these contractions are delayed in women with stress urinary incontinence.[40,74] A significant difference in pelvic floor muscle function between women with and without low back pain also has been demonstrated.[70–72] It seems clear that motor control of the lumbopelvic area involves highly coordinated activation between the pelvic floor muscles, deep abdominal muscles, and deep erector spinae. A variety of changes in this coordination has been demonstrated for those with low back pain or urinary incontinence, and the best treatments require a greater understanding of this coordination and the coordination impairments found in the clinical population.

Squat Lift Versus Stoop Lift

The prevalence of back problems in the general population and the difficulties of resolving these problems has generated a great deal of research, both to explain the mechanisms involved in lifting and to determine the best method of lifting so that back injuries can be prevented. Focus has primarily been on the use of a squat versus stoop lift (Fig. 4–73). During a squat lift (Fig. 4–73A), the spine remains as erect as possible and trunk flexion is achieved primarily by hip and knee flexion. During a stoop lift (Fig. 4–73B), trunk flexion is achieved primarily by thoracolumbar flexion, and there is little to no knee flexion.

FIGURE 4-73

A. Squat lift versus **B.** Stoop lift.



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The literature is not clear regarding whether there is biomechanical evidence to support advocating one lifting technique over another to prevent low back pain. Although the points that follow appear to support the use of the squat lift, it is likely that the best posture for lifting is task-specific. There are some advantages and disadvantages to each technique.

The spinal extensor muscles are at a disadvantage in the fully flexed position of the spine in a stoop lift because of shortened extensor moment arms in this position, a change in the line of pull, and the potential active insufficiency resulting from active contraction of muscles that are in an elongated state. In a neutral lumbar spine posture (squat lift), the deep layer of the erector spinae is capable of producing a posterior shear force that can help to offset the large anterior shear produced by body weight

during trunk flexion, particularly if the person is carrying an additional load.[75,76] The diminished capacity of the spinal extensors to generate torque and to counteract the anterior shear forces in the forward flexed position are important reasons that stoop lifting is discouraged.

Stoop lifting also appears to be contraindicated because of intradiscal pressures that are substantially higher when a load is held in a stooped position than in a squat position.[77,78] Similarly, shear forces on lumbar vertebrae were found to be two to four times higher for the stoop lift than for the squat lift,[41] and there is evidence that the spine is better capable of resisting compressive forces than shear forces.[76] Bazrgari and colleagues analyzed the dynamics of squat and stoop lifting and concluded that "net moments, muscle forces at different levels, passive forces and internal compression/shear forces are larger in stoop lifts than squat ones."[79]

Less controversial but still critical factors in lifting in any posture appear to be the distance from the body of the object to be lifted, the velocity of the lift, and the degree of lumbar flexion. The farther away the load is from the body, the greater the gravitational moment acting on the vertebral column will be. Greater muscle activity is required to perform the lift and, consequently, greater pressure is created in the discs. The higher the velocity of the lift, the greater the amount of weight that can be lifted, but the greater the load on the lumbar discs.

Effects of Aging

Over the life span, the vertebral column is exposed to recurrent loads that change the morphology of the column. However, normal age-related changes are also occurring in

the structures of the vertebral column.

The vertebral bone undergoes changes in the amount and form of the trabeculae. The numbers of both horizontal and vertical trabeculae decrease with age, and the horizontal trabeculae become significantly thinner. This loss can decrease the loads that the vertebrae are able to withstand before failure.

Each of the structures of the intervertebral disc undergoes changes that include loss in the amount of proteoglycans and change in the specific type of proteoglycan, with resultant loss of water content. In addition, there is an increase of collagen in these structures and loss of elastin. These changes in connective tissue composition result in a loss of the ability of the disc to transfer loads from one vertebra to another as the swelling ability of the nucleus decreases. The overall disc height will also decrease.

The vertebral endplate, with aging, gradually becomes more collagenous and the process of diffusion is hindered.[80] Because the disc relies on the diffusion of nutrients through the vertebral endplate, nutrition of the disc becomes impaired with changes in the endplate. Eventually, the intervertebral disc can desiccate. Lamellae of the annulus separate and can result in fissures and tears that can decrease the ability of the disc to provide stiffness during movement.[81]

A disc can prolapse or protrude as a result of the pressure on the nucleus and the inability of the annulus fibrosus to restrain it. Schmorl's nodes are formed when the nuclear material prolapses through the vertebral endplate and into the cancellous bone of the vertebra. This material may cause an autoimmune response when it comes in contact with the blood supply in the cancellous bone.[80] This presentation is typically

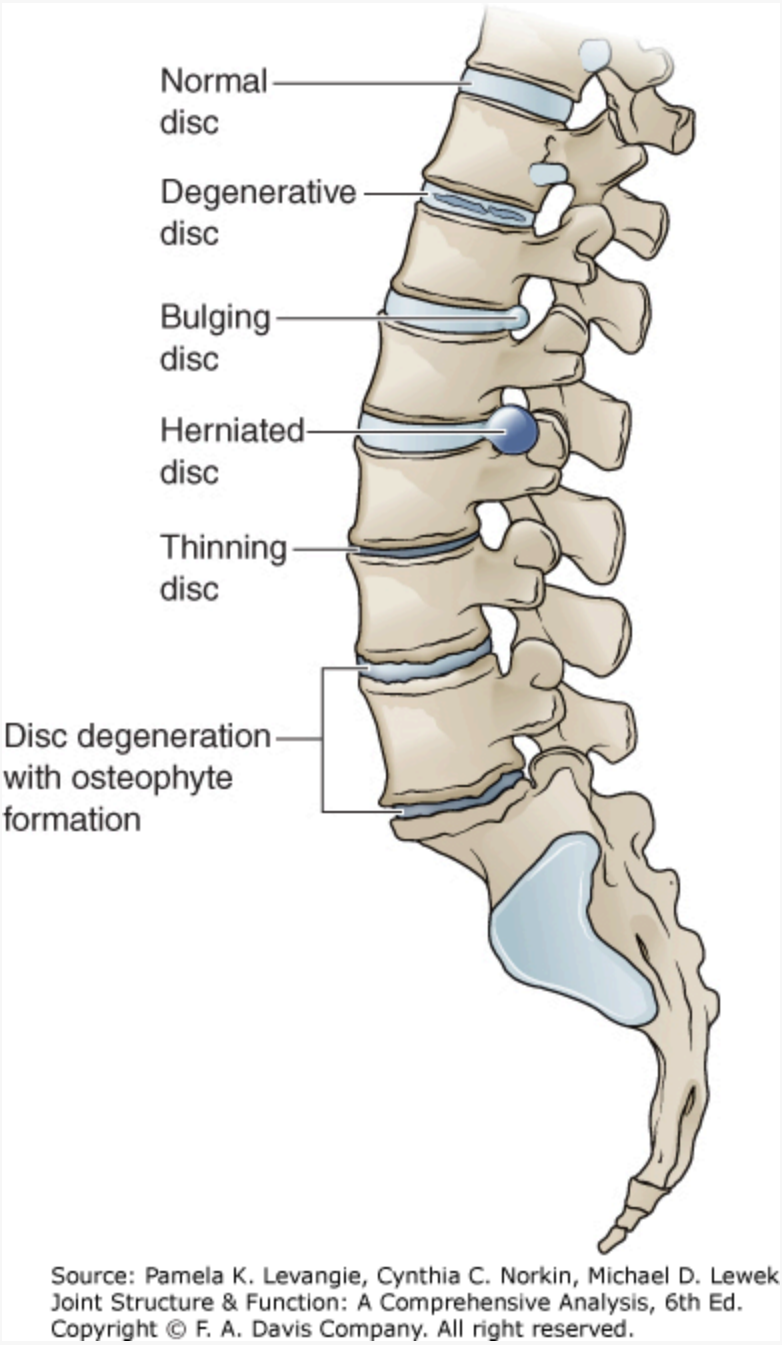
labeled degenerative disc disease.

With degenerative disc disease, there is a more substantial loss of disc height that can cause vertebral ligaments to become slack. The ligamentous pre-stress normally provided by the ligamentum flavum will decrease, which in turn will reduce spinal stiffness (stability). The reduction in tension in the ligamentum flavum can also result in the ligament buckling on itself with movement, potentially compressing the spinal cord. In addition, the ligamentum flavum may calcify with age, potentially leading to hypertrophic ossification that can cause compression of the spinal nerve in the vicinity of the zygapophyseal joints or compress the spinal cord within the canal.[12] On occasion, blood vessels grow into the usually avascular adult discs, triggering ossification.

The zygapophyseal joints can also demonstrate age-related changes and eventual degeneration. With degeneration of the disc, more compressive loads are borne at the zygapophyseal joints that then may lead to deterioration and arthritic changes. The result of these changes to the zygapophyseal joints is the same as what happens to the larger synovial joints of the extremities: damage of the cartilage, including fissures and cysts, and osteophyte formation.[80] These changes can lead to localized pain or pressure on spinal nerves or the central canal or, in the cervical region, compression of the vertebral artery in the transverse foramen. The cascade of effects seen with age-related changes to the spine are shown in Figures 4–74 and 4–75.

FIGURE 4-74

Age-related changes that can occur in the spine are shown, including disc degeneration, bulging of the annulus fibrosus with or without herniation of the nucleus pulposus, thinning of the disc, and disc degeneration with formation of osteophytes on the vertebral bodies. Disc herniation is exaggerated in magnitude for emphasis.

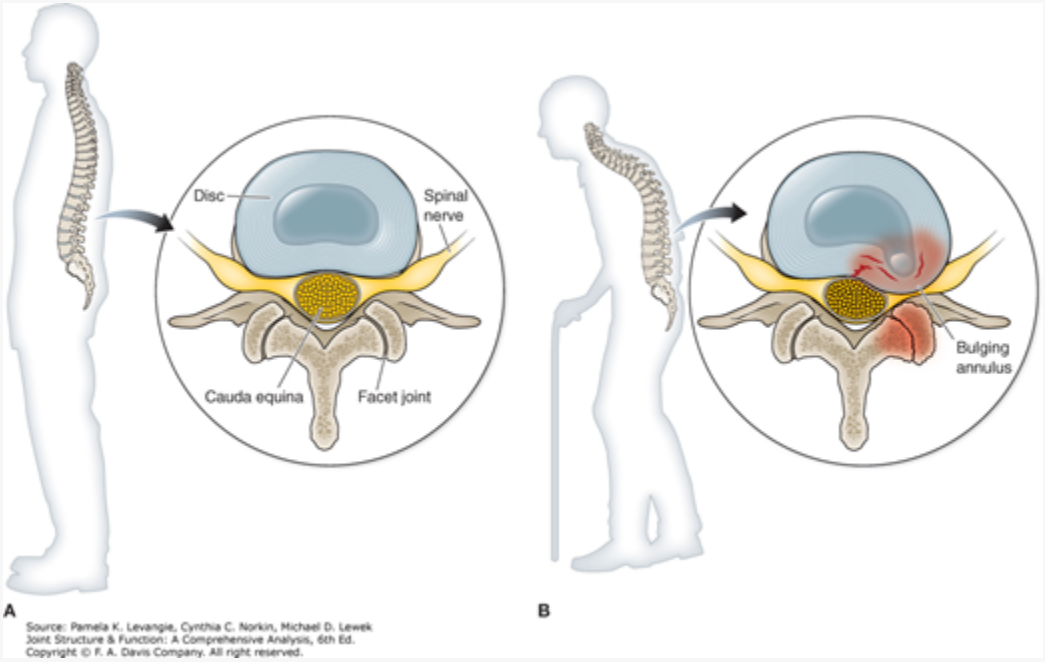


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FIGURE 4-75

Age-related changes to the spine. **A.** In the typical adult, the annulus fibrosus maintains the central location of the nucleus pulposus. The

spinal cord and spinal nerves have adequate space in the vertebral canal and intervertebral foramen, respectively. **B.** Age-related changes to the disc, vertebral ligaments like the ligamentum flavum, and to the zygapophyseal facets can lead to impingement of the spinal nerve or spinal cord.



The joints of Luschka, or uncovertebral joints (see Fig. 4–29), are frequent sites for age-related and degenerative changes. Osteophytes on the uncinate processes (see Fig. 4–28) occur predominantly in the lower segments, C5–C6 or C6–C7. The motion of lateral bending becomes extremely limited when these osteophytes occur.[80]

Summary

In summary, it is extremely important to understand the normal structure and function, including normal variability, of the vertebral column to understand the structures at risk

for injury and the best ways to treat people with dysfunction. Injury or failure occurs when the applied load is either insufficient or excessive. Repetitive strain causes injury by either the repeated application of a relatively low load or by application of a sustained load for a long duration (prolonged sitting or stooped posture).

The effects of injury, aging, disease, or development deficit on the vertebral column may be analyzed by taking the following points into consideration:

The normal function that the affected structure is designed to serve

The stresses that are present during normal situations

The anatomic relationship of the structure to adjacent structures

The functional relationship of the structure to other structures

Study Questions

1. Describe the functional goals of each of the following regions of the spinal column: the cervical spine, the thoracic spine, the lumbar spine, and the pelvis.
2. What are the components of a typical vertebra and what are the specific functions of each?
3. How do the shapes and sizes of the components of the vertebrae differ by region and how are the changes related to the forces to which each region is subject?
4. What are the roles of the intervertebral discs? Describe how the discs fulfill these

roles.

5. What are the motions that occur at the interbody joints with flexion, extension, lateral flexion, and rotation?

6. Describe the shape and orientation of the zygapophyseal joints in each spinal region. What motion is most facilitated in each region based on the shape and orientation of the facets?

7. What are the motions that occur at the zygapophyseal joints with flexion, extension, lateral flexion, and rotation? What is unique about the lumbar zygapophyseal joint motion?

8. Describe the relative strength of the longitudinal ligaments in the lumbar region. How does this strength differ from that in the other regions? Are some structures more or less susceptible to injury in this region on the basis of this variation?

9. Which structures are most affected if a person has a forward head posture? Describe the type and location of stresses that may occur, as well as how other structures might react to those changes in stresses.

10. Which structures are most affected if a person has an increased lumbar lordosis? Describe the type and location of stresses that may occur, as well as how other structures might react to the change in stresses.

11. Which muscles cause extension of the lumbar spine? In which position of the

spine are these muscles most effective?

12. Describe what kinds of forces act on the spine during motion and at rest.

13. Explain how creep may adversely affect the stability of the vertebral column.

14. Identify the prime movers of each spinal region.

15. Identify the muscles that function specifically to control segmental motion in the cervical and lumbar spine.

16. What role has been attributed to the thoracolumbar fascia in the stability of the lumbopelvic region?

17. Describe the dynamic and static restraints to anterior shear in the lumbar and cervical regions.

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Chapter 5: The Thorax and Chest Wall

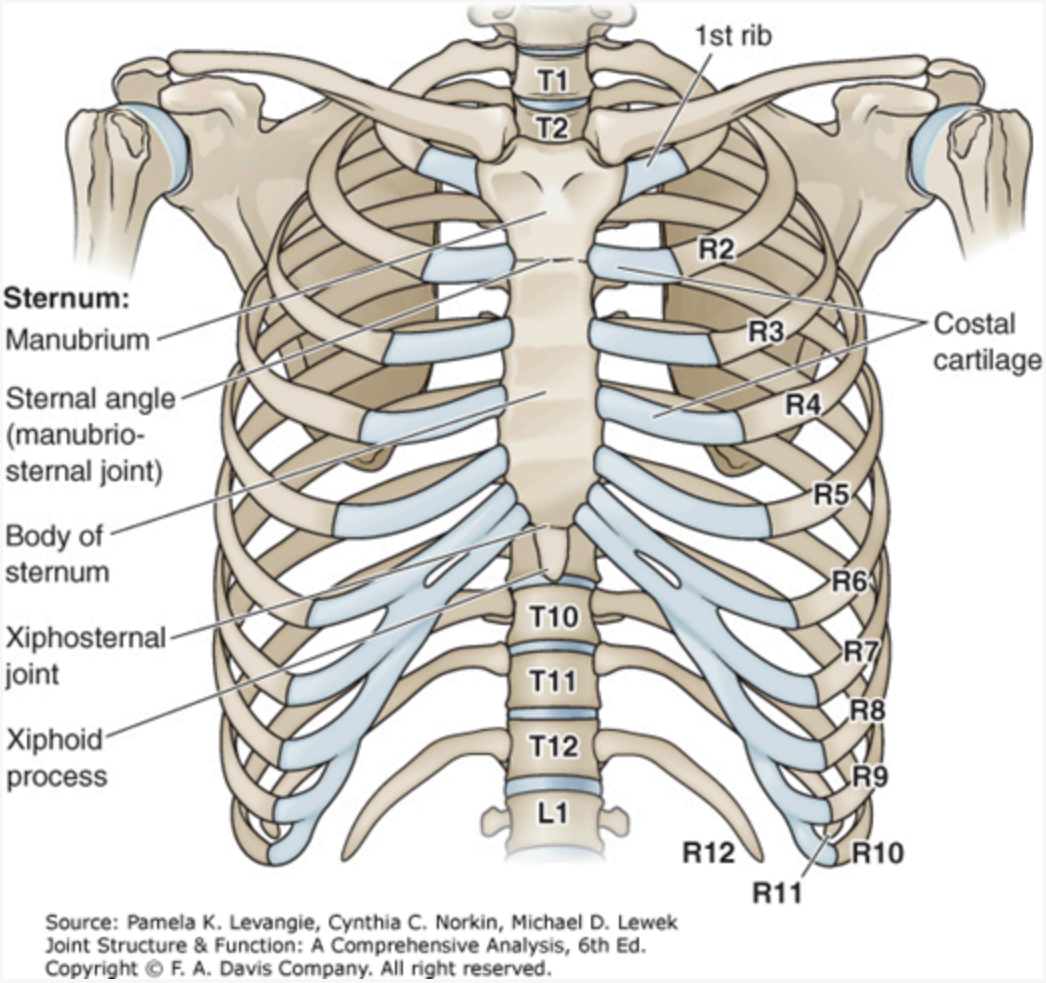
Julie Ann Starr; Diane Dalton

Introduction

Thorax is a term that is used to describe the bones of the rib cage, the fascia and muscles that attach to the rib cage, the visceral organs within the rib cage, and even the skin that covers the rib cage. The **rib cage,** also called the thoracic cage or the bony thorax, consists of the thoracic vertebrae, the ribs, and the sternum (Fig. 5–1). The rib cage provides a foundation for many of the muscle attachments of the upper extremities, head and vertebral column, and pelvis. The rib cage also provides protection for the heart, lungs, and viscera. Therefore, there needs to be a certain amount of inherent stability to the thorax. The structure of the rib cage significantly increases the stability of the thoracic spine during flexion/extension, lateral bending, and rotation.[1,2] One of the important functions of the chest wall is its role in ventilation. The process of ventilation, including inhalation/exhalation(inspiration/expiration), depends on the mobility of the rib cage and the ability of the muscles of ventilation to move it.[3]

FIGURE 5-1

Anterior view of the thorax including its component parts: the sternum, 12 pairs of ribs (R1–R12), the costocartilages, and the thoracic vertebrae.



Structure and Function of the Thorax

Structure

Structure of the Rib Cage

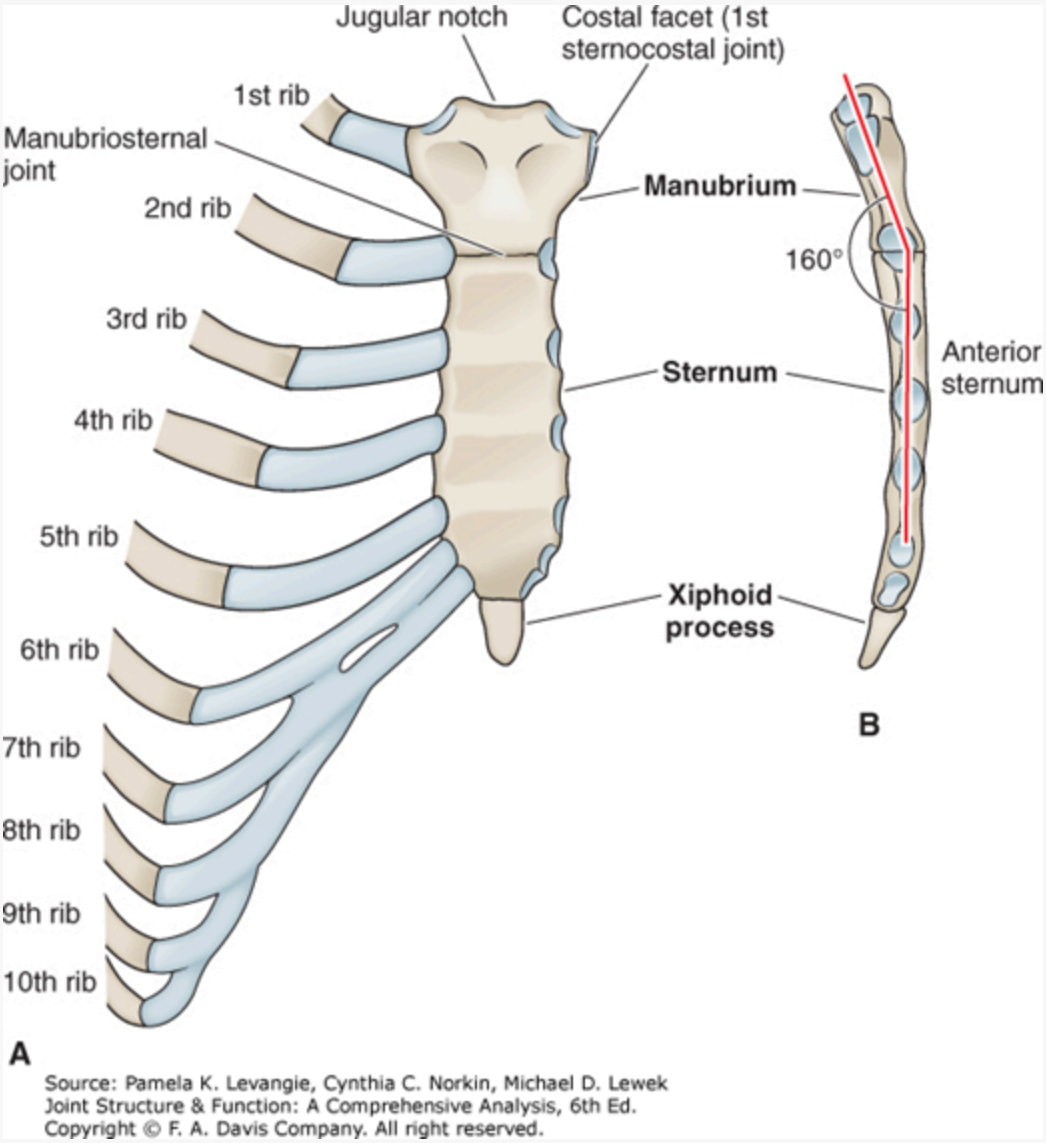
The rib cage forms a closed chain that involves many joints and muscles. The anterior border of the rib cage is the sternum, the lateral borders are the ribs, and the posterior border is formed by the thoracic vertebrae. The superior border of the rib cage is formed by the manubrium of the sternum, by the superior borders of the first costal cartilages, and by the first ribs and their contiguous first thoracic vertebra. The inferior

border of the rib cage is formed by the xiphoid process, the shared costal cartilages of ribs 7 through 10, the inferior portions of the 11th and 12th ribs, and the 12th thoracic vertebra (see Fig. 5–1).

The sternum is an osseous protective plate for the heart and is composed of the **manubrium, body of the sternum,** and **xiphoid process** (Fig. 5–2). The manubrium and the body of the sternum form a posteriorly concave angle of approximately 160°. The xiphoid process often angles posteriorly from the distal end of the body of the sternum and therefore may be difficult to palpate.

FIGURE 5-2

The sternum. **A.** The sternum is composed of the manubrium, the body of the sternum, and the xiphoid process. The costal notches for the chondrosternal joints are also evident in this anterior view. **B.** Viewed laterally, the dorsal (posterior) concavity of the manubrium and sternum is evident.

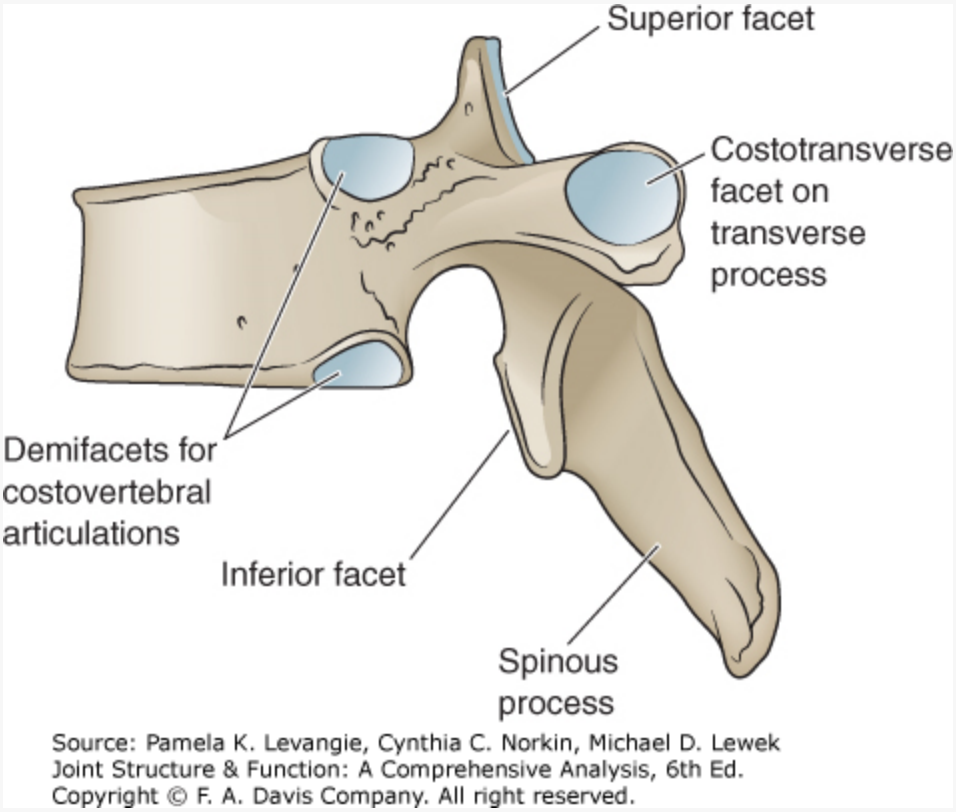


There are 12 thoracic vertebrae that make up the posterior portion of the rib cage. One of the unique aspects of the typical thoracic vertebra is that the vertebral body and transverse processes have six costal articular surfaces, four on the body (a **superior** and an **inferior costovertebral facet,** or **demifacet,** on each side), and one costotransverse facet on each of the two transverse processes (Fig. 5–3). The rib cage also includes 12 pairs of ribs. As shown in Figure 5–4, the ribs are curved flat bones (Fig. 5–4A) that gradually increase in length from rib 1 to rib 7 and then decrease in length from rib 8 to

rib 12. The posteriorly located head of each rib articulates with contiguous thoracic vertebral bodies, whereas the costal tubercles of ribs 1 to 10 articulate with the transverse processes of thoracic vertebrae (Fig. 5–4B). Anteriorly, ribs 1 to 10 are joined either directly or indirectly to the sternum through their costal cartilages (Fig. 5–5). Ribs 1 through 7 are classified as **vertebrosternal** (or **"true") ribs** because each rib, through its respective **costrochondral** and **chondrosternal** joints, attaches directly to the sternum. The costocartilages of ribs 8, 9, and 10 articulate with the costocartilage of the rib immediately above it, indirectly articulating with the sternum via rib 7. These ribs are classified as **vertebrochondral** (or **"false") ribs.** The 11th and 12th ribs are called **vertebral** (or **"floating") ribs** because they have no attachments to the sternum.

FIGURE 5-3

The costal facets on the typical thoracic vertebrae are found on the superior and inferior aspects of the posterior body (demifacets for the costovertebral articulations) and on the anterior transverse processes (facets for the costotransverse articulations), as shown in this.



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FIGURE 5-4

Articulation of the ribs with the vertebrae. **A.** A typical rib (ribs 2 through 9) is a curved flat bone. The posteriorly located head of this right 6th rib has superior and inferior facets (also known as demifacets) that articulate, respectively, with the right inferior demifacet of T5 and the right superior demifacet of T6 to form a single costovertebral joint. **B.** The facet on the costal tubercle of the 6th rib articulates with the transverse process of T6; the rib articulates anteriorly with the costal cartilage (via a costochondral joint) that then attaches to the sternum.

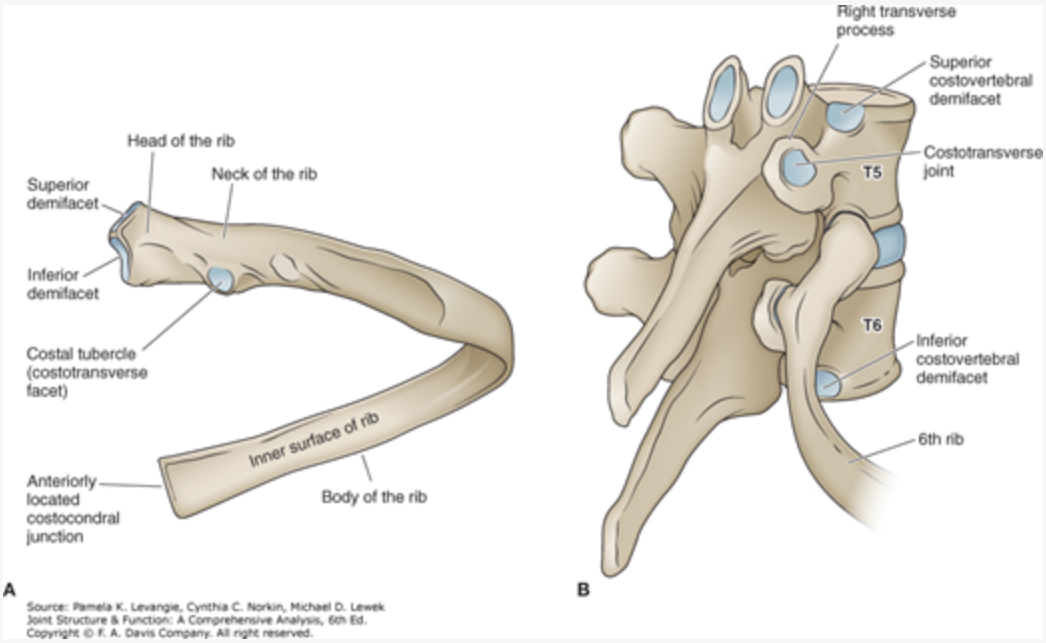
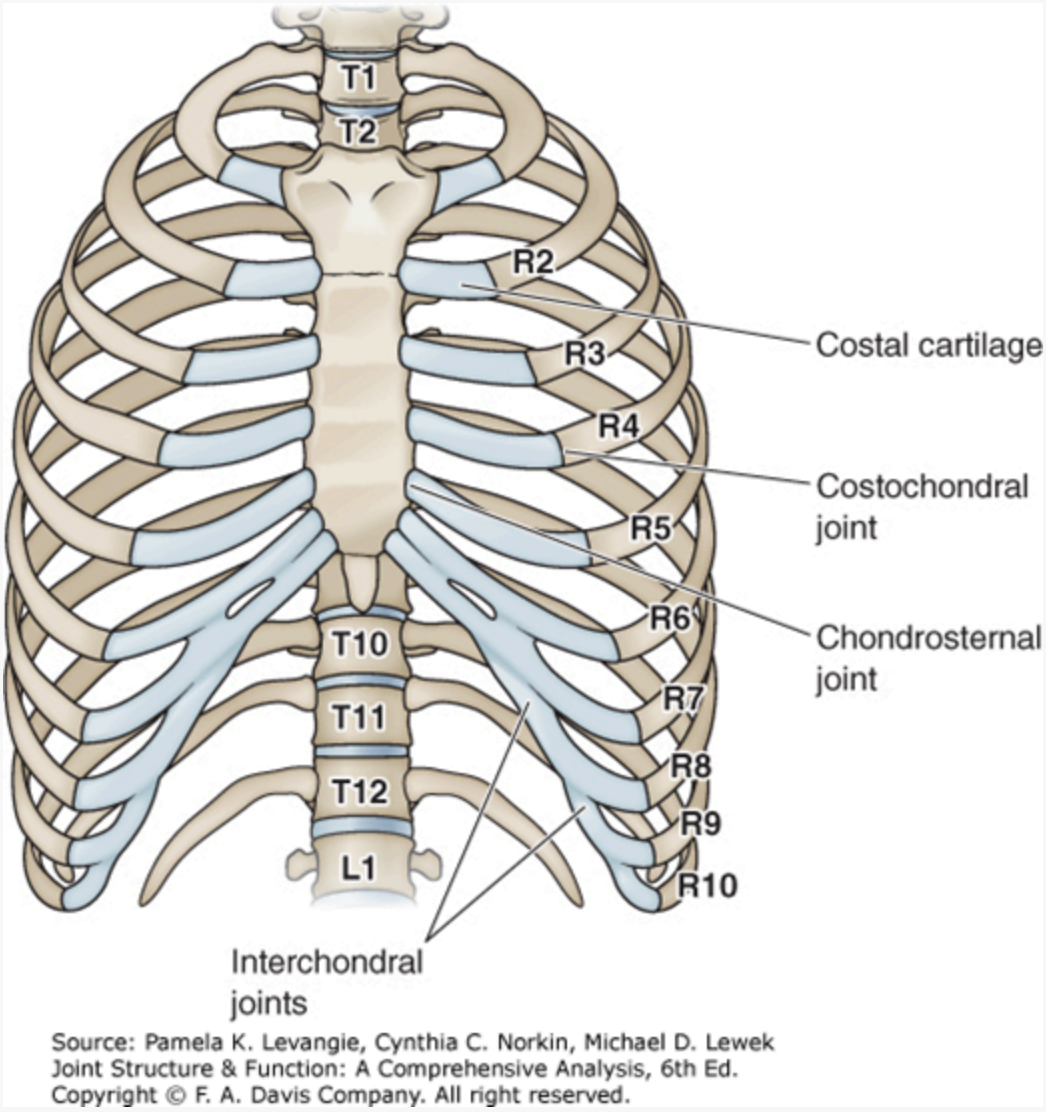


FIGURE 5-5

In this anterior view of the rib cage, the ribs articulate with the costal cartilages at the costochondral joints. The costal cartilages of the 1st through 7th ribs articulate directly with the sternum through the chondrosternal joints. The costal cartilages of the 8th through the 10th ribs articulate indirectly with the sternum through the costal cartilages of the adjacent superior rib at the interchondral joints.



Articulations of the Rib Cage

The articulations that join the bones of the rib cage include the **manubriosternal, xiphisternal,** costovertebral, costotransverse, costochondral, chondrosternal, and the **interchondral joints** (see Fig. 5–5).

MANUBRIOSTERNAL AND XIPHISTERNAL JOINTS

The manubrium and the body of the sternum articulate at the manubriosternal joint. This joint is also known as the sternal angle or the angle of Louis and is readily palpable

as a horizontal ridge at the level of the second ribs' anterior attachments. The manubriosternal joint is asynchondrosis and has a fibrocartilaginous disc between the articulating ends of the manubrium and the body of the sternum. Ossification of the manubriosternal joint occurs in persons older than 60 years.[4] The xiphoid process joins the inferior aspect of the sternal body at the xiphisternal joint, which is also asynchondrosis and tends to ossify by 40 to 45 years of age.[4]

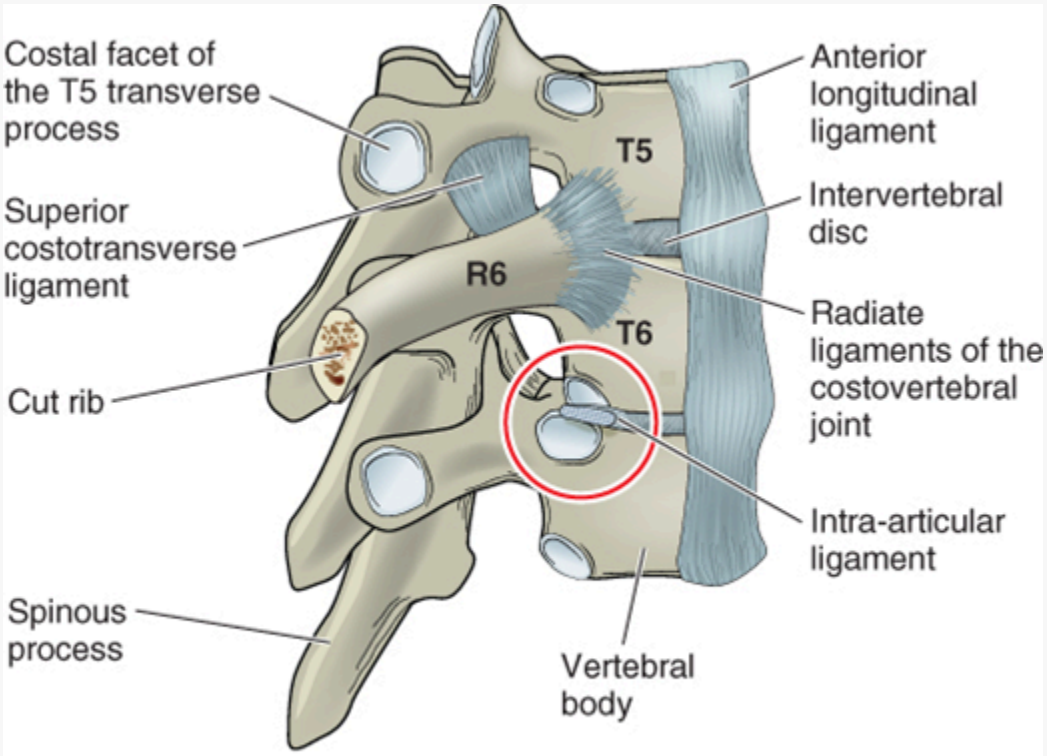
COSTOVERTEBRAL JOINTS

The typical costovertebral joint is a synovial joint formed by the head of the rib, two adjacent vertebral bodies, and the interposed intervertebral disc (see Fig. 5–4B). Ribs 2 through 9 and their respective thoracic vertebrae (T1–T9) have typical costovertebral joints, since the heads of these ribs have two articular facets, or so-called demifacets. The small, oval, and slightly convex demifacets of the ribs are called the superior and inferior costovertebral facets (see Fig. 5–4A). Adjacent thoracic vertebrae have facets corresponding to those on the heads of the ribs that articulate with them. The heads of ribs 2 through 9 fit snugly into the surface formed by the adjacent vertebral demifacets and the intervening disc. Each rib's superior facet articulates with the inferior facet of the vertebrae above it. Each rib's inferior facet articulates with the superior facet of its correspondingly numbered vertebrae. For example, the 6th rib's superior facet articulates with the vertebral body above (T5), and the rib's inferior facet articulates with the superior facet of the vertebral body below (T6) (see Fig. 5–4B). Ribs 1, 10, 11, and 12 are atypical ribs because they articulate with only one vertebral body and are numbered by that body. The costovertebral facets of T10 through T12 are located more posteriorly on the pedicle of the vertebra.

The typical costovertebral joint (Fig. 5–6) is divided into two cavities by the **interosseous** or **intra-articular ligament.** This ligament extends from the crest of the head of the rib to attach to the anulus fibrosus of the intervertebral disc. The **radiate ligament** is located within the capsule, with firm attachments to the anterolateral portion of the capsule. A fibrous capsule surrounds the entire articulation of each costovertebral joint.

FIGURE 5-6

A lateral view of the costovertebral joints and ligaments. The radiate ligament reinforces the costovertebral joints. The circled costovertebral joint is shown with the radiate ligament removed to reveal the intra-articular ligament that attaches the head of the rib to the annulus fibrosus.



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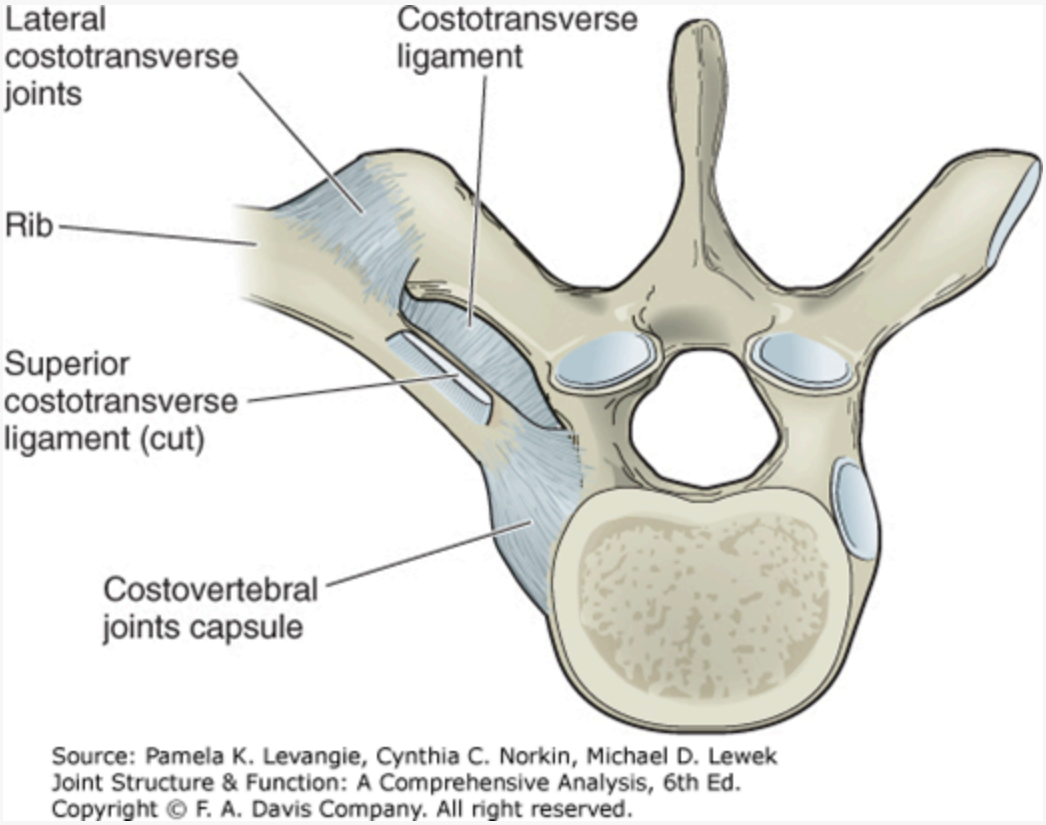
The atypical costovertebral joints of ribs 1, 10, 11, and 12 are more mobile than the other more typical costovertebral joints because the rib head articulates with only one vertebra. The interosseous ligament is absent in these joints; therefore, these each have only one cavity. The radiate ligament is present in these joints. Both rotation and gliding motions occur at each of the costovertebral joints.

COSTOTRANSVERSE JOINTS

The costotransverse joint is a synovial joint formed by the articulation of the costal tubercle of the rib with a costal facet on the transverse process of the corresponding vertebra (Fig. 5–7). There are 10 pairs of costotransverse joints joining vertebrae T1 through T10 with the rib of the same number. The costotransverse joints on T1 through approximately T6 have slightly concave costal facets on the transverse processes of the vertebrae and slightly convex costal tubercles on the corresponding ribs. This allows slight rotation movements between these segments. At the costotransverse joints of approximately T7 through T10, both articular surfaces are flat and gliding motions predominate. The transverse processes of T11 and T12 are shorter than the other thoracic vertebrae and do not have facets for articulations with their respective ribs.

FIGURE 5-7

A superior view of the costovertebral and costotransverse joints shows the capsuloligamentous structures. The vertically directed superior costotransverse ligament has been cut because it attaches to the cephalad transverse process.



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The costotransverse joint is surrounded by a thin, fibrous capsule. Three major ligaments support the costotransverse joint capsule. These are the **lateral costotransverse ligament,** the **costotransverse ligament,** and the **superior costotransverse ligament** (see Fig. 5–7). The lateral costotransverse ligament is a short, stout band located between the lateral portion of the costal tubercle and the tip of the corresponding transverse process. The costotransverse ligament is composed of short fibers that run within the costotransverse foramen between the neck of the rib posteriorly and the transverse process at the same level. The superior costotransverse ligament runs from the crest of the neck of the rib to the inferior border of the cephalad transverse process.

COSTOCHONDRAL AND CHONDROSTERNAL JOINTS

The costochondral joints are formed by the articulation of the 1st through 10th ribs anterolaterally with the costal cartilages (see Fig. 5–5). The costochondral joints are synchondroses. The periosteum and the perichondrium are continuous, giving support to the union. The costochondral joints have no ligamentous support and begin to calcify by age 35 years.[5]

The chondrosternal joints are formed by the articulation of the costal cartilages of ribs 1 through 7 anteriorly with the sternum. These joints also ossify with age. Rib 1 attaches to the lateral facet of the manubrium, rib 2 is attached via two demifacets at the manubriosternal junction, and ribs 3 through 7 articulate with the lateral facets of the sternal body. The chondrosternal joints of ribs 1, 6, and 7 are synchondroses. The chondrosternal joints of ribs 2 through 5 are synovial joints.

The chondrosternal joints of ribs 1 through 7 (see Fig. 5–5) have capsules that are continuous with the periosteum and support the connection of the cartilage as a whole. Ligamentous support for the capsule includes **anterior** and **posterior radiate costosternal ligaments.** The **sternocostal ligament** is an intra-articular ligament, similar to the intra-articular ligament of the costovertebral joint that divides the two demifacets of the second chondrosternal joint. The **costoxiphoid ligament** connects the anterior and posterior surfaces of the seventh costal cartilage to the front and back of the xiphoid process.

INTERCHONDRAL JOINTS

The costal cartilages of ribs 7 through 10 each articulate with the cartilage immediately above them to form interchondral joints. For ribs 8 through 10, this articulation forms

the rib's only connection to the sternum, albeit indirectly (see Fig. 5–5). The interchondral joints are synovial joints and are supported by a capsule and interchondral ligaments. The interchondral articulations, like the chondrosternal joints, tend to fuse with age.

Foundational Concepts 5–1

Summary of the Rib Cage

- Ribs 1 through 10 articulate posteriorly with the vertebral column by two synovial joints (the costovertebral and costotransverse joints) and anteriorly through the costocartilages, either directly or indirectly, to the manubriosternum. These joints form a closed chain in which the segments are interdependent and motion is restricted. These articulations and their associated ligaments give the thoracic cage the stability necessary to protect internal organs and yet provide enough flexibility to maximize function.
- Ribs 11 and 12 have a single costovertebral joint, no costotransverse joint, and no attachment anteriorly to the sternum. These last two ribs form an open chain so that motion of these ribs is less restricted.

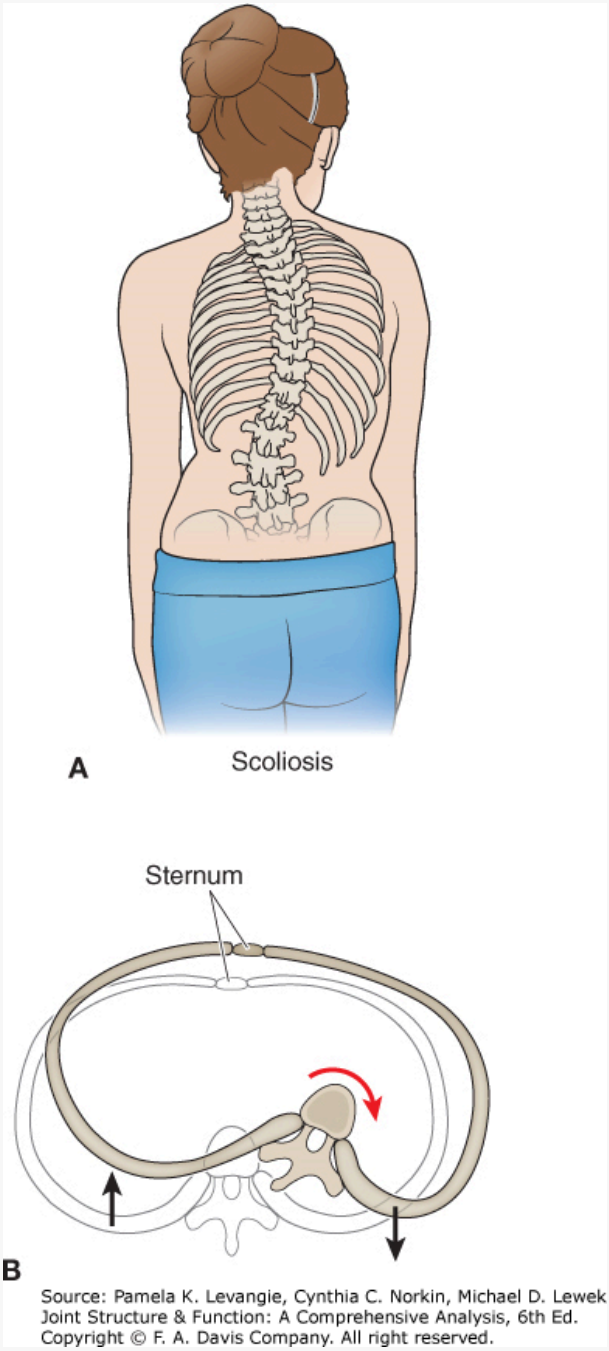
Patient Application 5–1

Joanne Mann is a 14-year-old female who has just been diagnosed with a right thoracic, left lumbar idiopathic scoliosis, a pathological lateral curvature of the spine without a known underlying cause. As is often the case, Joanne's scoliosis is associated with substantial rotation of the thoracic vertebrae (Fig. 5–8).[6] A right thoracic scoliosis is named for the side of the convexity of the vertebral curve and is a left lateral flexion of the thoracic spine. As in Joanne's case, an idiopathic right thoracic curvature is most often accompanied by some degree of left lumbar curvature (lateral flexion of the lumbar vertebrae to the right). Joanne's vertebral rotation, as is typically found in a right thoracic scoliosis, caused the bodies of the vertebrae to rotate to the right (spinous processes rotated to the left), and the right transverse processes of the vertebrae to rotate posteriorly, carrying the right ribs with them. Although the amount of lateral curvature and amount of vertebral rotation are not necessarily proportional, the vertebral rotation is responsible for the classic right posterior rib hump of scoliosis that Joanne demonstrates (Fig. 5–9). On the concave

side of Joanne's scoliotic curve, the effects are just the opposite. The left transverse processes of the vertebrae move anteriorly, bringing the articulated ribs forward. The rib distortion that results from the vertebral rotation is evident bilaterally. These musculoskeletal abnormalities limit the range of motion of the rib cage. Given the limitation in rib cage range of motion and the potential distortion in the symmetry of the pleural space, Joanne will be assessed for any impairment to pulmonary function that may result from her current curvature.

FIGURE 5-8

A. A right thoracic, left lumbar scoliosis (named for the region and side of the convexity) shows the curvature of the vertebrae and the asymmetrical spacing of the ribs (separation on the right and crowding on the left) that results from right rotation of the involved thoracic vertebrae. **B.** The bodies of the thoracic vertebrae in a right thoracic scoliosis typically rotate to the right, resulting in posterior displacement of the right transverse process and the attached right ribs, as well as anterior displacement of the opposite transverse processes and left ribs.

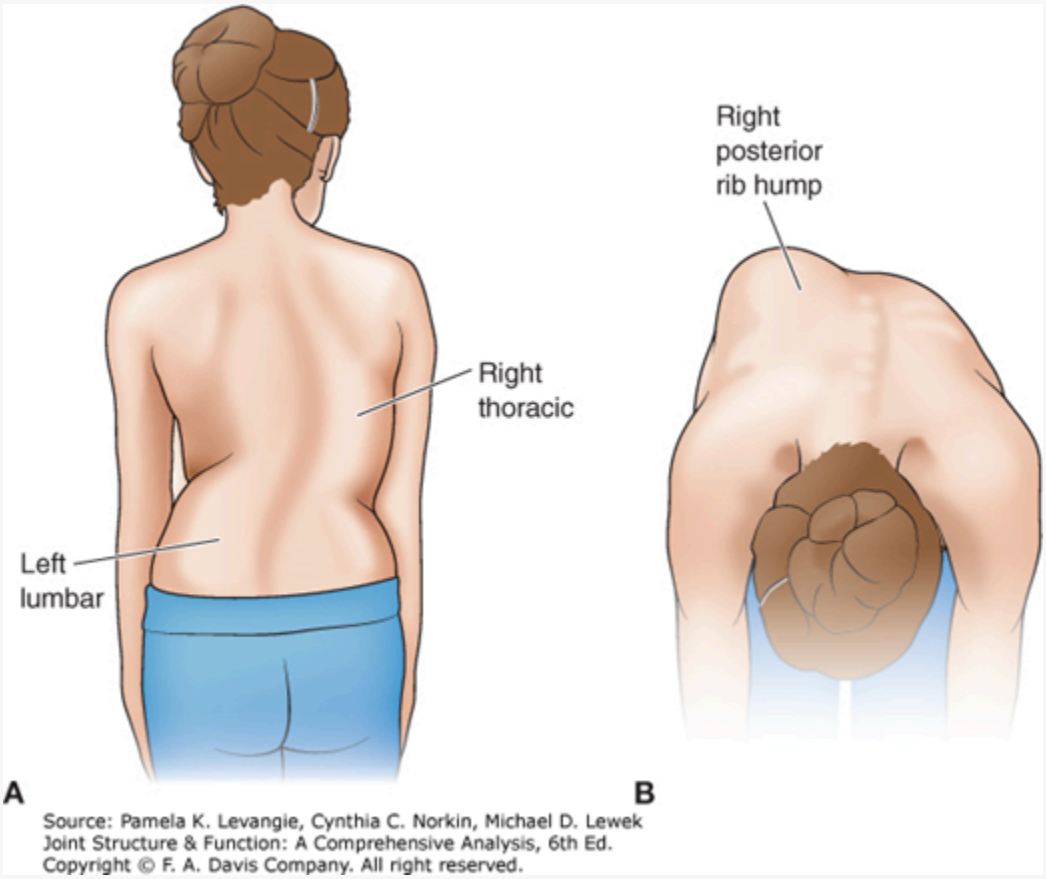


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FIGURE 5-9

Scoliosis. **A.** From a posterior perspective in the upright position, the right thoracic and left lumbar curvature can be seen. **B.** The amount of

rib rotation is often not appreciable until the subject does a forward bend.



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Function

Kinematics of the Ribs and Manubriosternum

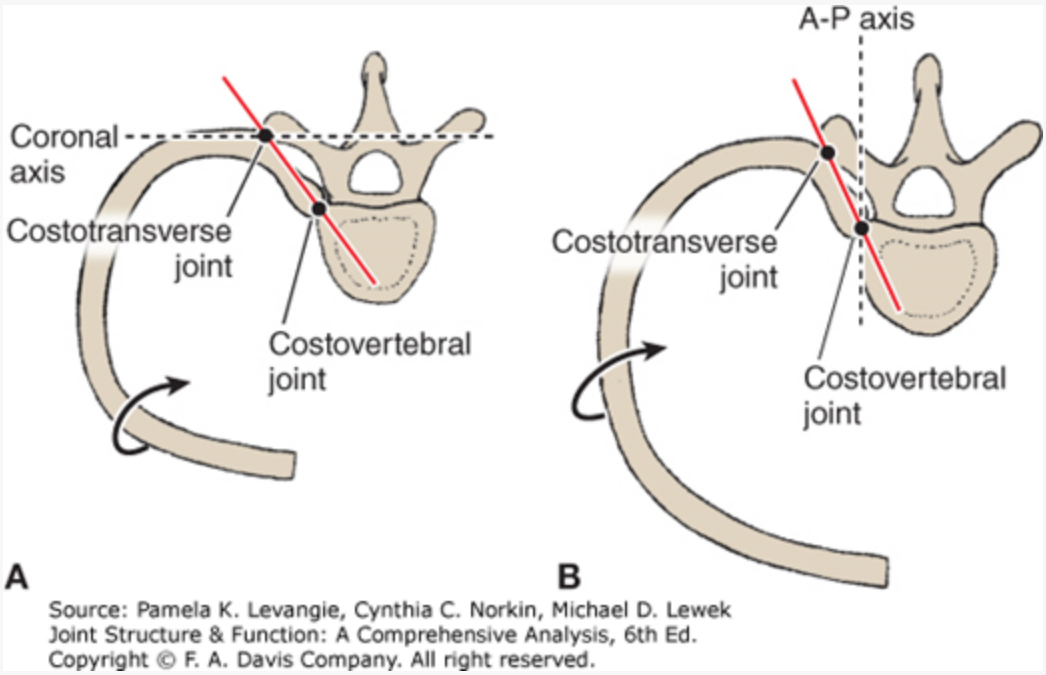
The movement of the rib cage is a combination of complex geometrics that are governed by (1) the types and angles of the articulations, (2) the movement of the manubriosternum, and (3) the elasticity of the costal cartilages.

The costovertebral and costotransverse joints are mechanically linked, with a single axis of motion for elevation and depression passing through the centers of both joints (Fig. 5–10).[3] The length, shape, and downward angle of each rib is unique; therefore, the axis

of rotation for each rib is slightly different. The axes of rotation for the upper ribs most closely approximate a coronal axis (Fig. 5–10A), allowing the motion of those ribs to occur predominantly in the sagittal plane. The axes of rotation for the lower ribs most closely approximate an anteroposterior (A-P) axis (Fig. 5–10B), allowing the motion of those ribs to be closer to the frontal plane. The axes of rotation for ribs 11 and 12 pass through the costovertebral joint only, because there is no joint present between ribs 11 and 12 and the associated transverse processes. The axes of rotation for these last two ribs also approximate a coronal axis. The ribs not only articulate with the vertebrae posteriorly but also, for most of the ribs, with the costal cartilages anteriorly, forming a closed chain. Movement around the costovertebral and costotransverse joints, therefore, also creates movement at the costochondral and chondrosternal joints.

FIGURE 5-10

A. The common axis of motion for the upper ribs passes through the centers of the costovertebral and costotransverse joints and most closely approximates a coronal axis. **B.** The axis through the costovertebral and costotransverse joints for the lower ribs more closely approximates an A-P axis.



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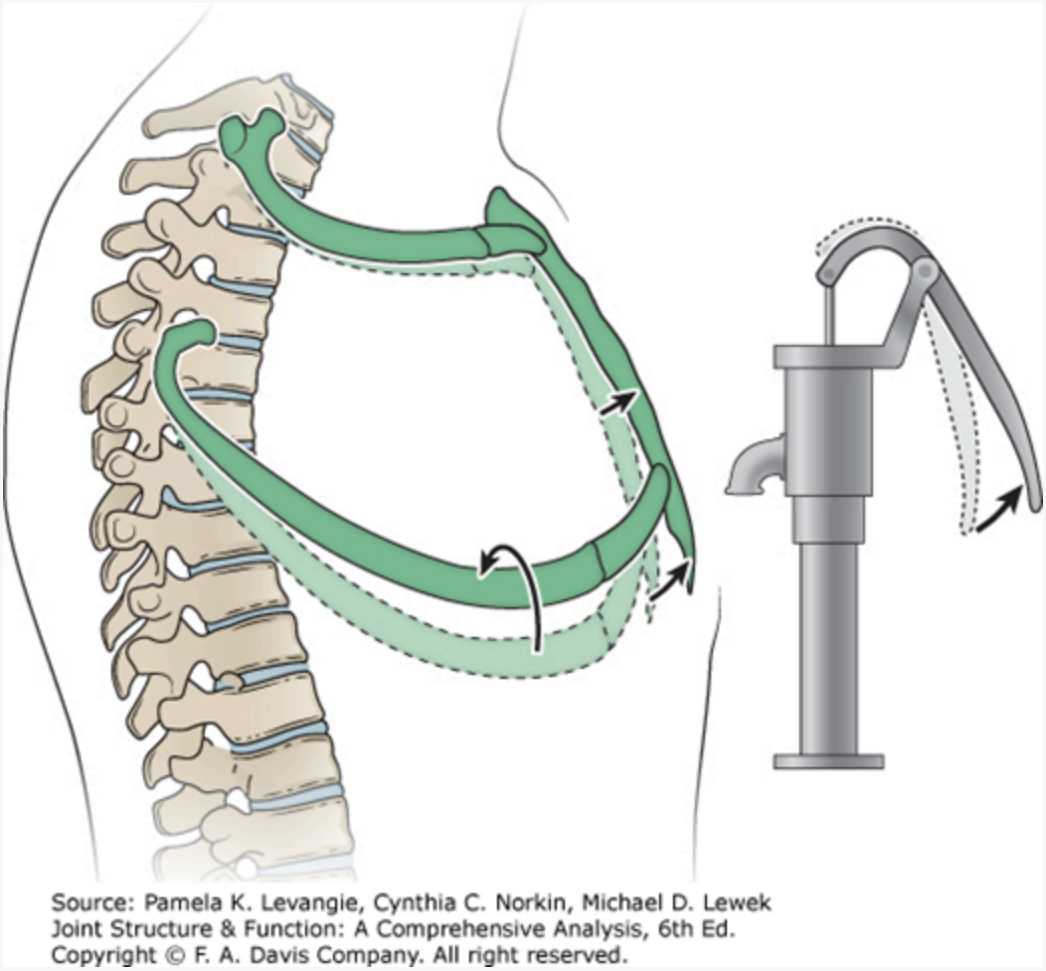
The first rib is unique because its anterior articulation is larger and thicker than that of any other rib. The first costal cartilage is stiffer than the other costal cartilages. Also, the first chondrosternal joint is cartilaginous (synchondrosis), not synovial, and therefore is firmly attached to the manubrium. Finally, the first chondrosternal joint is just inferior and posterior to the sternoclavicular joint. For these reasons, there is very little movement of the first rib at its attachment on the manubrium. Posteriorly, the costovertebral joint of the first rib has a single facet, which increases the mobility of that joint. Mobility of the costovertebral joints will be seen to be important in inhalation, in this instance so the first rib can elevate.

Ribs 2 through 7 that are attached to the body of the sternum increase in length and mobility the more caudal the rib. In the upper ribs (2 through 7), most of the movement occurs at the anterior aspect of the rib, given the nearly coronal axis at the vertebrae. The costocartilage rotates upward, becoming more horizontal with inhalation. The

movement of the ribs pushes the sternum anteriorly and superiorly (Fig. 5–11). The manubrium moves less than the body of the sternum because the shortest and least mobile first rib is attached to the manubrium. The increased movement of the body of the sternum in relation to the movement of the manubrium results in slight movement at the manubriosternal joint. The greatest effect of the motion of the upper ribs and sternum is the increase in the AP diameter of the thorax. This combined rib and sternal motion that occurs in a predominantly sagittal plane has been termed the **"pump-handle" motion** of the thorax.

FIGURE 5-11

Elevation of the upper ribs at the costovertebral and costotransverse joints results in anterior and superior movement of the sternum (and accompanying torsion of the costal cartilages) as well as an increased anteroposterior diameter of the thorax. This rib motion is referred to as the "pump-handle" motion of the thorax.

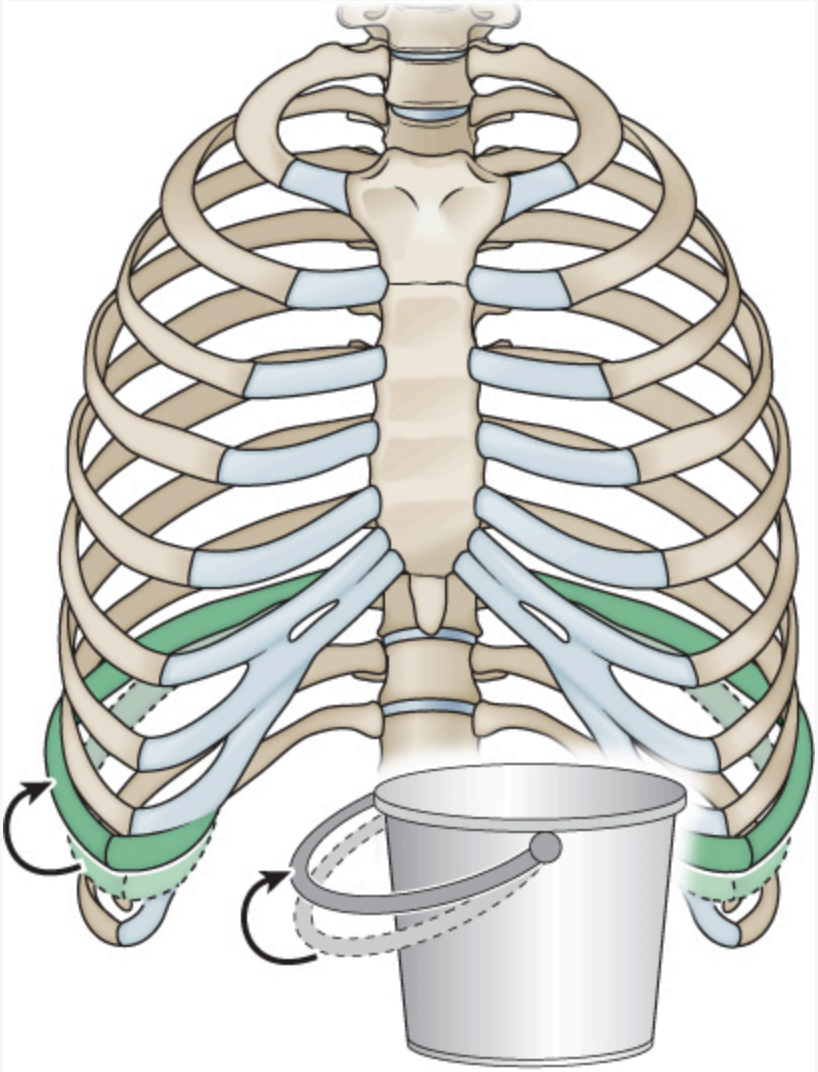


Elevation of ribs 8 through 10 occurs about an axis of motion lying more toward the sagittal plane. The lower ribs have a more angled shape (downward obliquity increases from rib 1 to rib 10) and an indirect attachment anteriorly to the sternum. These factors allow the lower ribs more motion at the lateral aspect of the rib cage. The greatest effect of the upward rotation and elevation of the lower ribs is the increase in the transverse diameter of the lower thorax. This motion that occurs in a more frontal plane has been termed the **"bucket-handle" motion** of the thorax (Fig. 5–12). There is a gradual shift in the orientation of the ribs' axes of motion from cephalad to caudal; therefore, the intermediate ribs function in a transitional zone, with qualities of both pump-handle

and bucket-handle types of motion.[7] The 11th and 12th ribs each have only one posterior articulation with a single vertebra and no anterior articulation to the sternum; therefore, they do not participate in the closed chain motion of the thorax.

FIGURE 5-12

Elevation of the lower ribs at the costovertebral and costotransverse joints results in a lateral motion of the rib cage, referred to as “bucket-handle” motion of the thorax.



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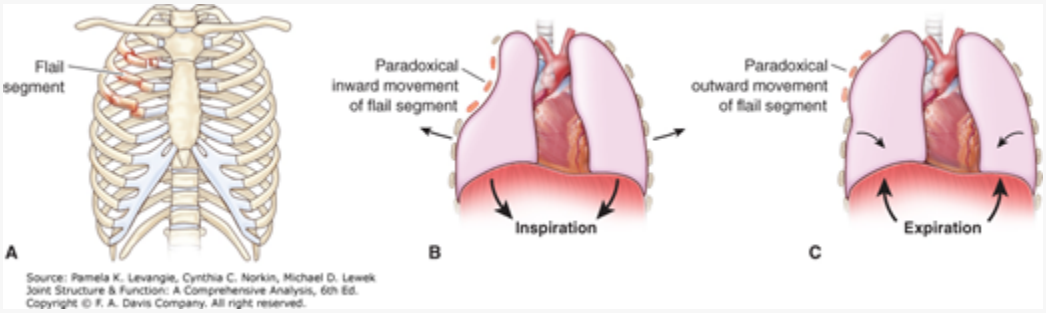
Patient Application 5–2

Henry Olsson is an 82-year-old who recently had a serious fall at home. He missed the last step leading to his driveway and fell sideways into the fender of his car. He experienced immediate severe pain in his ribs and was having problems breathing. In the emergency department, Henry was told that he broke several ribs in several places, resulting in a flail chest with a pulmonary contusion. When Henry tried to take a breath in, the broken segment of ribs that was detached from the rest of the ribcage by the fractures actually went inward rather than outward as one might expect (Fig.

5–13). Ordinarily, the chest wall is pulled upward and outward into an inhalation mode by the respiratory muscles; this increase in thoracic volume causes a resultant drop in intrapulmonary pressure. Henry's flail segment, without its usual connections, was "sucked" inward by that subatmospheric pressure (Fig. 5–13A). During exhalation when the rib cage ordinarily drops back down into an exhalation mode, Henry's decreased thoracic volume increased the intrapulmonary pressure, which forced the flail section outward (Fig. 5–13B). This paradoxical motion of the flail segment was the result of the disruption of the closed chain. Henry was admitted to the hospital for management of his ventilatory dysfunction.

FIGURE 5-13

A flail chest results from the fracture of two or more ribs in two or more places. The flail segment is no longer attached to the rest of the thorax, disrupting the closed chain action of the ribs. **A.** During inspiration, the ribs are lifting up and out while the flail segment is pulled inward. **B.** During expiration, the ribs are moving down and in while the flail segment moves outward.



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Muscles Associated With the Rib Cage

The muscles that act on the rib cage are generally referred to as the **ventilatory muscles.** The ventilatory muscles are striated skeletal muscles that differ from other skeletal muscles in a number of ways: (1) they have increased fatigue resistance and greater

oxidative capacity; (2) they contract rhythmically throughout life rather than episodically; (3) they work primarily against the elastic properties of the lungs and airway resistance rather than against gravitational forces; (4) neurological control of these muscles is both voluntary and involuntary; and (5) the actions of these muscles are life-sustaining.

Any muscle that attaches to the chest wall has the potential to contribute to ventilation. The recruitment of muscles for ventilation is related to the type of breathing being performed.[8–10] In breathing at rest, only the primary inspiratory muscles are needed for ventilation. During active or forced breathing that occurs, for example, with increased activity or pulmonary pathologies, accessory muscles of both inhalation and exhalation are recruited to help meet the increased demand for ventilation.



Foundational Concepts 5–2

Measures of Lung Volume and Capacity

All the air that is housed in the lungs can be divided into four segments. **Tidal volume** is the amount of air that is moved in or out of the thorax during a resting breath. **Inspiratory reserve volume** is the amount of air that can be inhaled after a resting breath has already been inhaled. **Expiratory reserve volume** is the amount of air that can be exhaled after a resting breath has already been exhaled. Finally, **residual volume** is the amount of air that cannot be voluntarily exhaled no matter how hard someone tries.

The term **capacity** is used when two or more lung volumes are added together. For example, **vital capacity** is a combination of inspiratory reserve volume, tidal volume, and expiratory reserve volume. Vital capacity is the volume of air that can be blown out of the lungs from a full inhalation to a full exhalation. **Inspiratory capacity** is a combination of inspiratory reserve volume and tidal volume; it is the volume of air that can be breathed in from resting exhalation. **Functional residual capacity** is a combination of expiratory reserve volume and reserve volume; it is the volume of air

that remains in the lungs after an exhalation at rest. Total lung capacity is a combination of all four lung volumes. Table 5–1 summarizes the definitions graphically.

Table 5-1

Lung Volumes and Lung Capacities

The ventilatory muscles can be classified as either **primary** or **accessory muscles of ventilation.** A muscle's action during the ventilatory cycle is neither simple nor absolute, which makes the categorizing of ventilatory muscles as either inspiratory muscles or expiratory muscles inaccurate and misleading.

PRIMARY MUSCLES OF VENTILATION

The primary muscles of ventilation are those recruited for ventilation at rest. These include the **diaphragm,** the **intercostal muscles** (particularly the **parasternal muscles** and some of the **external intercostals),** and the **scalene muscles.**[9,10] These muscles all act on the rib cage to promote inhalation. There are no primary muscles for exhalation at rest because it is a passive function.

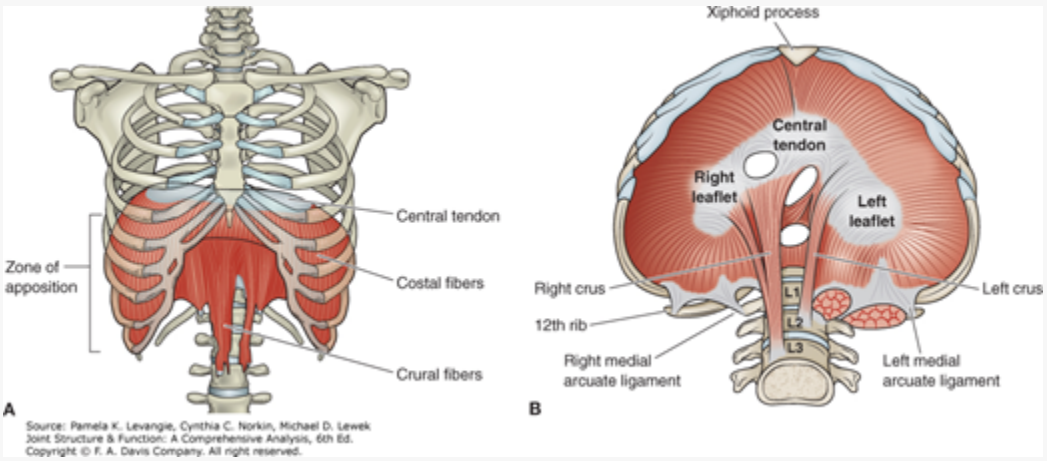
DIAPHRAGM

The diaphragm is the primary muscle of ventilation, accounting for approximately 70% to 80% of inhalation force during breathing at rest.[9] The diaphragm (**Fig. 5–14**) is composed of a circular set of muscle fibers that arise from the sternum, ribs, and vertebral bodies. The fibers travel cephalad (superiorly) from their origin to insert into a

central tendon.[11] There is no bony insertion site, making the diaphragm unique. The boomerang-shaped central tendon of the diaphragm forms the top of the domes of the right and left hemidiaphragms. Functionally, the muscular portion of the diaphragm is divided into posterior and anterior segments. The anterior segment, called the **costal fibers,** arises from the sternum and ribs. The posterior segment of the diaphragm, termed the **crural fibers,** arises from the vertebral bodies (Fig. 5–14A).[12]

FIGURE 5-14

A. In an anterior view, the fibers of the diaphragm can be seen arising from the sternum, costocartilages, and ribs (costal fibers) and from the vertebral bodies (crural fibers). The costal fibers run vertically upward from their origin in close apposition to the rib cage and then curve and become more horizontal before inserting into the central tendon. **B.** An inferior view of the diaphragm shows the leaflets of the central tendon, the right crus, left crus, and the medial and lateral arcuate ligaments bilaterally.



The costal fibers of the diaphragm originate from muscular slips attached to the

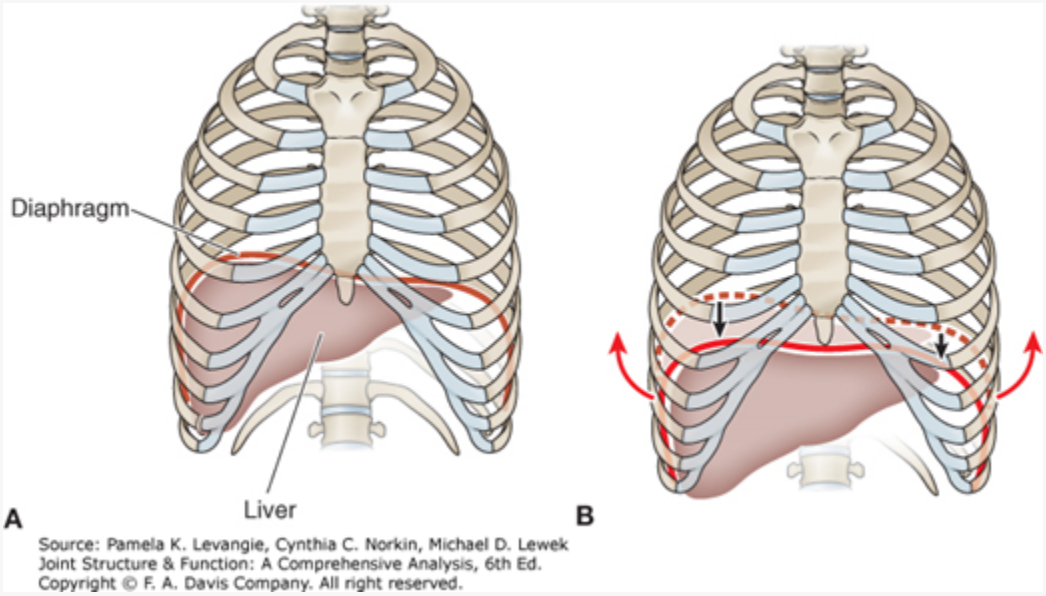
posterior aspect of the xiphoid process, the inner surfaces of the lower six ribs, and the costal cartilages of those ribs. The costal fibers of the diaphragm run vertically from their origin, in close apposition to the rib cage, and then curve to become more horizontal before inserting into the central tendon. The vertically traveling costal fibers of the diaphragm, which lie close to the inner wall of the lower rib cage, form a functional unit called the **zone of apposition** (see Fig. 5–14A).[12]

The crural fibers of the diaphragm originate from two tendinous structures, called the **right** and **left crus,** and from the right and left aponeurotic **medial and lateral arcuate ligaments** (**Fig. 5–14B**). The right and left crus are tendons that arise from the L1 to L3 vertebrae. The tendons pass anteriorly and medially, joining together to form an arch just anterior to the aorta. The medial arcuate ligament extends from L1 or L2, covers the psoas muscles, and returns to the transverse processes of L1, L2, or L3. The lateral arcuate ligament extends from the transverse process of L1, L2, and L3, arches up and over the quadratus lumborum muscles, and returns to the caudal margin of the 12th rib.[13]

During **tidal breathing** (**Fig. 5–15**), the fibers in the zone of apposition of the diaphragm contract, causing a descent (but only a slight change in contour) of the dome of the diaphragm. As the dome descends, the abdominal contents are compressed, which increases the intra-abdominal pressure.[12] The compressed abdominal contents stabilize the central tendon of the diaphragm so that a continued contraction of the costal fibers of the diaphragm against the stabilized central tendon cause the lower ribs to lift and rotate outwardly in the bucket-handle motion (see Fig. 5–12).[14]

FIGURE 5-15

Tidal breathing. **A.** Before a new inhalation is initiated, the diaphragm is domed. **B.** During inhalation, the diaphragm contracts, the dome descends from its resting position, compressing the abdominal contents. The resulting increase in intra-abdominal pressure limits further descent of (i.e., stabilizes) the central tendon of the diaphragm so that continued contraction of the costal fibers of the diaphragm on the stabilized central tendon results in expansion (bucket-handle motion) of the lower ribs.



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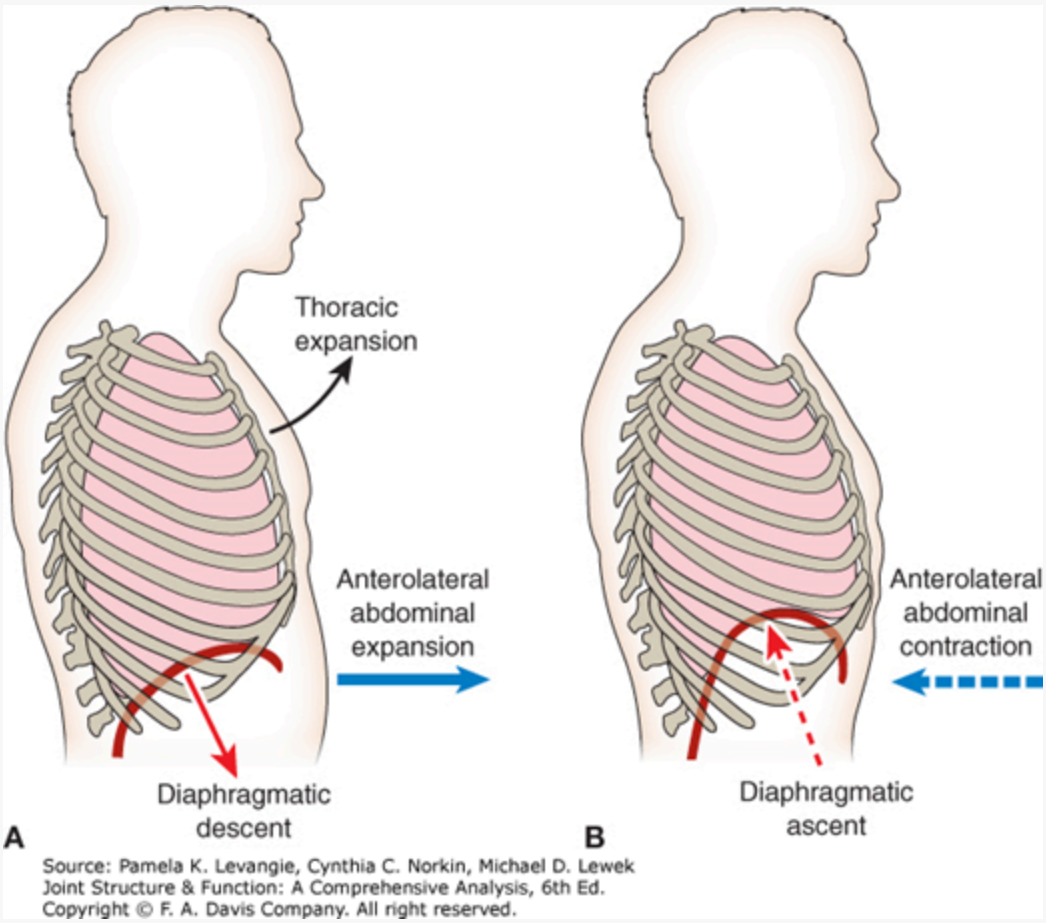
The crural fibers of the diaphragm have an inspiratory effect on the central tendon of the diaphragm, but, unlike the costal fibers, a less direct effect on the lower rib cage.[3,12] The action of the crural fibers of the diaphragm results in descending of the central tendon that increases intra-abdominal pressure. This increased pressure is transmitted across the apposed diaphragm to help the costal fibers of the diaphragm expand the

lower rib cage.[3,12]

The thoracoabdominal movement during inhalation at rest is a result of the pressures that are generated by the contraction of the diaphragm (Fig. 5–16). When the diaphragm contracts and the central tendon descends, there is an increase in volume within the thorax and a resultant decreased intrapulmonary pressure that is responsible for air entering the thorax, that is, inhalation. There is also an increase in abdominal pressure, which causes the abdominal contents to be displaced anteriorly and laterally (Fig. 5–16A). The abdominal muscles are active, when standing or sitting, to offer some counterbalance to this increase in abdominal pressure.[14] When active muscle contraction of the diaphragm ceases, the domes return to their higher resting position within the thorax. As thoracic volume decreases, intrapulmonary pressure increases, and, as a result, exhalation occurs. The abdominal contents return to their starting position (Fig. 5–16B).

FIGURE 5-16

Quiet inspiration and expiration. **A.** With quiet inspiration, the normal thoracoabdominal movement is caused by contraction of the diaphragm. The diaphragm descends, increasing thoracic size and displacing the abdominal viscera anteriorly and laterally. **B.** With passive exhalation, the thorax decreases in size, and the abdominal viscera return to their resting position.



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Expanded Concepts 5–1

Abdominal Compliance

Compliance (inverse of stiffness) is a measurement of the extensibility or distensibility of a structure or system. During diaphragm contraction, the abdominal cavity becomes the fulcrum for lateral expansion of the rib cage. Therefore, compliance of the abdomen is a factor in the inspiratory movement of the thorax.

Thoracic compliance = Δ volume/Δ pressure
Thoracic compliance = change in volume per unit of pressure

Patient Application 5–3

Hannah Rosen is a 16-year-old who sustained a C6 complete spinal cord injury when she dove into a pond and hit her head on a submerged tree trunk. Hannah's

abdominal musculature is no longer innervated because all muscular function below the level of cervical injury was lost. Although Hannah's diaphragm is fully innervated and functioning, her pulmonary function is still impaired compared with other individuals her age.

When an individual sustains a spinal cord injury at or above the mid-thoracic level, loss of innervation to the intercostal and abdominal musculature affects the process of ventilation in many ways. Although the loss of abdominal musculature (and intercostals) will affect both inhalation and exhalation, the loss of abdominal musculature affects the function of the intact diaphragm. Without intact abdominal musculature, there is increased compliance (distensibility) of the abdomen. Without the ability of the abdominal muscles to control anterior displacement of the abdominal contents, the central tendon of the diaphragm will not stabilize against the abdominal viscera, and the costal fibers of the diaphragm cannot lift the lower ribs upward and outward. The ability to inhale, as measured by vital capacity, is decreased. One strategy to help Hannah optimize her diaphragmatic function is to have her wear an elastic abdominal binder when she is sitting. The abdominal binder will replace some of the resistance to expansion that the abdominal muscles previously provided. Reducing the excessive compliance facilitates improved, although not normalized, stabilization of the central tendon of the diaphragm, allowing the costal fibers to act on the lower ribs during inhalation.

Brittany Ludke is a 22-year-old woman in her 8th month of pregnancy. She is now noticing that she is getting a bit short of breath when she negotiates her usual two flights of stairs to her office at work. She assumed that it was due to her weight gain but her obstetrician told her that it wasn't that as much as it was the increased size of the fetus. The obstetrician explained that the presence of the growing baby decreased compliance of her abdomen, which limits the caudal diaphragmatic excursion in inhalation. The presence of the fetus stabilizes the diaphragm earlier in the ventilatory cycle and causes earlier lateral and upward motion of the lower rib cage during inhalation. These changes can limit Brittany's vital capacity that she would usually tap into with increased exertion. The weight that Brittany gained with the baby's recent growth increased her respiratory challenge as she ascends stairs while, at the same time, her ability to meet that challenge has been reduced.

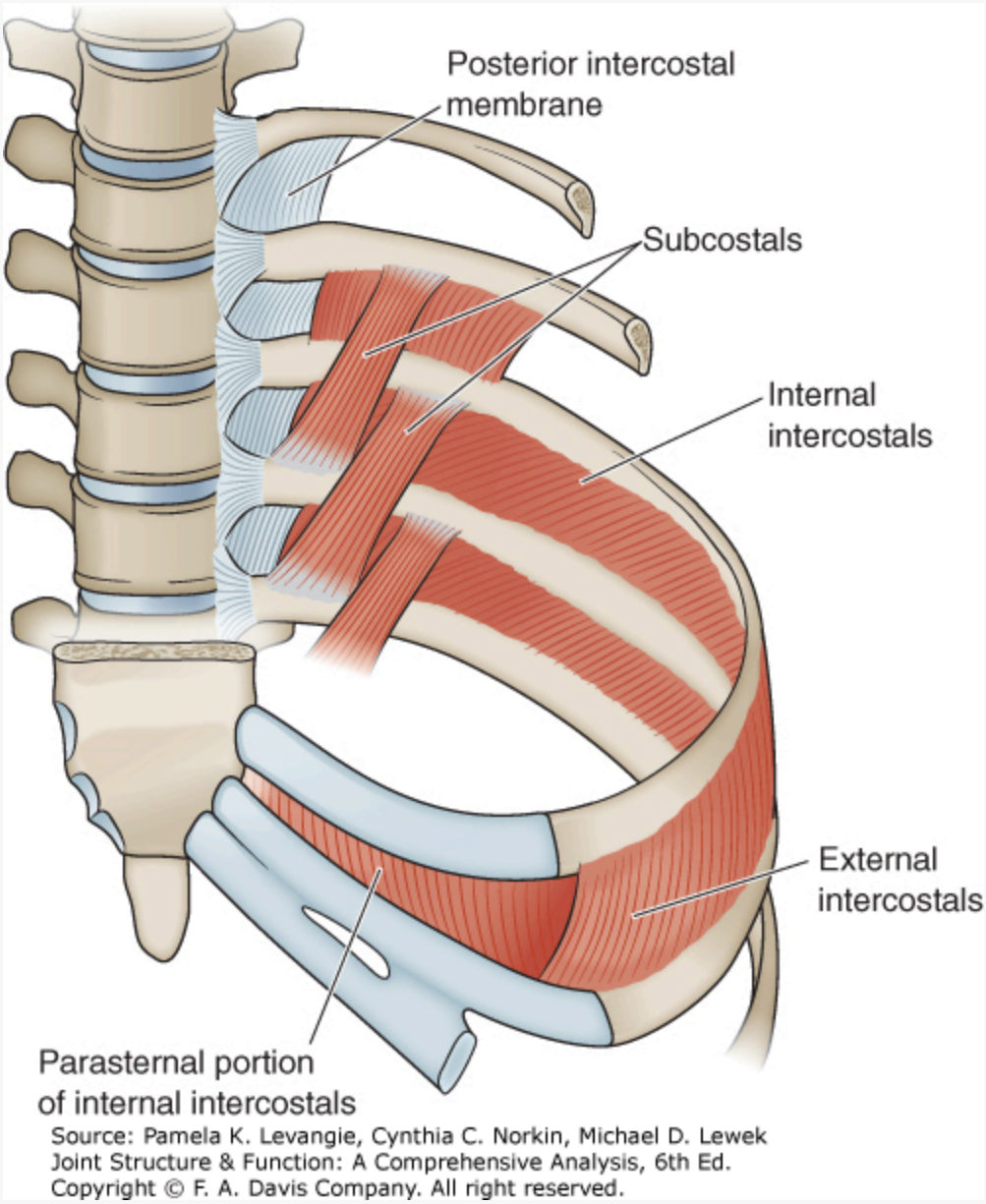
INTERCOSTAL MUSCLES

The **internal intercostals,** the **external intercostals,** and the **subcostals** muscles (Fig.

5–17) connect adjacent ribs to each other and are named according to their anatomic orientation and location. The internal intercostal muscles arise from the inner surfaces of ribs 1 through 11 and insert into the rib below. The fibers of the internal intercostal muscles lie deep to the external intercostal muscles and run caudally and posteriorly. The internal intercostals begin anteriorly at the chondrosternal junctions and continue posteriorly to the angles of the ribs, where they become an aponeurotic layer called the **posterior intercostal membrane.**

FIGURE 5-17

Intercostal muscles.



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The external intercostal muscles originate on ribs 1 through 11 and insert into the rib below. The fibers are more superficial to the rib cage and run caudally and anteriorly at an oblique angle to the internal intercostal muscles (see Fig. 5–17).[15] The external intercostal muscles begin posteriorly at the tubercles of the ribs and extend anteriorly to the costochondral junctions, where they form the **anterior intercostal membrane.**[15]

Given the described attachments, only the internal intercostal muscles are present anteriorly from the chondrosternal junctions to the costochondral joints. These are the segments of the internal intercostal muscles that are referred to as the **parasternal muscles** (see Fig. 5–17) and are found mainly in the first through fifth intercostal spaces.[15] There are only external intercostal muscles present posteriorly from the tubercle to the angle of the ribs. Laterally, both internal and external intercostal muscle layers are present and may be referred to in this location as the **interosseous** or **lateral intercostal muscles.**[15]

The subcostal muscles (see Fig. 5–17) are also intercostal muscles, but are generally found only in the posterior lower rib cage. The subcostal muscles originate at the rib angles and may span more than one intercostal space before inserting into the inner surface of a caudal rib. Their fiber directions are similar to those of the internal intercostal muscles.

The function of the intercostal muscles during ventilation is not as simple as once thought. The accepted idea that the external intercostal muscles are active during inhalation and the internal intercostal muscles are active during exhalation is not completely accurate. Both sets of intercostal muscles have been shown to be active during both phases of ventilation depending on type of breathing and neural recruitment of muscles.[15] Activation of the external intercostals and the parasternals (the anterior portion of the internal intercostals) has an inspiratory effect on the rib cage. The action of the parasternal muscles appears at every interspace to be a rotation of the costosternal junctions, resulting in elevation of the ribs and sternum as well as anterior movement of the sternum.[16] Another function of the parasternal muscles

seems to be the stabilization of the rib cage.[17,18] This stabilizing action of the parasternal muscles opposes the decreased intrapulmonary pressure generated during diaphragmatic contraction (inhalation), preventing a paradoxical, or inward, movement of the upper chest wall during inhalation.[19]

Activity of the external intercostals, although minimal during breathing at rest, increases with increasing ventilation.[20] The external intercostals in the posterior and superior thorax have an inspiratory effect, although this inspiratory effort diminishes gradually as you move lower and anterior toward the middle of the rib cage. By the anterior lowest intercostal interspaces, the respiratory effect of the external intercostals has changed to an expiratory one.[16]

The internal intercostals, throughout all interspaces but especially the more caudal portions, have an expiratory effect.[16]

The function of the lateral portion of the internal and external intercostal muscles (the so-called interosseous intercostals) involves mainly trunk rotation.[16] The lateral intercostal muscles have a relatively small amount of activity in the ventilator cycle in comparison with the parasternal muscles and the diaphragm.[21] The major role of the lateral intercostal muscles is in axial rotation of the thorax, with the contralateral internal and external intercostal muscles working synergistically to produce trunk rotation (e.g., right external and left internal intercostal muscles are active during trunk rotation to the left).[21]

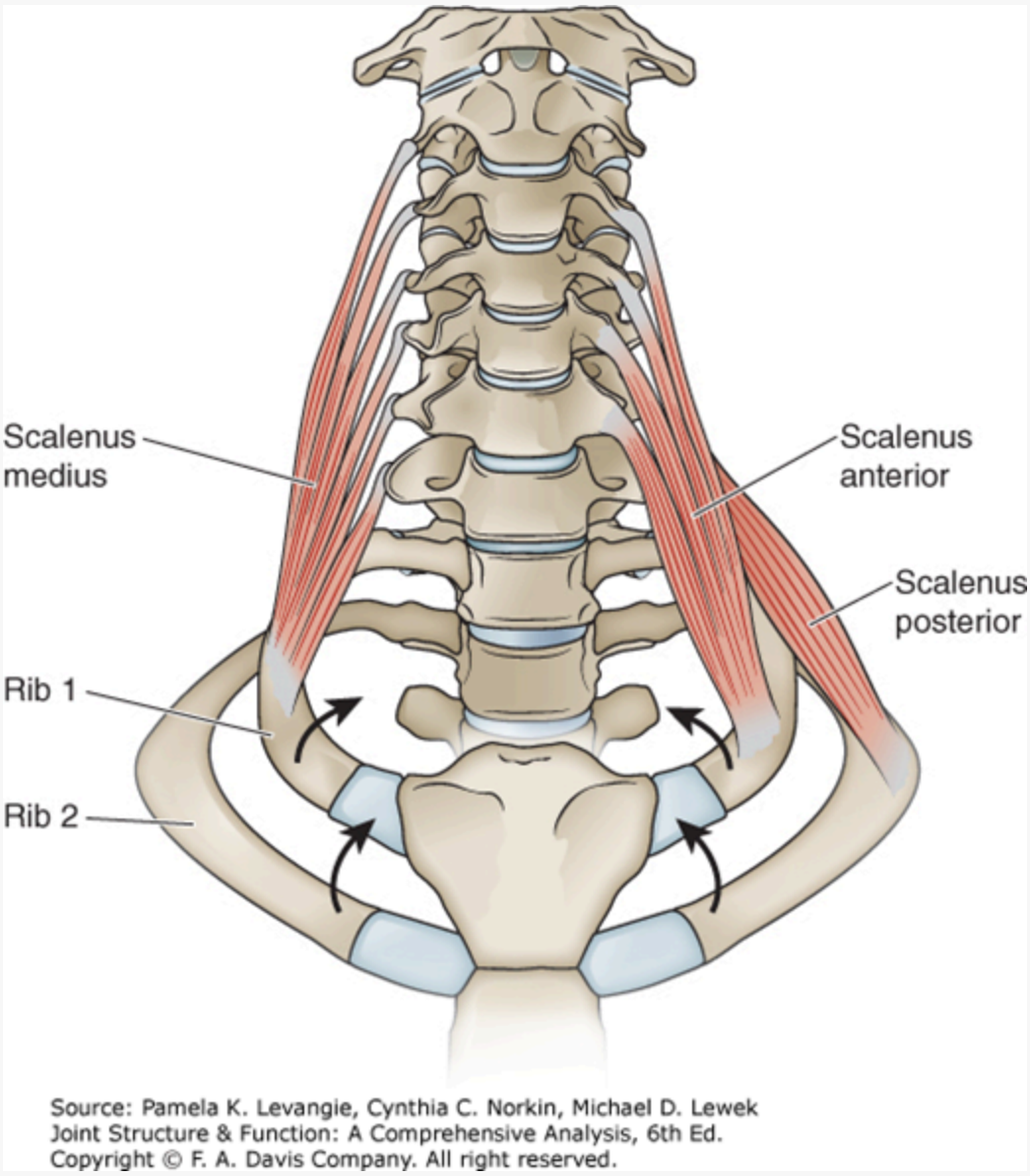
SCALENE MUSCLES

The scalene muscles are also primary muscles of ventilation at rest.[10] The scalene

muscles attach to the transverse processes of C3 to C7 and descend to the upper borders of the first rib (anterior scalene and medial scalene) and second rib (posterior scalene) (Fig. 5–18).

FIGURE 5-18

The scalenus anterior, scalenus medius, and scalenus posterior. Their action lifts the sternum and the first two ribs in the pump-handle motion.



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Their action lifts the first two ribs, and therefore the sternum, in the pump-handle motion of the upper rib cage (see Fig. 5–11).[22] Activity of the scalene muscles begins at the onset of inhalation and increases as inhalation gets closer to total lung capacity. The length-tension relationship of the scalene muscles allows them to generate *a* greater force late into the respiratory cycle, when the force from the diaphragm is decreasing. The scalene muscles also function as stabilizers of the rib cage. The scalene

muscles, along with the parasternal muscles, counteract the potential paradoxical movement of the upper chest caused by a decrease in intrapulmonary pressure created by the diaphragm's contraction.

Posteriorly, the fibers of the **levatores costae** run from the transverse processes of vertebrae C7 through T11 to the posterior external surface of the next lower rib between its tubercle and the angle and can assist with elevation of the upper ribs during inhalation.[23]

ACCESSORY MUSCLES OF VENTILATION

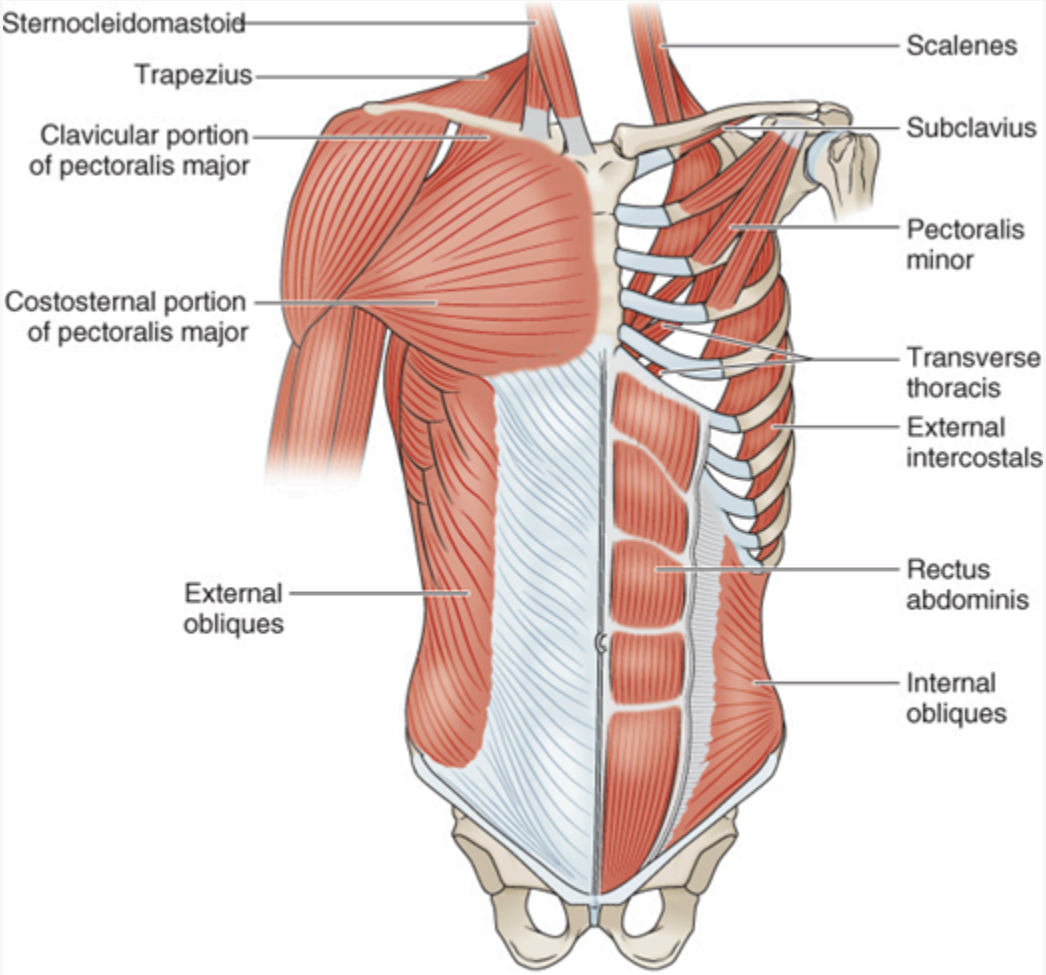
The muscles that attach the rib cage to the shoulder girdle, head, vertebral column, or pelvis may be classified as accessory muscles of ventilation. These muscles assist with inhalation or exhalation in situations of stress, such as increased activity or disease.

When the thorax is stabilized, the accessory muscles of ventilation move the vertebral column, arm, head, or pelvis on the trunk. During times of increased ventilatory demand, the rib cage can become the mobile segment. The accessory muscles of inhalation, therefore, increase the thoracic diameter by moving the rib cage upward and outward. The accessory muscles of exhalation move the diaphragm upward and the thorax downward and inward. The most commonly described accessory muscles are shown in Figure 5–19 and are discussed in the following paragraphs.

FIGURE 5-19

Accessory muscles of ventilation are those used during times of increased ventilatory demand. The right side of the figure shows some

of the anterior superficial muscles of the thorax that can be accessory muscles of ventilation, and the left side of the thorax shows the deeper accessory muscles of ventilation.



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The **sternocleidomastoid** muscle runs from the manubrium and superior medial aspect of the clavicle to the mastoid process of the temporal bone. The usual bilateral action of the sternocleidomastoid is flexion of the cervical spine. With the help of the **trapezius** muscle to stabilize the head, the bilateral action of the sternocleidomastoid muscles can pull the rib cage superiorly, expanding the upper rib cage in the pump-handle

motion. The recruitment of this muscle seems to occur toward the end of maximal inhalation.[24]

The sternocostal (sternal) portion of the **pectoralis major** muscle can elevate the upper rib cage rather than act on the humerus when the humerus is stabilized. The clavicular head of the pectoralis major can be either inspiratory or expiratory in action, depending on the position of the humerus. When the arms are positioned so that the humeral attachment of the pectoralis major is below the level of the clavicle (arms below horizontal), the clavicular portion acts as an expiratory muscle by pulling the manubrium and upper ribs downward. When the humeral attachment of the pectoralis major is above the level of the clavicle (arms above horizontal), the muscle becomes an inspiratory muscle by pulling the manubrium and upper ribs up and out. The **pectoralis minor** can help elevate the third, fourth, and fifth ribs during active inhalation. The **subclavius,** a small muscle between the clavicle and the first rib, can also assist in raising the upper chest for inhalation.

The **serratus posterior superior** and **serratus posterior inferior** are located on the posterior thorax with attachments from cervical and thoracic spinous processes to ribs (therefore are not seen in Fig. 5–19). These muscles have been assumed to be accessory muscles of respiration based largely on their attachments to the ribs. There is no evidence to support a ventilatory role for these muscles and should not, at present, be considered to have a respiratory function.[25,26]

The abdominal muscles include the **rectus abdominis, external oblique abdominis, internal oblique abdominis,** and **transversus abdominis** (deep to the other abdominals so not shown in Fig. 5–19). The abdominals are also accessory muscles of ventilation as

well as trunk flexors and rotators. The major function of the abdominal muscles in ventilation is to assist with forced exhalation. The muscle fibers pull the ribs and costocartilages caudally, into a motion of exhalation. By increasing intra-abdominal pressure, the abdominal muscles can force the diaphragm upward into the thoracic cage, increasing both the volume and speed of exhalation.

Although usually considered accessory muscles of exhalation, the abdominal muscles play two significant roles during inhalation. First, the increased abdominal pressure created by lowering of the diaphragm in inhalation must be countered by tension in the abdominal musculature. Abdominal tension reduces abdominal compliance, allowing the central tendon of the diaphragm to stabilize so that the stabilized diaphragm can produce lateral chest wall expansion. Secondly, the increased intra-abdominal pressure created by active abdominal muscles during forced exhalation pushes the diaphragm cranially and exerts a passive stretch on the costal fibers of the diaphragm.[3] These changes prepare the respiratory system for the next inhalation by optimizing the length-tension relationship of the muscle fibers of the diaphragm. During periods of increased ventilatory needs, the increased muscular activity of the abdominal muscles assists in both exhalation and inhalation.[3,11]

The **transversus thoracis (triangularis sterni)** muscles are a flat layer of muscle that runs deep to the parasternal muscles. The transversus thoracis muscles originate from the posterior surface of the lower sternum and run cranially and laterally, inserting into the costal cartilages of ribs 3 through 7.[3] These muscles are recruited, along with the abdominal muscles, to pull the rib cage caudally. The mechanical advantage of the transverses thoracis is greater farther down the ribcage.[15] Studies have shown that

these muscles are primarily expiratory muscles, especially when exhalation is active, as in talking, coughing, or laughing, or in forced exhalation into functional residual capacity.[27,28]

Gravity acts as an accessory force for ventilation in the supine position. Gravity, acting on the abdominal viscera, performs the same function as the abdominal musculature in stabilizing the central tendon of the diaphragm. In fact, in the supine position, the abdominal muscles and the trangularis sterni are silent on the electromyographic monitoring during breathing at rest.[27,28]

Coordination and Integration of Ventilatory Motions

The coordination and integration of the skeletal and muscular chest wall components during breathing are complex, as can be seen, minimally, by the changes in how muscles may contribute under different conditions. The recruitment of ventilatory muscles is also dependent on the activities in which a person is participating (there is a different recruitment pattern when a person is at rest than during activity), whether the body is upright against gravity or lying supine, whether the person is stationary or locomoting, and whether the person is phonating or not. Additionally, recruitment can change with disorders of the chest wall, the lung parenchyma, or the neuromuscular system.[22,23, 29-31] A high and complex level of coordination is necessary for the primary and accessory muscles of ventilation to perform the necessary functions of ventilation.



Foundational Concepts 5–3

Summary of the Ventilatory Sequence During Breathing

Although the coordinated function and sequence of breathing are complex when activities are combined, the following sequence of motions and muscle actions is

typical of a healthy person at rest.

- The diaphragm contracts and the central tendon moves caudally.
- The parasternal, external intercostals, and scalene muscles elevate the ribs and move the sternum anteriorly and superiorly. The muscles also stabilize the anterior upper chest wall to prevent a paradoxical inward movement caused by the decreasing intrapulmonary pressure.
- As intra-abdominal pressure increases, the abdominal contents are displaced so that the anterior epigastric abdominal wall is pushed anteriorly. Further outward motion of the abdominal wall is countered by the abdominal musculature, with that resistance allowing the central tendon to stabilize on the abdominal viscera.
- With continued shortening of the appositional (costal) fibers of the diaphragm, the lower ribs are pulled cephalad and laterally, which results in the bucket-handle movement of the lower ribs.
- With continued inhalation, the parasternal, scalene, and levatores costarum muscles actively rotate the upper ribs and elevate the manubriosternum, which results in an anterior (pump-handle) motion of the upper ribs and sternum.
- The lateral motion of the lower ribs and anterior motion of the upper ribs and sternum can occur simultaneously.
- Exhalation during breathing at rest is mostly passive, using the elastic recoil properties of the lungs and the cessation of inspiratory muscle contraction.

Lifespan and Clinical Considerations

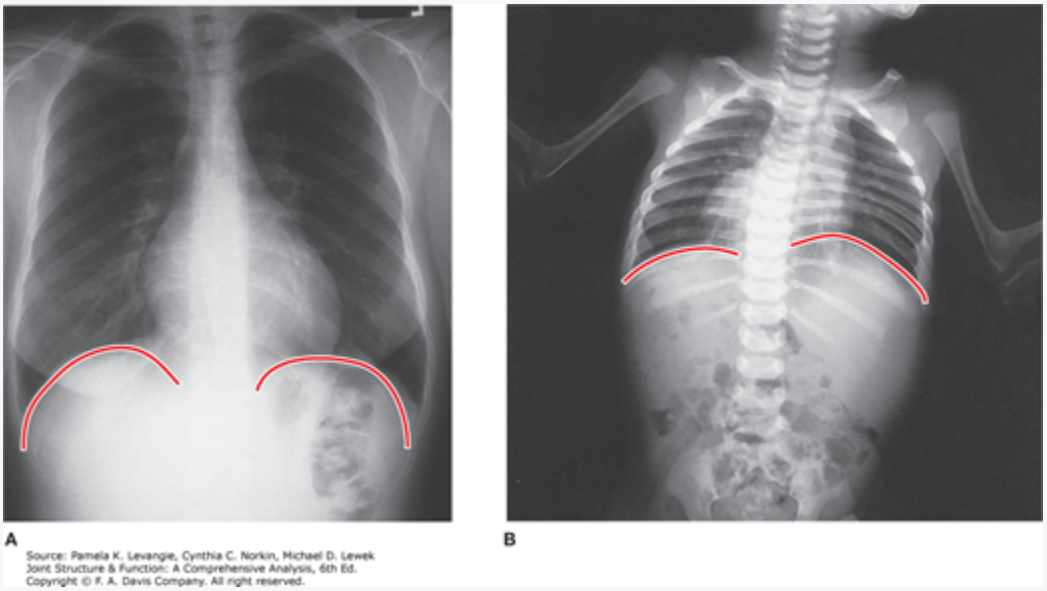
Differences Associated With the Neonate

The compliance, configuration, and muscle action of the chest wall changes significantly from infants to the elderly (Fig. 5–20). A newborn has a cartilaginous and, therefore, extremely compliant chest wall that allows the distortion necessary for the infant's thorax to travel through the birth canal. The increased compliance of the rib cage is at the expense of thoracic stability. An infant's chest wall muscles must act as stabilizers, rather than mobilizers, of the thorax to counteract the reduced intrapulmonary pressure created by the lowered diaphragm during inhalation that

would otherwise pull the ribs in. Complete ossification of the ribs does not occur for several months after birth.

FIGURE 5-20

A. In an adult, the ribs slope downward, and the diaphragm has an elliptical shape. **B.** The rib cage of an infant shows a nearly horizontal alignment of the ribs, resulting in the angle of insertion of the costal fibers of the diaphragm also more horizontal.



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The ribs in the adult thorax slope downward and the diaphragm is elliptically shaped (Fig. 5–20A). The rib cage of an infant shows a more horizontal and circular alignment of the ribs (Fig. 5–20B), with the angle of insertion of the costal fibers of the diaphragm also more horizontal than that of an adult. There is an increased tendency for the diaphragmatic fibers to pull the lower ribs inward, thereby decreasing ventilation efficiency and increasing distortion of the chest wall.[32] There is very little motion of the rib cage during an infant's tidal breathing.

Only 20% of the muscle fibers of the diaphragm are fatigue-resistant fibers in a healthy newborn, compared with 50% in an adult. This discrepancy predisposes infants to earlier diaphragmatic fatigue.[32] Accessory muscles of ventilation are also at a disadvantage in the infant because the inability of the infant to stabilize their upper extremities, head, and spine makes it difficult for the accessory muscles of ventilation to produce the action needed to be helpful during increased ventilatory demands.

With increasing age, the infant's rib cage ossifies and ventilatory muscles can begin to mobilize rather than stabilize the thorax. Improving head control also allows the infant to begin to use accessory muscles for increased ventilation. As a toddler assumes independent sitting and standing, the force of gravity and postural changes allow the anterior rib cage to angle obliquely downward over time. This elongated thorax allows for a greater bucket-handle motion of the rib cage. The attachments for the muscles of ventilation move with the increasingly angled ribs, improving their action on the thorax.

Throughout childhood, the numbers of alveoli and airways continue to increase, and in early adolescence they continue to expand, as demonstrated by increases in pulmonary function test results.[33]

Differences Associated With the Elderly

Skeletal changes that occur with aging affect pulmonary function. Many of the articulations of the chest wall undergo fibrosis with advancing age.[34,35] The interchondral and costochondral joints can fibrose, and the chondrosternal joints may be obliterated. The xiphisternal and the manubriosternal junctions ossify with age. The chest wall articulations that are true synovial joints may undergo morphological changes associated with aging, including reduced mobility. The costal cartilages ossify,

which interferes with their axial rotation.[14] Overall, chest wall compliance is significantly reduced (stiffness increases) with age. Reduction in diaphragm-abdomen compliance has also been reported and is at least partially related to the decreased rib cage compliance, especially in the lower ribs that are part of the zone of apposition.[36]

Aging also brings anatomical changes to the lung tissues that affect ventilation. The airways narrow, the alveolar duct diameters increase, and the alveolar sacs lose surface area for gas exchange. There is a reorientation and a decrease of the elastic fibers in the lung parenchyma. Overall, there is a decrease in elastic recoil and an increase in pulmonary compliance.[35] Because the resting position of the thorax depends on the balance between the elastic recoil properties of the lungs pulling the ribs inwardly and the outward pull of the ribs and costal cartilages, the reduced recoil property of the lung tissue allows the thorax to rest with an increased AP diameter (a relatively increased inspiratory position). An increased kyphosis often observed in older individuals will further decrease the mobility not only of the thoracic spine but also of the rib cage.

Skeletal muscles of ventilation in the elderly have a documented loss of strength, fewer muscle fibers, a lower oxidative capacity, a decrease in the number or the size of type IIX (fast-twitch) fibers, and an increase in the time to peak tension.[35,36] The decrease in abdominal tension that occurs with aging allows the resting position of the diaphragm to be lower in the thorax, that is, less domed. There is an earlier recruitment pattern for accessory muscles of ventilation. For example, the transverse thoracic muscles are active during exhalation at rest in older subjects in the standing position.[26]

Overall in the elderly, there is likely to be a decreased compliance of the bony rib cage,

an increased compliance of the lung tissue, and an overall decreased compliance of the respiratory system. The result of these age-related skeletal and tissue changes is an increase in the amount of air remaining in the lungs after a normal exhalation. If the lungs retain more air at the end of exhalation (an increase in the functional residual capacity), there will be a decrease in inspiratory capacity (tidal volume plus inspiratory reserve volume) of the thorax. The decrease in the effectiveness of the ventilatory muscles means that ventilation becomes more energy-expensive with age. Functionally, the result of these multiple changes with increasing age is a decrease in the ventilatory reserve available during times of need, such as during an illness or increased activity.

Pathological Changes in Structure and Function

In this chapter, the effects of the musculoskeletal system on ventilation have been discussed. It is interesting to note the opposite can also be true; changes in the pulmonary system can affect the bony thorax. A brief discussion of this relationship is presented using **chronic obstructive pulmonary disease (COPD)** as the framework.



Patient Application 5–4

Jon Nguyen is a 58-year-old former Fed Ex driver who smoked two to three packs of cigarettes per day for 40 years. Several years ago, Jon was diagnosed with COPD. Jon cut his smoking back to one pack per day, and is still trying to quit smoking completely. He was forced to retire last year because his shortness of breath was limiting his ability to get in and out of his truck repeatedly throughout the day. In the past year, he has had repeated bouts of bronchitis and was admitted to the hospital a couple of times for pneumonia. Jon reports that he is now finding it more difficult to breathe even at rest and is continually fatigued. A good friend now does his food shopping for him. Pulmonary function studies show a seriously compromised respiratory system, with impairments in ventilatory capacity and lung volume

common to COPD (Fig. 5–21). Jon's appearance is typical of an individual with COPD, with a barrel-shaped chest in spite of his slight build (Fig. 5–22).

FIGURE 5-21

Lung volumes and capacities of a healthy person and of a patient with COPD. COPD, chronic obstructive pulmonary disease; ERV, expiratory reserve volume; IRV, inspiratory reserve volume; RV, residual volume; TV, tidal volume.

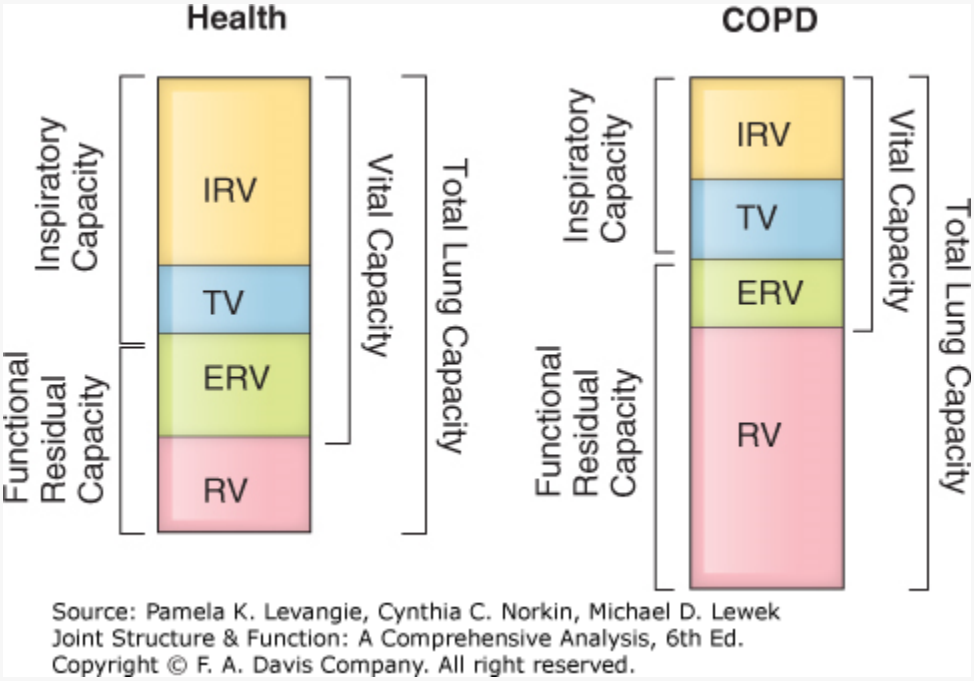
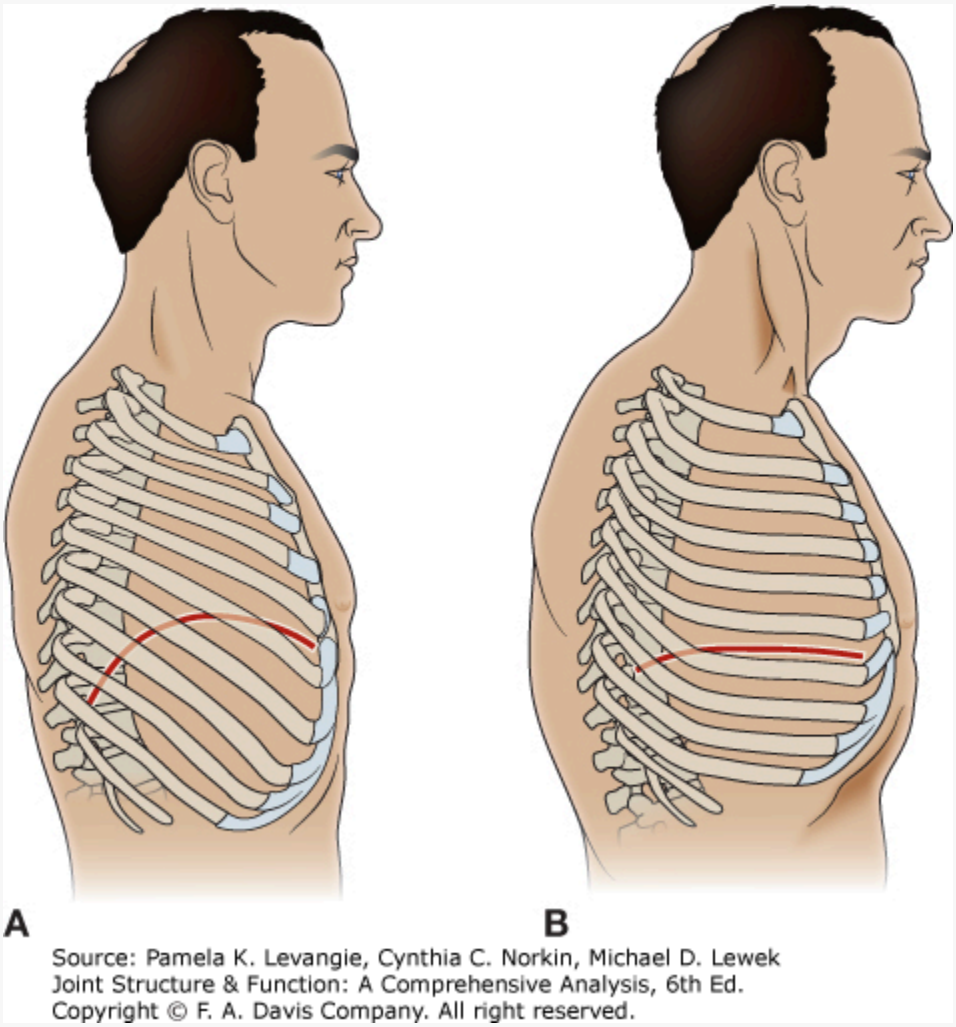


FIGURE 5-22

Chest changes at rest with chronic obstructive pulmonary disease (COPD). **A.** The alignment of clavicle, ribs, and diaphragm in a typical adult at rest. **B.** In individuals with COPD, the resting AP diameter of the chest wall increases and the accompanying hyperinflation of the

lungs is associated with elevation of the clavicle and sternum, more horizontally-oriented ribs, a flattened diaphragm, and tension in the accessory muscles of ventilation (e.g., the sternocleidomastoid muscles).



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The major manifestation of COPD is damage to the airways and destruction of the alveolar walls. As tissue destruction occurs with disease, the elastic recoil property of the lung tissue diminishes. Exhalation, either passive or active, is dependent upon this elastic recoil property. In patients with COPD, exhalation becomes ineffective in

removing adequate amounts of air from the thorax, leading to air trapping and hyperinflation.

The static or resting position of the thorax is a function of the balance between the elastic recoil properties of the lungs pulling inward and the normal outward spring of the rib cage. In patients with COPD, there is an imbalance in these two opposing forces that alters both lung volumes and ventilatory capacities (see Fig. 5–21). As elasticity decreases and more air remains within the lungs, an increase in the AP diameter of the hyperinflated thorax is apparent, showing a classic barrel-shaped appearance and accompanying changes to the thorax (see Fig. 5–22).

The range of motion, or excursion, of the thorax is limited in obstructive pulmonary disease. Although the basic characteristic of COPD is an inability to exhale sufficiently, it is clear that inspiratory capacity is compromised as a result.

Hyperinflation affects not only the bony components of the chest wall but also the muscles of the thorax. Within the lungs, the increased air does not allow the diaphragm to return to its usual high domed shape and location so that there is a flattening of the diaphragm at rest. The fibers of the diaphragm remain shortened even after exhalation, decreasing the available range of contraction. The angle of pull of the flattened diaphragm fibers becomes more horizontal with a decreased zone of apposition. In severe cases of hyperinflation, the fibers of the diaphragm are aligned more horizontally than vertically. Contraction of this very flattened diaphragm will pull the lower rib cage inward, actually working against lung inflation.[37,38]

With the compromise of the diaphragm in COPD, the majority of inhalation is performed

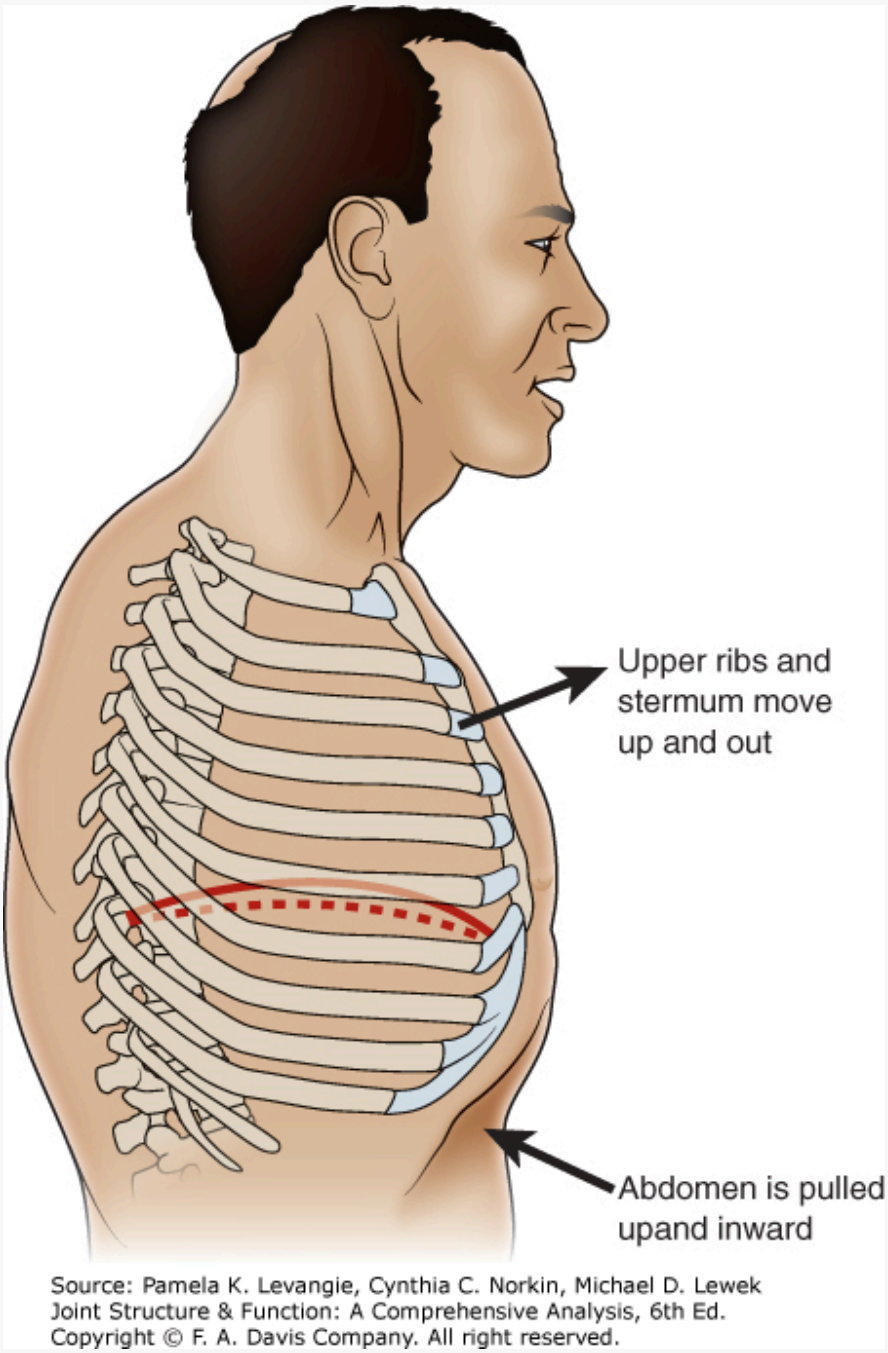
by other inspiratory muscles, which are not as efficient as the diaphragm. The barrel-shaped and elevated thorax puts the sternocleidomastoid muscles in a shortened position, making them much less efficient. The parasternal and scalene muscles are able to generate a greater force as the lungs approach total lung capacity; consequently, hyperinflation has a less dramatic effect on them.

The altered muscle power during inhalation with COPD now favors movement of the upper, rather than the lower, rib cage. With a forceful contraction of the functioning inspiratory muscles of the upper rib cage in a patient with an ineffective diaphragm, the diaphragm and abdominal contents may actually be pulled upward, into the thorax (Fig. 5–23).[39,40] During exhalation, the abdomen is pushed back down and out. This paradoxical thoracoabdominal breathing pattern can be seen in patients with obstructive disease who are in respiratory distress. The disadvantages of these biomechanical alterations with hyperinflation are compounded by the increased demand for ventilation in COPD. More work is required of a less effective system. The energy cost of ventilation, or the work of breathing, is markedly increased in patients with COPD.

FIGURE 5-23

Paradoxical thoracoabdominal movement in chronic obstructive pulmonary disease (COPD). During inspiration, the strong pull of the accessory muscles of inspiration, there is an increase in the motion of the upper chest. Rather than flattening of the diaphragm and protrusion of the abdomen usually seen with normal inspiration (see

Fig. 5–16A), the expansion of the rib cage pulls the abdominal viscera and the typical flattened diaphragm of the person with COPD (dotted line) slightly upward into the thorax (solid line). With exhalation, the thorax decreases in size, and the abdominal viscera return to their resting position.



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Summary

In this chapter, comprehensive coverage of the structure and function of the bony thorax and the ventilatory muscles has been provided. Additional information on the

structure and function of accessory muscles of ventilation as these muscles may affect the shoulder complex will be presented in Chapter 7.

Study Questions

1. Describe the articulations of the chest wall and thorax, including the costovertebral, costotransverse, costochondral, chondrosternal, and manubriosternal joints.

2. What are the different motions of the chest wall motions during breathing? Explain where these motions occur.

3. What are the roles of the diaphragm, the intercostal muscles, and the abdominal muscles during breathing at rest?

4. Describe the accessory muscles of ventilation and explain their functions.

5. Describe the inspiratory and expiratory functions of the abdominal muscles.

6. What effect does COPD have on the biomechanics of the thorax and the inspiratory muscles?

7. How does the aging process affect the structure and function of the thorax?

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Chapter 6: The Temporomandibular Joint

Pamela D. Ritzline

Anatomy Overview

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MUSCLE ACTIONS OF THE TEMPOROMANDIBULAR JOINT		
TABLE KEY: Primary movers Secondary movers		
Sagittal Plane (rotation)	Elevation (closing mouth)	Depression (opening mouth)
	Temporalis Masseter Medial pterygoid	Lateral Pterygoid Digastric Stylohyoid Mylohyoid Geniohyoid Omohyoid Sternohyoid Sternothyroid Thyrohyoid Platysma
Sagittal Plane (translation)	Protrusion	Retraction
	Lateral Pterygoid	Temporalis

	Masseter medial pterygoid	Masseter

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TEMPOROMANDIBULAR JOINT MUSCLE ATTACHMENTS		
Muscles	**Proximal Attachment**	**Distal Attachment**
Temporalis	Broad attachment to floor of temporal fossa	Narrow attachment to coronoid process and anterior border of ramus of mandible
Masseter	Quadrate muscle attaching to inferior border and medial surface of the zygomatic arch	Angle and lateral surface of ramus of mandible
Lateral Pterygoid	Triangular two-headed muscle from infratemporal surface and crest of greater wing and lateral pterygoid plate of sphenoid	Superior head attaches primarily to joint capsule and articular disc of temporomandibular joint; inferior head attaches primarily to condyle of mandible
Medial Pterygoid	Quadrangular two-headed muscle from medial surface of lateral pterygoid plate, palatine bone, and maxilla	Medial surface of ramus of mandible
Digastric (suprahyoid)	Inner surface of anterior body of mandible (anterior belly); mastoid process of temporal bone (posterior	Hyoid bone

	belly)	
Stylohyoid (suprahyoid)	Styloid process of temporal bone	Hyoid bone
Mylohyoid (suprahyoid)	Medial surface of mandible	Hyoid bone
Geniohyoid (suprahyoid)	Inner surface of anterior body of mandible	Hyoid bone
Omohyoid (infrahyoid)	Superior surface of scapula	Hyoid bone
Sternohyoid (infrahyoid)	Manubrium of sternum	Hyoid bone
Sternothyroid (infrahyoid)	Posterior surface of manubrium of sternum	Thyroid cartilage
Thyrohyoid (infrahyoid)	Thyroid cartilage	Hyoid bone

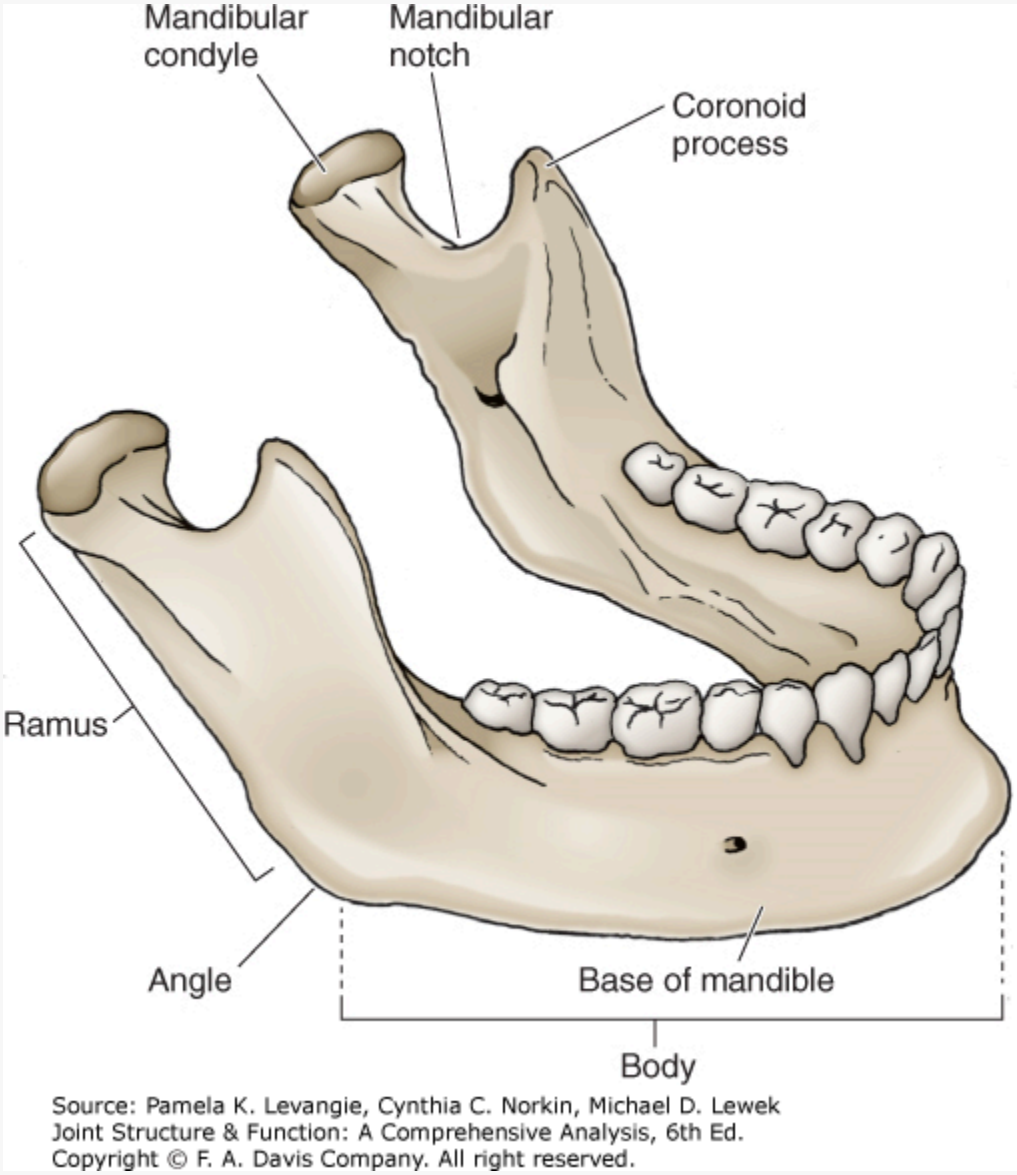
Introduction

The **temporomandibular (TM) joint** is unique in both structure and function. Structurally, the **mandible** is a horseshoe-shaped bone (Fig. 6–1) that articulates with the **temporal bone** at each of its posterosuperiorly located condyles and produces two distinct but highly interdependent articulations. Each TM joint contains a disc that separates the joint into an upper and a lower articulation. Functionally, mandibular movement involves concurrent movement in the four distinct joints (two per TM joint), resulting in a complex structure that moves in all planes of motion to achieve normal

function.

FIGURE 6-1

The mandible.



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A discussion of the structure and function of the TM joint will allow the reader to appreciate and understand its unique features, its relationship with the cervical spine, and the impact of impairments and pathologies of the TM joint. Although the purpose

of this chapter is to discuss the normal function and structure of the TM joint, TM disorders are a common subgroup of orofacial pain disorders.[1] This chapter will introduce some of the common conditions that involve deviations of normal structure.

Structure and Function of the TM Joint

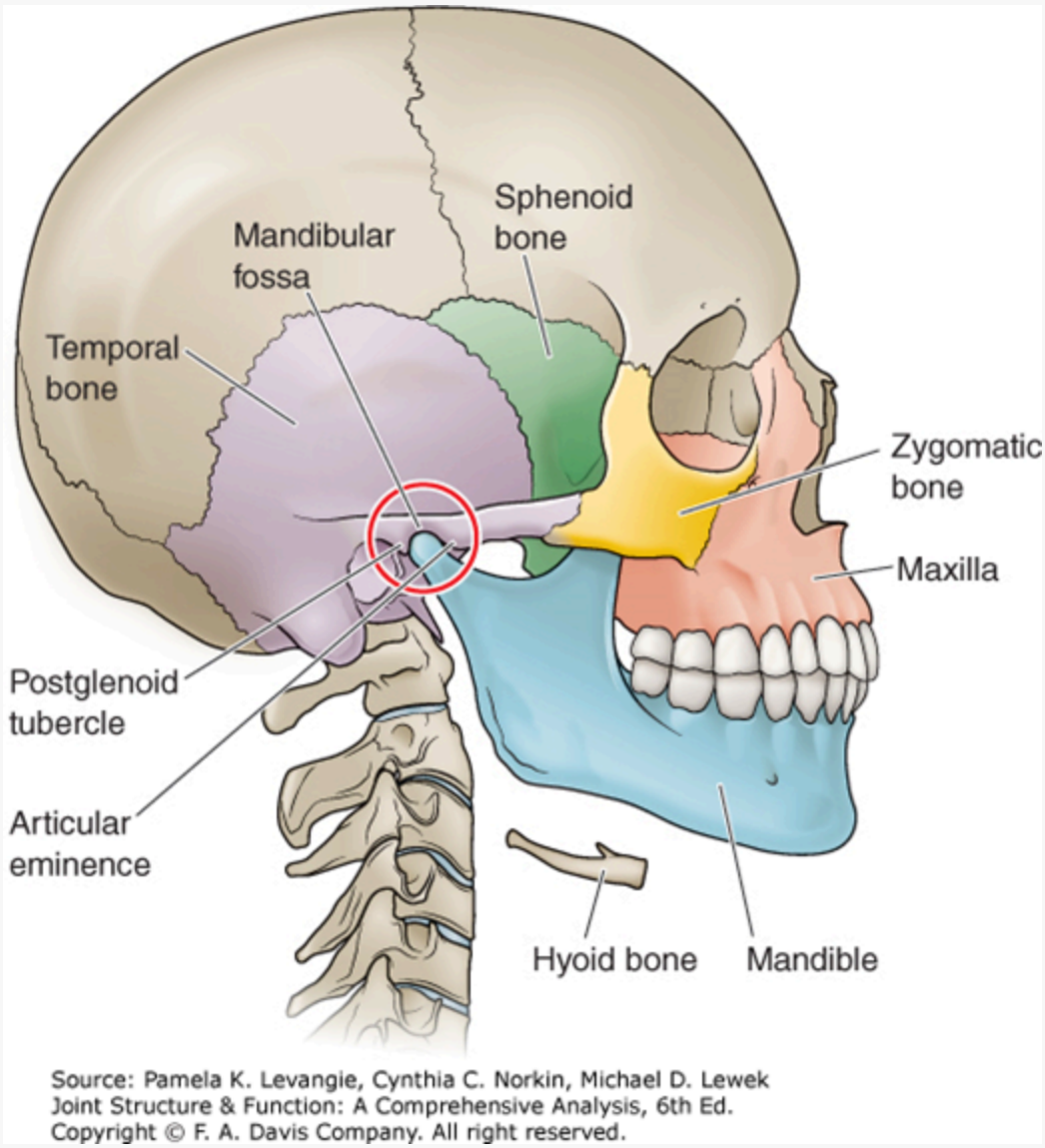
Structure

Articular Structures

Multiple bones merge to form the structure and contribute to the function of the TM joints. These bones include the mandible, **maxilla,** temporal, **zygomatic, sphenoid,** and **hyoid** bones (Fig. 6–2).[2] The proximal or stationary segment of the TM joint is the temporal bone. Although we typically think of the mandible as moving on the stationary maxilla, the maxilla is not part of the TM joint. The condyles of the mandible sit in the **mandibular fossa** of the temporal bone. The mandibular fossa is located between the **postglenoid tubercle** and the **articular eminence** of the temporal bone (Fig. 6–2).[2–4]

FIGURE 6-2

Lateral view of the bones and bony landmarks that make-up the TM joint (circled) and that serve as attachments for TM joint musculature.



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The temporal bones and the mandible form two TM joints that are part of a closed chain and must function together; however, each is an anatomically separate joint, and can vary from each other structurally as well as functionally. The TM joints are considered synovial joints formed by the **condyle of the mandible** inferiorly and the articular eminence of the temporal bone superiorly.[2,4,5] Although the condyle of the mandible sits in the mandibular fossa, the bone in the fossa is thin and translucent, therefore not at all appropriate as an articular surface. In contrast, the articular

eminence contains a major area of trabecular bone and serves as the primary articular surface for the mandibular condyle. Thus, functionally, the mandibular condyles articulate with the articular eminence of the temporal bone on each side. The articular eminence and the condyle are both convex structures (see Fig. 6–2), resulting in an incongruent joint.[2–4] Incongruent joints require rolling and sliding for normal range of motion (ROM) to occur.

The TM joint is an atypical synovial joint because no hyaline cartilage covers the articular surfaces.[5,6] The articular surfaces of the articular eminence and the mandibular condyle are covered with dense, avascular, collagenous tissue that contains some cartilaginous cells. Because some of the cells are cartilaginous, the covering is often referred to as fibrocartilage. The articular collagen fibers are aligned perpendicular to the bony surface in the deeper layers to withstand stresses. The fibers near the surface of the articular covering are aligned in a parallel arrangement to facilitate sliding of the joint surfaces.[2,6,7] The presence of fibrocartilage rather than hyaline cartilage is significant because fibrocartilage can repair and remodel itself.[5–7] Typically fibrocartilage is present in areas that have to withstand repeated and high-level stress. The TM joints are subject to the repetitive stress of jaw motions as well as to tremendous bite forces that have been measured between 600 N and 1200 N.[8] Mueller and Maluf described the physical stress theory along with the predictable adaptive changes of tissues in response to the various levels of stress.[7] The TM joint surfaces are amenable to some degree of adaptation, but no clear-cut point exists between adaptive and maladaptive changes.[9] Tanaka and Koolstra[5] acknowledged that loading is necessary to stimulate the remodeling that occurs with normal functional demands to ensure homeostasis within a joint. Although no evidence exists to determine the

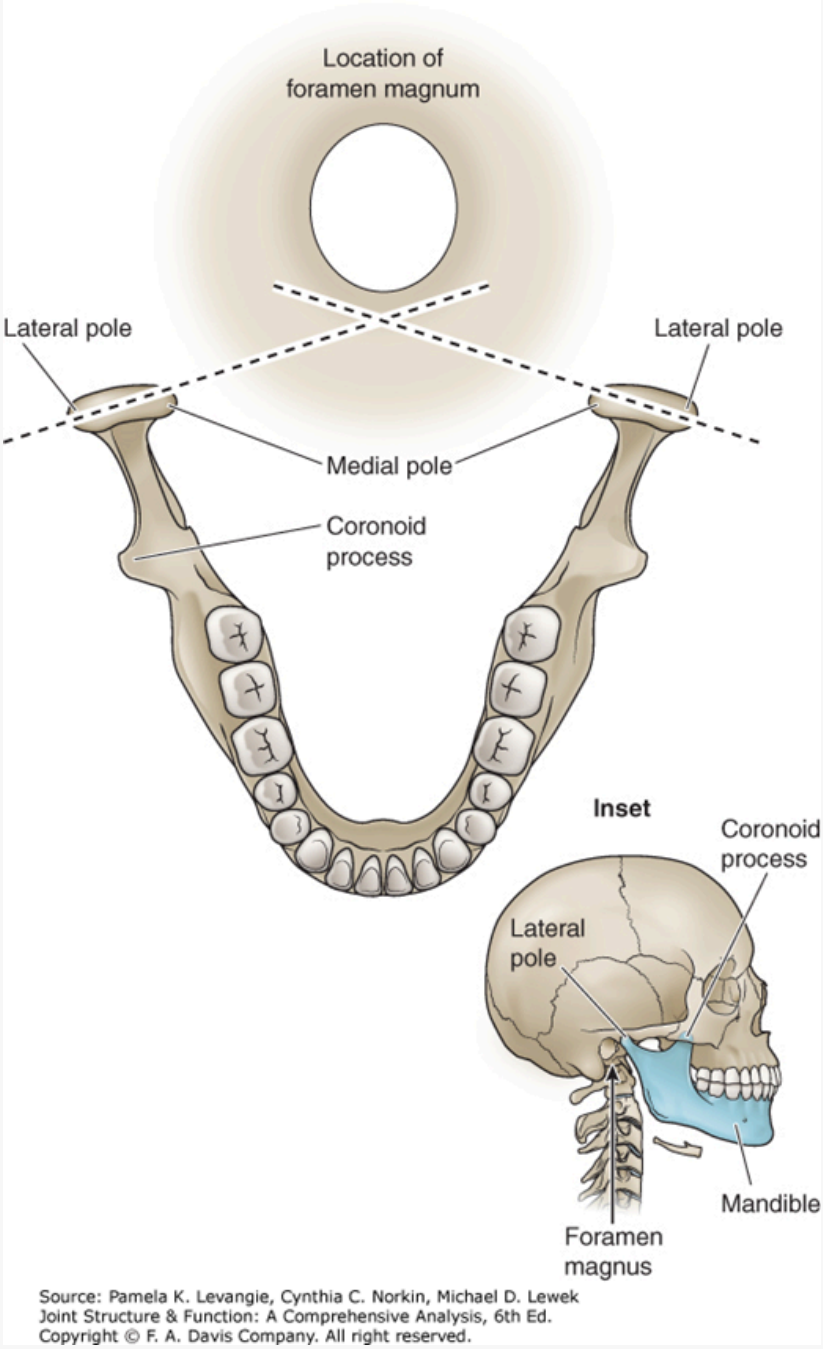
appropriate load for the TM joint, it has been proposed that aging and some physical and mental conditions may disrupt normal load.[5]

The mandible is the largest of the facial bones and is highly mobile, arch-shaped, and consists of a condyle at each posterior superior portion.[2] Each mandibular condyle has a medial and lateral pole (Fig. 6–3), and the lateral pole is readily palpable.[2] The posterior aspect of the condyle can be palpated if a fingertip is placed into the external auditory meatus and the pad of the finger is pushed anteriorly (Fig. 6–4).[6] The shape of each condyle may vary among individuals as well as side to side in the same individual.[6,10] The condyles are positioned anterior to the external auditory meatus and sit within the respective mandibular fossa of each of the temporal bones. The **coronoid process** is located anterior to the mandibular condyle and serves as an attachment for the temporalis muscle (Fig. 6–5).[2] The mandible interacts with the maxillae by way of the teeth. The importance of the articulations between the mandible and the temporal bones and maxillae will become apparent when we discuss impairments of the TM joints.

FIGURE 6-3

A superior view of the mandible shows the medial and lateral poles of the mandibular condyles and the coronoid processes. Mandibular rotation occurs around axes that pass through the medial and lateral poles of each condyle, with the extended lines of the axes intersecting anterior to the foramen magnum of the skull. Inset: In this right lateral view, the relationship of the lateral pole of the mandible to the

foramen magnum can be appreciated.



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FIGURE 6-4

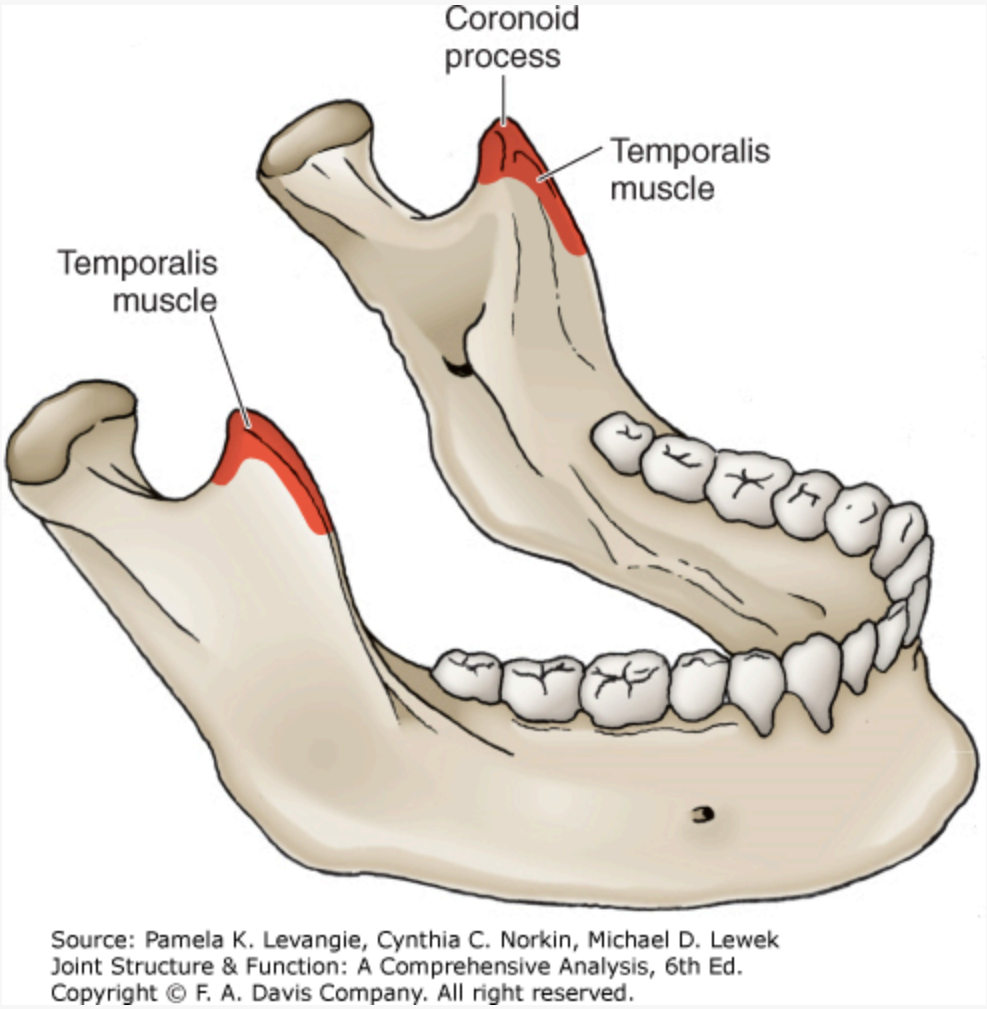
Palpation of the posterior mandibular condyle through the external auditory meatus, pushing fingertip(s) anteriorly.



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FIGURE 6-5

The temporalis muscle attaches to the coronoid process of the mandible.



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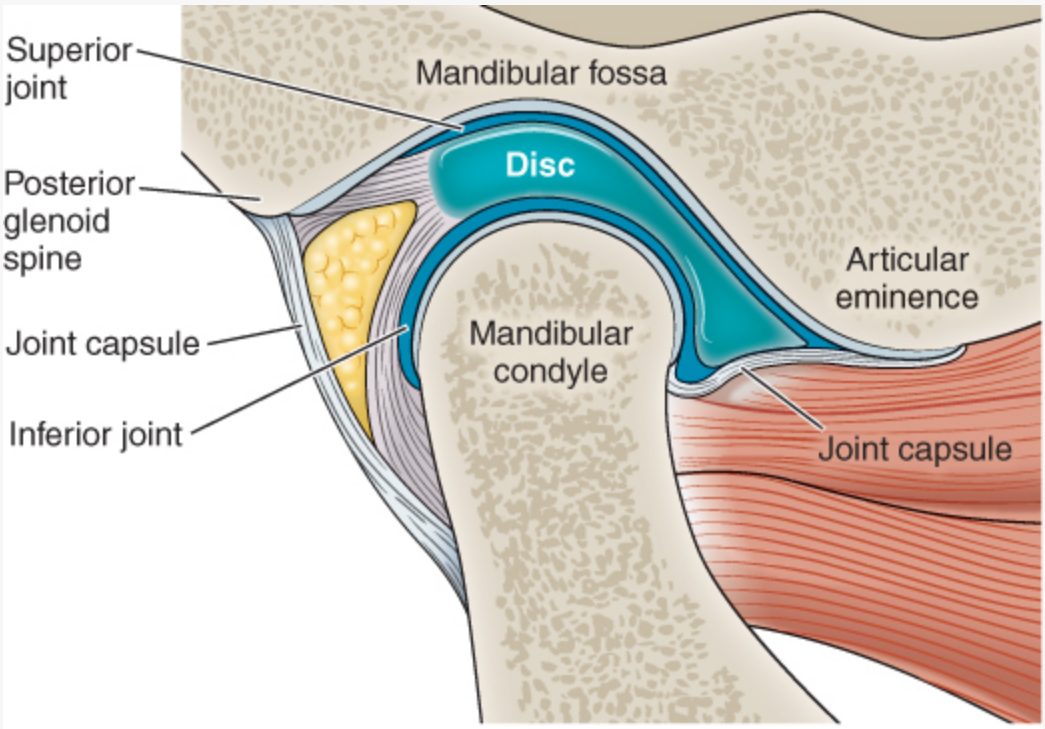
Accessory Joint Structures

The incongruence of the TM joint is addressed by a unique **articular disc.** A disc located within each TM joint separates the articulation into distinct superior and inferior joints with slightly different functions (Fig. 6–6).[2,6] Thus, mandibular motion involves the simultaneous movement of four different joints. The **inferior TM joint** is formed by the mandibular condyle and the inferior surface of the disc and functions as a simple hinge joint. The **superior TM joint** is larger than the inferior joint and is formed by the articular eminence of the temporal bone and the superior surface of the disc; it functions as a plane joint.[2] The articular disc of the TM joint is biconcave; that is, the superior and

inferior surfaces are both concave. Styles and Whyte describe the disc as having a "bowtie" appearance on magnetic resonance imaging (MRI) film, with the "knot" sitting at the thinnest portion.[11] The thickness of the articular disc varies within the joint from 2 mm anteriorly to 1 mm in the middle to 3 mm posteriorly (**Fig. 6–7**).[10–12] The purpose of the disc is to allow the convex surfaces of the articular eminence and the mandibular condyle to remain congruent throughout the ROM of the TM joint in all planes.[10] Sicher described the TM joint as a hinge joint with movable sockets, and later investigators supported this description.[5,10,13] The articular disc, therefore, serves multiple purposes. It increases stability, minimizes loss of mobility, reduces friction, and decreases biomechanical stress on the TM joint.[6,14-16]

FIGURE 6-6

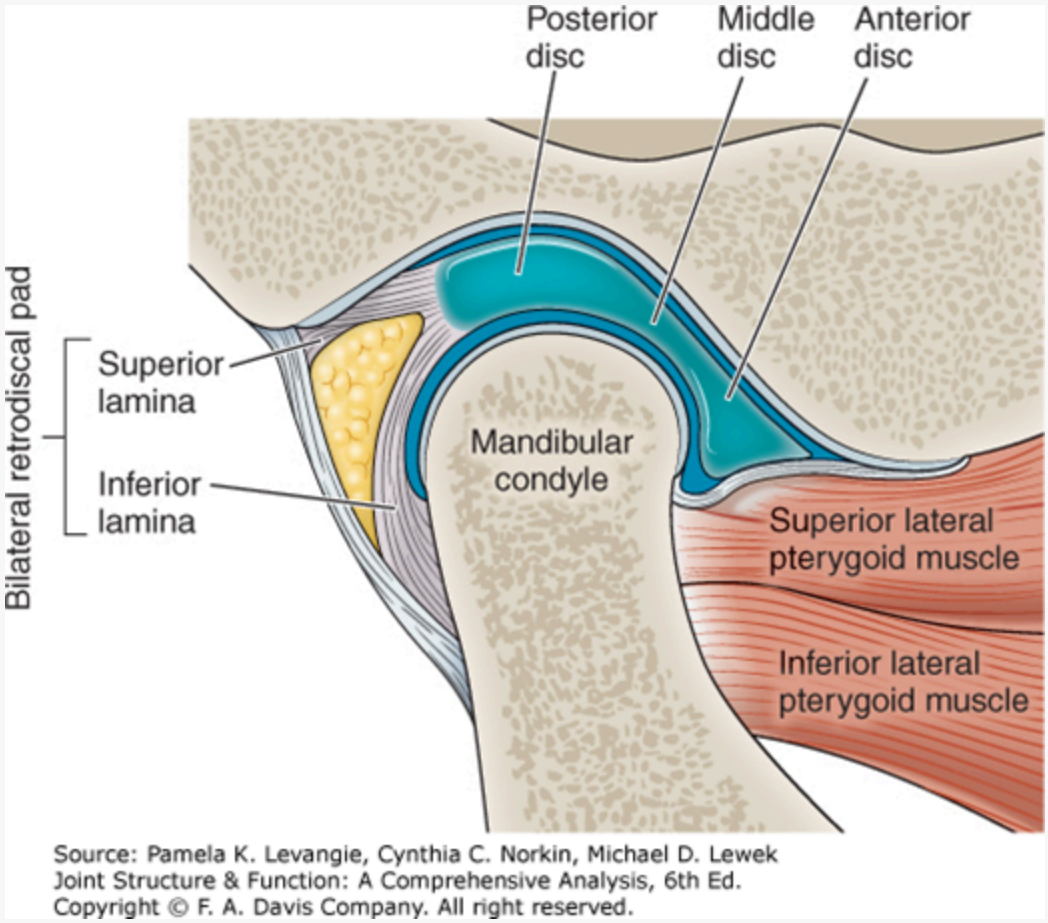
A cross-sectional lateral view of the TM joint shows the fibrocartilage-covered load-bearing surfaces on the condyle of the mandible and the articular eminence. The TM disc divides the articulation into superior and inferior joints, each with its own synovial lining. The anterior and posterior attachments of the joint capsule to the disc are shown.



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FIGURE 6-7

A cross-sectional lateral view of the temporomandibular joint showing the articular disc. The thickness of the disc varies within the joint, from 2 mm anteriorly to 1 mm in the center to 3 mm posteriorly.



The disc within each TM joint has a complex set of attachments (see Fig. 6–7). The disc is firmly attached to the medial and lateral poles of the condyle of the mandible, but it is not attached to the TM joint capsule medially or laterally.[10,17] These attachments allow the mandibular condyle to rotate freely on the disc in an anteroposterior direction. The disc is attached to the joint capsule anteriorly, as well as to the tendon of the **lateral pterygoid muscle.** The anterior attachments restrict posterior translation of the disc. Posteriorly, the disc is attached to a complex structure, collectively called the **bilaminar retrodiscal pad.** The two bands (or laminae) of the bilaminar retrodiscal pad are attached to the disc. The superior lamina is attached posteriorly to the tympanic plate (at the posterior mandibular fossa).[2,4,10] The superior lamina consists of elastic

fibers that allow the superior band to stretch. The superior lamina allows the disc to translate anteriorly along the articular eminence during mandibular depression (mouth opening); its elastic properties assist in repositioning the disc posteriorly during mandibular closing. The inferior lamina is attached to the neck of the condyle and is inelastic. The inferior lamina serves as a tether on the disc, limiting forward translation but does not assist with repositioning the disc during mandibular closing.[11,15] Neither of the laminae of the retrodiscal pad is under tension when the TM joint is at rest. Loose areolar connective tissue rich in arterial and neural supply is located between the two laminae.[4,15]

A healthy TM disc is viscoelastic and well suited for distribution of force, showing only minor changes in connective tissue fiber waviness even under significant stress.[18–20] The disc consists primarily of collagen, proteoglycans (PGs) and elastin. Collagen is largely responsible for maintaining the disc's shape. Elastin contributes to the disc's ability to regain its form during unloading. PG composition maintains disc resiliency and resists mechanical compressive force. The biomechanical behavior of the disc may change according to changes in its composition.[19] Such changes in composition may occur as a result of aging, mechanical stress, or both.[21] Unlike the fibrocartilage on the mandibular condyles and articular eminences, the articular disc of the TM joint does not have the ability to repair and remodel itself.[19,21]

The anterior and posterior segments of the disc are vascular and innervated. The central segment of the disc is avascular and aneural.[2,10,18] The lack of vascularity and innervation is consistent with the fact that the middle portion of the disc is the force-accepting segment. The capsule, lateral TM ligament, and retrodiscal tissues contain

mechanoreceptors and nociceptors, which are responsible for proprioception and pain sensation.[2]

Capsule and Ligaments

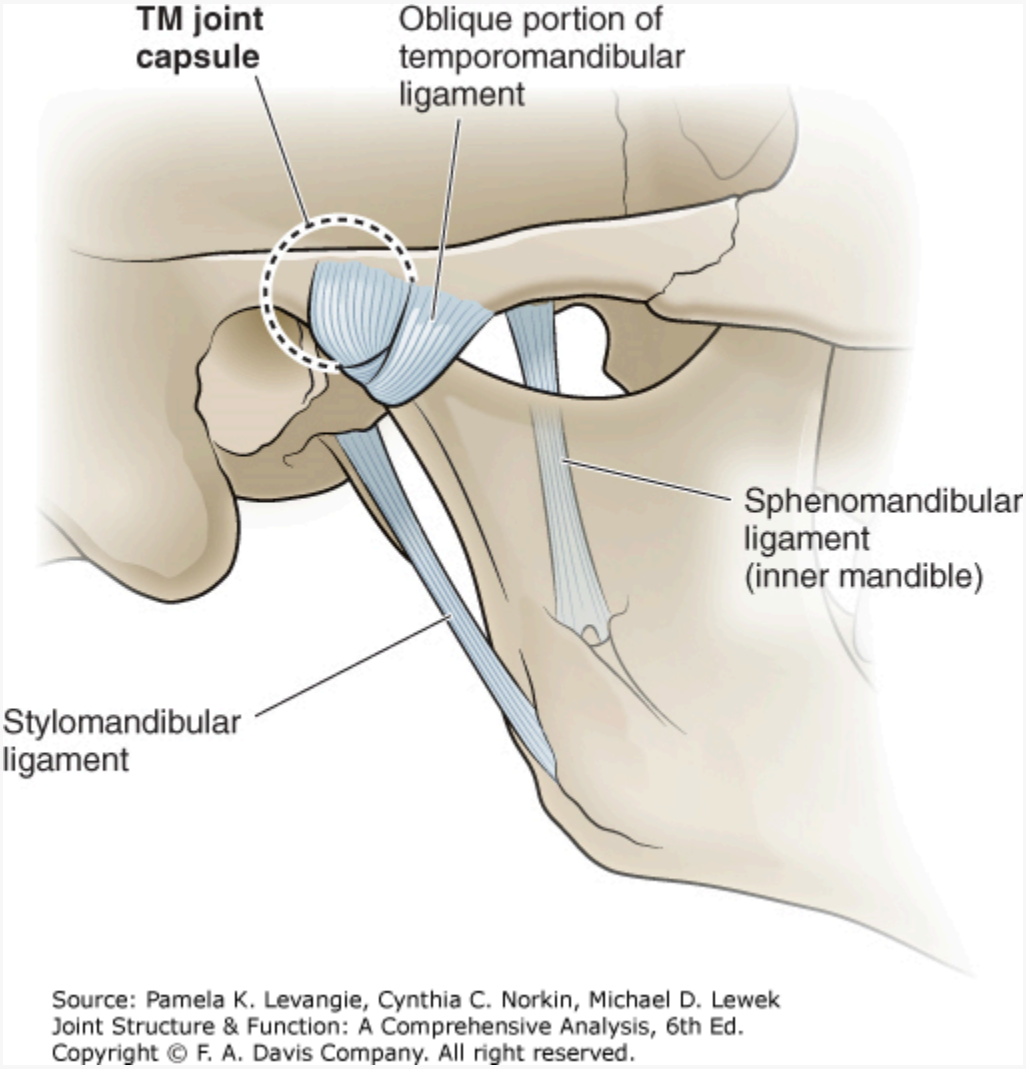
The elasticity of the joint capsule and ligaments determines the available motion at the TM joint in all planes. Motion can be enhanced or restricted depending on the flexibility of these structures. The portion of the capsule superior to the disc is quite lax, whereas the portion of the capsule inferior to the disc is taut.[4,10] Consequently, the disc is more firmly attached to the condyle below and freer to move on the articular eminence above. The capsule is thin and loose in its anterior, medial, and posterior aspects, but the lateral aspect is stronger and reinforced with long fibers (temporal bone to condyle).[4,17] The lack of strength of the capsule anteriorly and the incongruence of the bony articular surfaces predisposes the joint to anterior dislocation of the mandibular condyle.[22,23] The capsule is highly vascularized and innervated, which allows it to provide a great deal of information about position and movement of the TM joint.

The primary ligaments of the TM joint are the **TM ligament,** the **stylomandibular ligament,** and the **sphenomandibular ligament** (Fig. 6–8). The TM ligament is strong and composed of two parts: an outer oblique element and an inner horizontal element. The outer oblique element attaches to the neck of the condyle and the articular eminence. It serves as a suspensory ligament and limits downward and posterior motion of the mandible, as well as limiting rotation of the condyle during mandibular depression.[2,4,17] The inner horizontal component of the ligament is attached to the lateral pole of the condyle and posterior portion of the disc and to the articular eminence. Its fibers are aligned horizontally to resist posterior motion of the condyle. Limiting the posterior

translation of the condyle protects the retrodiscal pad.[24,25] The primary function of the TM ligament is to stabilize the lateral portion of the capsule.[2,4] Neither band of the TM ligament limits forward translation of the condyle or disc, but they do limit lateral displacement.[24–26]

FIGURE 6-8

A lateral view of the TM joint capsule and ligaments.



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The stylomandibular ligament is the weakest of the three ligaments and is considered a

thickened part of the parotid sheath joining the styloid process to the angle of the mandible.[4] Some investigators have identified the function of this ligament as limiting protrusion of the mandible,[24,25,27] but others have stated that it has no known function.[23]

The sphenomandibular ligament is described as the "strong" ligament that is the "swinging hinge" from which the mandible is suspended.[4,14] Some investigators have stated that it serves to protect the mandible from excessive anterior translation.[24,25,27] Others have stated that this ligament has little or no function.[4,25,28]

Function

Joint Kinematics

The TM joint is one of the most frequently used and mobile joints in the body. It is engaged during mastication, swallowing, and speaking.[2,10] Most of the time, the TM joint movements occur without resistance from chewing or contact between the upper and lower teeth.[10] However, the TM joint is designed to maintain its structure in spite of significant forces acting on it.[8] As previously noted, the articular surfaces are covered with a pseudofibrocartilage that has the ability to remodel and repair, thus able to tolerate repeated, high-level stress. Mastication requires tremendous power, while speaking requires intricate fine motor control. The musculature is designed to accomplish each of these potentially contradictory tasks.[8,9,21] Both **osteokinematic** and **arthrokinematic** movements are required for normal function of the TM joint. Osteokinematic motions include mandibular depression (mouth opening), elevation (mouth closing), protrusion, retrusion, and left and right lateral excursions.[2] Okeson provides a thorough overview of the detailed analysis of mandibular movements from

the classic work of Posselt.[29] Arthrokinematic movements involve rolling, anterior slide, distraction, and lateral glide.[2] This section describes the function of the various components of the TM joint during osteokinematic and arthrokinematic movements. For the purposes of this chapter, only movements that occur without resistance will be discussed.

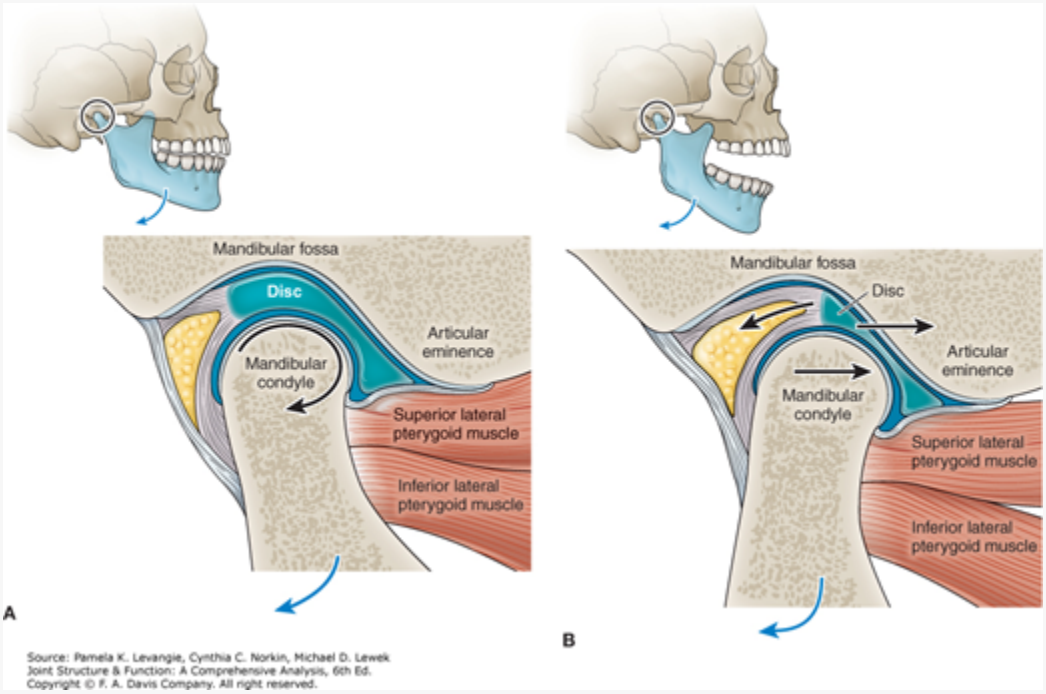
MANDIBULAR DEPRESSION AND ELEVATION

Mandibular depression and elevation are fundamental components of mastication.[2] Under normal circumstances, the motions of mandibular depression and elevation are relatively symmetrical, with the TM joints on both sides following a similar pattern. Mandibular depression and elevation each include a combination of translatory and rotary motion (curvilinear motion), where the mandibular condyle must roll and slide (Fig. 6–9).[2] Kraus stated that the literature is contradictory as to whether the rolling and sliding occur sequentially or concurrently.[6] Neumann, on the other hand, stated the rotation and translation are simultaneous.[2] During rotation, the mandibular condyle spins relative to the inferior surface of the disc in the lower joint (Fig. 6–9A). During translation, the mandibular condyle and disc slide together as a condyle-disc complex along the articular eminence in the superior joint (Fig. 6–9B).[2,6,14]

FIGURE 6-9

A. When the mouth begins to open, the motion at the TM joint may be limited to a spin of the condyle on the disc (clockwise rotation in this view). **B.** Anterior translation of the condyle and disc together on the articular eminence occur either after or simultaneously with rotation

in the later stages of mouth opening. The inelastic inferior lamina limits the amount of anterior translation whereas the elastic properties of the superior lamina control anterior translation.



Normal mandibular depression ROM is 40 to 50 mm when measured between the incisal edges of the upper and lower front teeth.[2,6,14] Mastication requires approximately 18 mm of mandibular depression.[2] Rolling occurs predominantly during the initial phase of mandibular depression with as little as 11 mm or as much as 25 mm of depression resulting from rotation of the condyle on the disc.[30] The remaining depression results primarily from anterior translation of the condyle-disc complex along the articular eminence. The shape of the condylar head and the steepness of the articular eminence positively correlate with the amount of rotation, but both configurations can be asymmetrical from one TM joint to the other, thus affecting the symmetry of motion.[6,30,31] As a quick screen, the clinician may use the adult knuckles

(proximal interphalangeal joints) to assess the degree of mandibular depression. Two knuckles placed between the upper and lower incisors is considered functional mandibular depression whereas three knuckles is considered normal (Fig. 6–10).[2,6] Gravity assists with mandibular depression, with the mandibular muscles of elevation possibly contributing eccentrically to control depression.[17]

FIGURE 6-10

Mandibular depression (mouth opening) is considered functional if the proximal interphalangeal joints of two fingers can be inserted between the teeth and normal if the joints of three fingers can be inserted.



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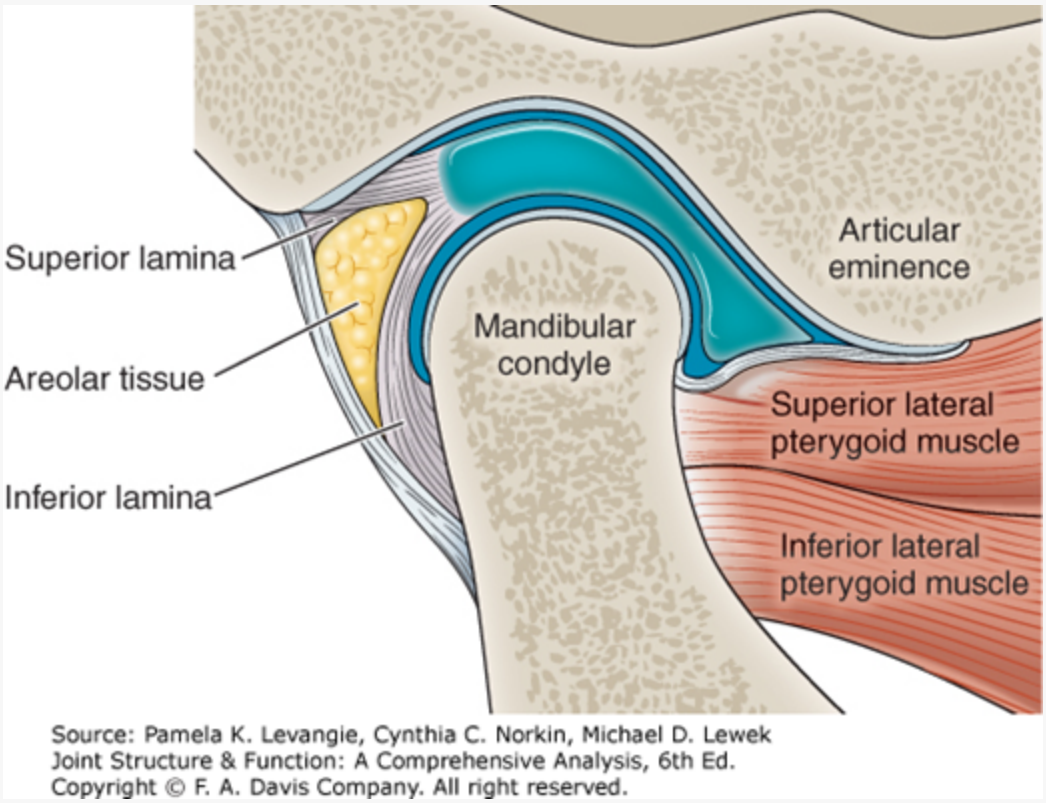
Mandibular elevation is the reverse of mandibular depression. The mandibular condyles rotate posteriorly on the disc in the inferior joint, and the condyle-disc complex translates posteriorly in the superior joint.

Active and passive control is exerted on the articular disc through its connections during mandibular depression and elevation (**Fig. 6–11**). Passive control occurs through the capsuloligamentous attachments of the disc to the condyle. The lateral pterygoid muscle attaches to the anterior portion of the disc, producing active control,[30] although evidence suggests that this attachment may not be consistently present.[32] During mandibular depression, the medial and lateral attachments of the disc to the condyle

limit the motion between the disc and condyle to rotation (see Fig. 6–9A). As the condyle translates (see Fig. 6–9B), the biconcave shape of the disc causes it to track anteriorly along with the condyle without any additional active or passive assistance. However, the inelastic inferior retrodiscal lamina limits forward excursion of the disc. The superior portion of the lateral pterygoid muscle attaches to the disc and appears to be positioned to assist with anterior translation; however, no activity is noted during mandibular depression as expected if the muscles assisted with this motion.[6,33]

FIGURE 6-11

The TM disc attaches posteriorly to the joint capsule and to the superior and inferior laminae (segments of the bilaminar retrodiscal pad). The disc attaches anteriorly to the joint capsule and to the lateral pterygoid muscle.



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During mandibular elevation (mouth closing), the elastic character of the superior retrodiscal lamina applies a posteriorly directed force on the disc. In addition, the superior portion of the lateral pterygoid demonstrates activity that is assumed to eccentrically control the posterior movement of the disc while maintaining the disc in an anterior position until the mandibular condyle completes posterior rotation to the normal resting position.[2,14,16,19,33] Abe and colleagues suggested that the sphenomandibular ligament also assists this action.[34] Again, the medial and lateral attachments of the disc to the condyle limit the motion to rotation of the disc around the condyle.[17]

Other muscle segments may also assist with maintaining the disc position during active movement, including the masseter muscle, which attaches to the anterolateral portion

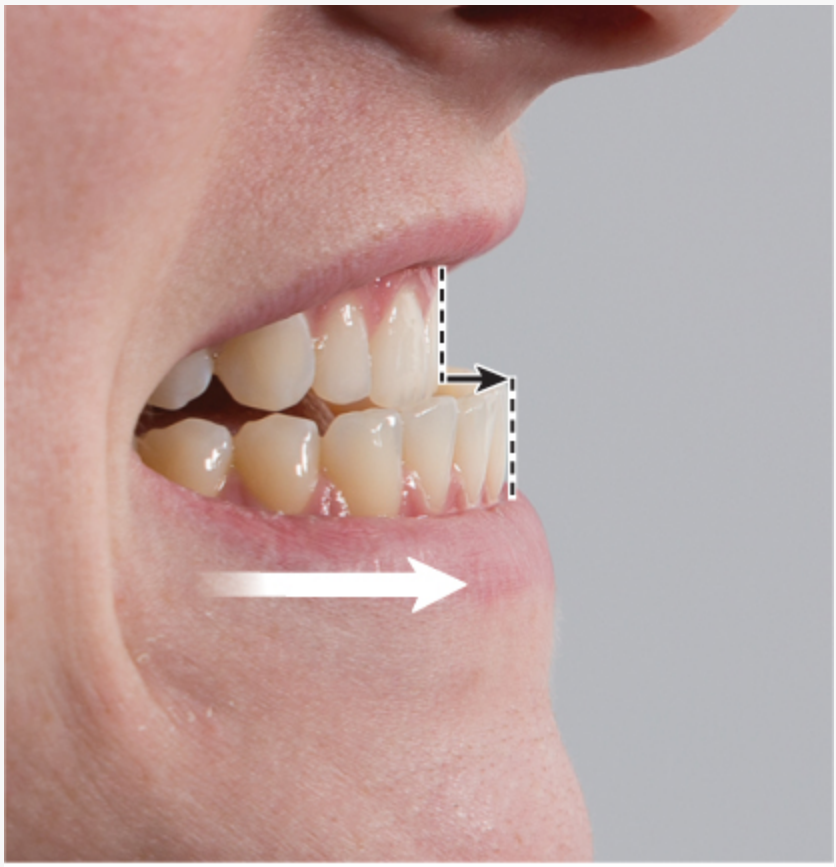
of the disc and may counteract the medial pull of the anteromedially directed lateral pterygoid.[10,33] The masseter, temporalis, and medial pterygoid muscles provide a constant pressure on the disc that is necessary to prevent dislocation.[33]

MANDIBULAR PROTRUSION AND RETRUSION

Mandibular protrusion and retrusion occur in the superior TM joint. The condyle-disc complex translates in an anterior inferior direction, following the downward slope of the articular eminence during protrusion, returning along a posterior superior path.[2] Rotation of the condyles is not present during protrusion and retrusion. The teeth are separated during these motions. Ideally, the lower teeth should surpass the upper teeth by several millimeters (Fig. 6–12); however, protrusion is considered adequate when the upper and lower front incisal edges touch.[6] Protrusion (anterior translation of the condyle and disc) is an important component necessary for maximal mandibular depression. Retrusion is an important component of mandibular elevation from a maximally depressed mandible.[2]

FIGURE 6-12

With maximum mandibular protrusion, the lower teeth should be in front of the upper teeth.



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During protrusion, the posterior attachments of the disc (the bilaminar retrodiscal tissue) stretch 6 to 9 mm to allow completion of the motion.[14,27] The degree of retrusion is limited by tension in the TM ligament as well as by compression of the soft tissue in the retrodiscal area between the condyle and the posterior glenoid spine. An estimated 3 mm of translation occurs during retrusion; however, this motion is rarely measured.[14]

MANDIBULAR LATERAL EXCURSION

Lateral excursion involves moving the mandible to the left and to the right. The degree of lateral excursion considered normal for an adult is 8 to 11 mm.[2,6] One functional screen to estimate whether this motion is normal is to observe whether the mandible

can move the full width of one of the **central incisors** in each direction (Fig. 6–13).[6] Active lateral excursion is described as *contralateral* (the opposite side) or *ipsilateral* (the same side) relative to the primary muscle action.[2] To accomplish lateral excursion, the ipsilateral mandibular condyle spins around a vertical axis within the mandibular fossa, whereas the contralateral mandibular condyle translates anteriorly along the articular eminence. A slight degree of spin and lateral slide of the contralateral mandibular condyle is necessary to achieve maximal lateral excursion (Fig. 6–14).[6]

FIGURE 6-13

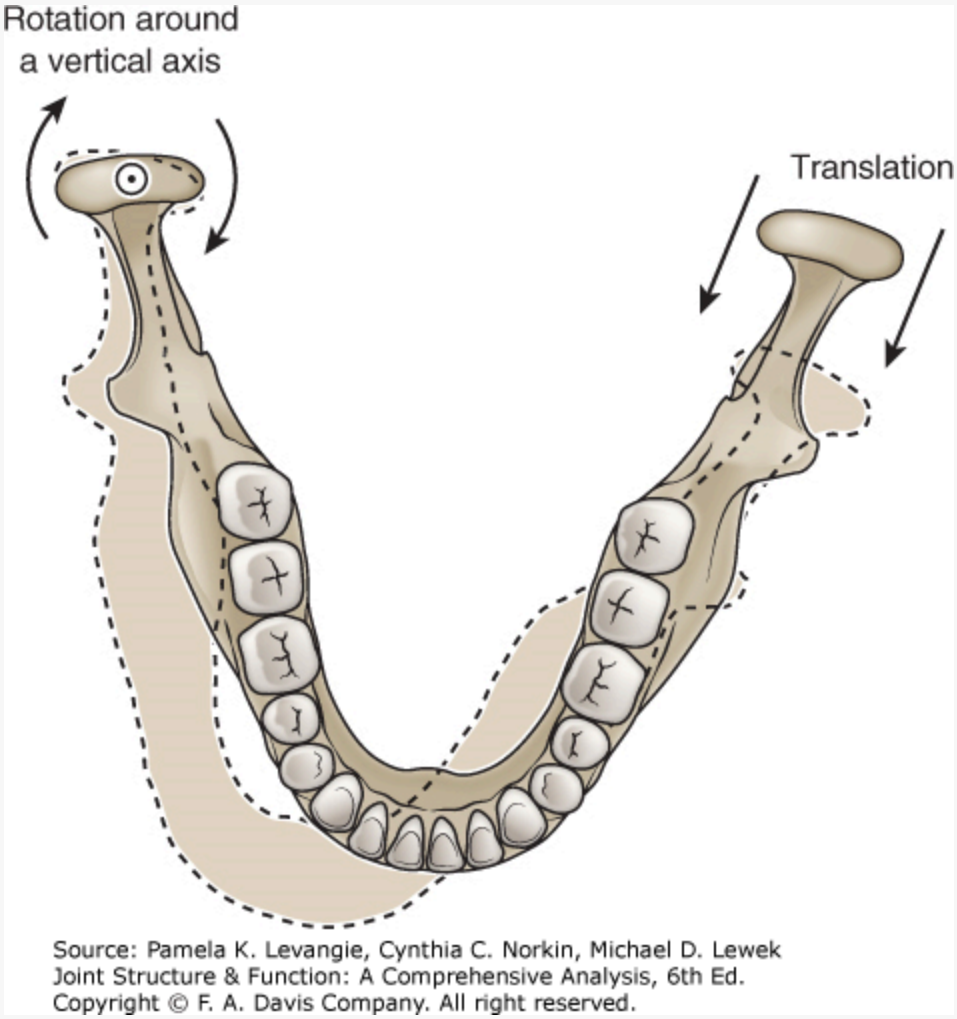
With normal lateral deviation of the mandible to the right, the midline of the lower teeth should move the full width of the right upper central incisor.



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FIGURE 6-14

In this superior view of the mandible, lateral deviation of the mandible (chin) to the right occurs effectively as a rotation (spin) of the right condyle around a vertical axis, and the left condyle translates anteriorly.



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Another normal asymmetrical movement of the TM joint involves rotating one condyle around an anteroposterior axis while the other condyle depresses.[14] This movement results in a frontal plane motion of the mandible, with the chin moving downward and deviating from the midline toward the condyle that is spinning. These motions are generally combined into one complex motion used for chewing and grinding food.[35]

Deviations and **deflections** may be noted during osteokinematic movements of the mandible. A deviation is a motion that produces an “S” curve as the mandible moves away from the midline during mandibular depression or protrusion and returns to

midline at end range. A deflection is a motion that creates a “C” curve, with the mandible moving away from midline during mandibular depression or protrusion without a return to midline by end range. Deviations and deflections may result from mandibular condyle head shapes differing from right to left. If no other signs or symptoms accompany these asymmetries, then deviation or deflection is considered inconsequential. However, as will be discussed later in this chapter, deviations and deflections may indicate a pathology.[6]

Muscles

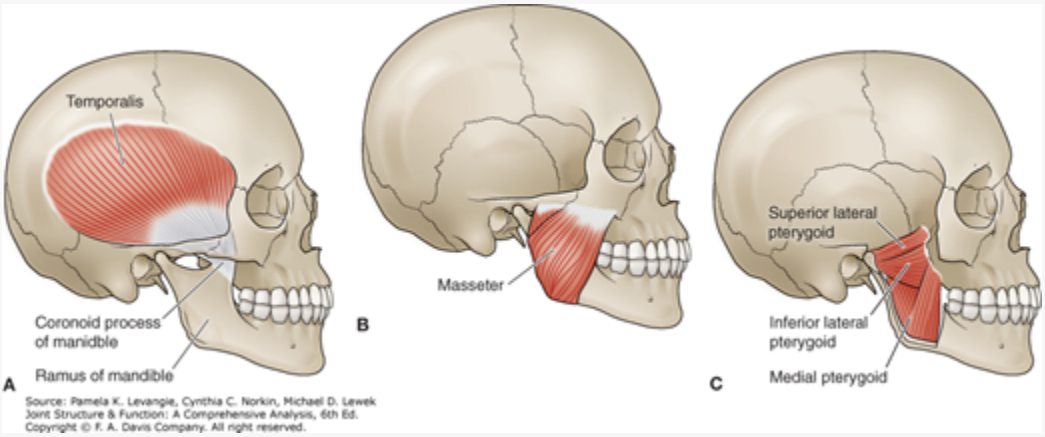
The muscles of mastication include the medial and lateral pterygoids, masseter, and temporalis, along with other accessory muscles. Mastication involves depression and elevation of the mandible and lateral excursion. Specifically, the lateral pterygoid, stylohyoid, mylohyoid, and geniohyoid muscles, with assistance from the digastric muscle, depress the mandible, whereas the temporalis, medial pteryogoid, and masseter muscles elevate the mandible. Protrusion is accomplished by the muscles responsible for mandibular depression, whereas retrusion predominately occurs as the mandible settles back into its rest position in the glenoid fossa. Alternate contraction of the inferior belly of the lateral pterygoid is responsible for lateral excursion of the mandible.[10] The information below provides details specific to the muscles of mastication.

PRIMARY MUSCLES

The muscles acting on the TM joint are divided into primary and secondary muscle groups (**Fig. 6–15**). The primary muscles include the temporalis, masseter, lateral pterygoid, and medial pterygoid.[5]

FIGURE 6-15

Primary TM joint muscles. **A.** The temporalis muscle attaches to the medial aspect of the coronoid process and ramus of the mandible. **B.** The masseter muscle attaches to the external surface of the ramus of the mandible. **C.** The superior and inferior lateral pterygoid and medial pterygoid muscles lie inside the mandible (shown cut away here), attaching to the neck of the mandible, disc and TM joint capsule.



The temporalis is a flat, fan-shaped muscle that is wide at the proximal portion and narrow at the inferior portion (Fig. 6–15A). The superior fibers attach to the cranium, whereas the inferior fibers attach to the coronoid process and the medial surface of the ramus of the mandible. The temporalis fills the concavity of the temporal fossa and can be palpated easily over the temporal bone.[2,4] The masseter is a thick, powerful muscle with its superior attachments on the zygomatic bone and its inferior attachment on the external surface of the ramus of the mandible (Fig. 6–15B). This muscle can be palpated easily superior to the angle of the mandible.[4] Okeson noted that the temporalis and masseter are responsible for elevating the mandible. Additionally, the temporalis

assists with retrusion whereas the masseter contributes to protrusion.[29] Van der Bilt and colleagues discovered that the bilateral masseters and anterior temporalis muscles were 30% more active during bilateral maximum voluntary bite force than during unilateral bite force.[9] Furthermore, these investigators discovered that the muscle activity of the masseters was symmetrical with unilateral clenching. However, the muscle activity during unilateral clenching was significantly greater for the ipsilateral anterior temporalis muscles than for the contralateral anterior temporalis muscles.[9]

The lateral pterygoid, as already noted, consists of superior and inferior segments (**Fig. 6–15C**) that travel in a horizontal direction and combine posteriorly to attach to the neck of the mandible, the articular disc, and the joint capsule (see Fig. 6–11).[2,4] The literature is contradictory as to whether this muscle can be palpated. The lateral pterygoid muscles are thought to assist with mandibular depression.[3,17,36] Okeson noted the superior fibers stabilize the condyle and disc during mandibular loading, whereas the inferior fibers protrude the mandible and contribute to lateral movements and mandibular depression.[29,33]

The medial pterygoid parallels the masseter in line-of-force and size. The superior fibers attach to the medial surface of the lateral pterygoid plate on the sphenoid bone, and the inferior attachment is on the internal surface of the ramus near the angle of the mandible (see Fig. 6–15C).[2,4] The medial pterygoid muscles elevate the mandible and contribute to protrusion.[29]

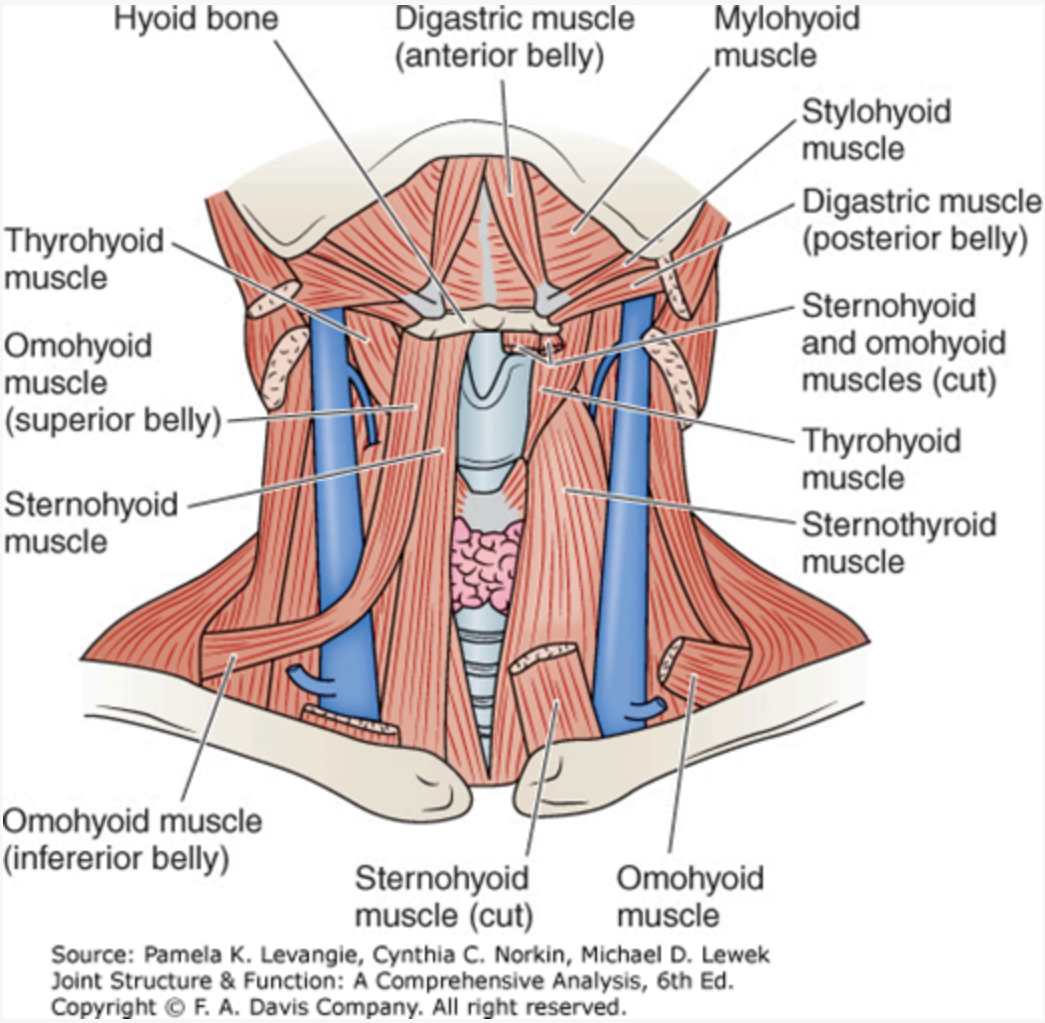
SECONDARY MUSCLES

The secondary muscles are smaller than the primary muscles and consist of the **suprahyoid** and **infrahyoid groups** (Fig. 6–16). The digastric, geniohyoid, mylohyoid, and

stylohyoid muscles compose the suprahyoid group. The infrahyoid group includes the **omohyoid, sternohyoid, sternothyroid,** and **thyrohyoid** muscles.[2] The suprahyoid muscles assist with mandibular depression, whereas the infrahyoid muscles are responsible for stabilizing the hyoid bone. Both the suprahyoid and infrahyoid muscle groups are involved in speech, tongue movements, and swallowing.[2] The suprahyoid muscles attach between the base of the cranium, the hyoid, and the mandible. The superior fibers of the infrahyoid muscles attach to the hyoid and the inferior fibers attach to the thyroid cartilage, sternum, and scapula.

FIGURE 6-16

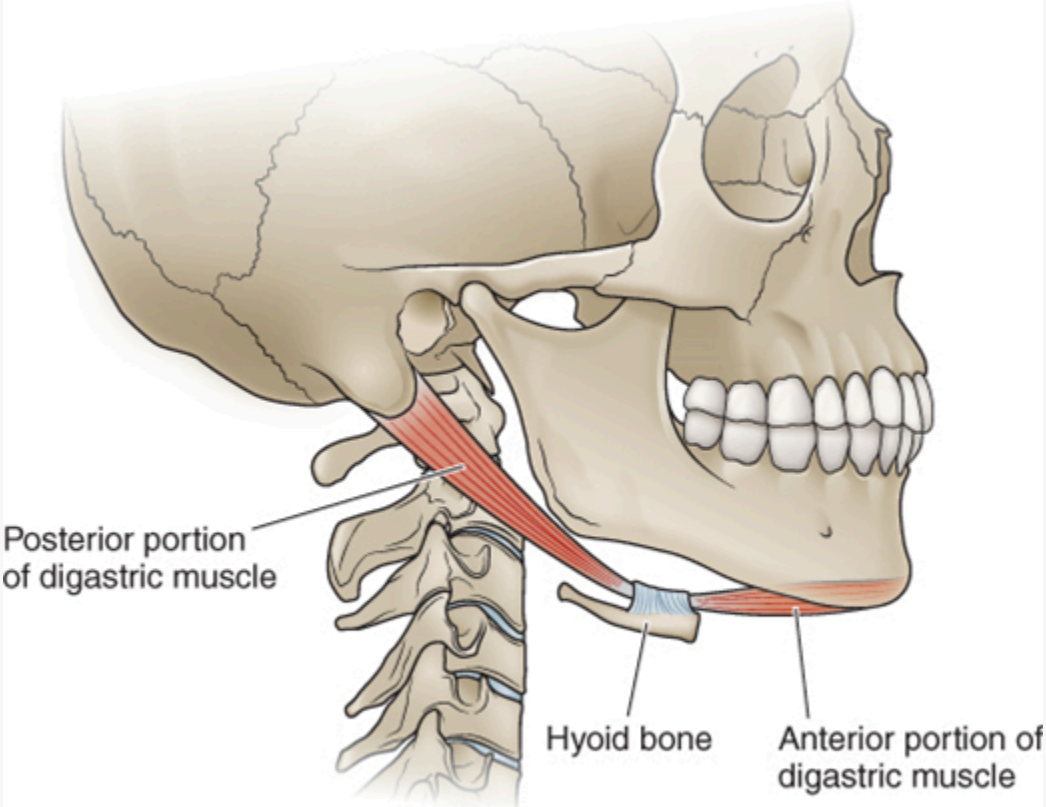
The suprahyoid and infrahyoid muscle groups shown from an anterior view of the neck.



The posterior fibers of the digastric muscle attach to the mastoid notch and the anterior fibers attach to the inferior mandible; the tendon joining the anterior and posterior segments is connected to the hyoid bone in the neck by a fibrous loop (Fig. 6–17).[2] The digastric muscle is predominantly responsible for mandibular depression, but also plays a role in elevating the hyoid bone.[29] The hyoid bone has to be stabilized for the digastric muscle to depress the mandible. This stabilization is provided by the infrahyoid muscles.[2]

FIGURE 6-17

The posterior portion of the digastric muscle arises from the mastoid notch and the anterior portion arises from the inferior mandible. The tendon that joins the anterior and posterior portions is connected by a fibrous loop to the hyoid bone in the neck.



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The muscles associated with the TM joint are innervated directly or by branches of the cranial nerves. The neurological evidence supports the possibility of inaccurate diagnoses of trigeminal neuralgia or an ear problem when in fact the person may have a TM joint condition. Loughner and colleague[28] suggest that pain felt in the ear may be referred from the TM joint as a result of injury or inflammation.[28]

COORDINATED MUSCLE ACTIONS

Mandibular depression occurs from the concentric action of the bilateral digastric muscles in conjunction with the inferior portion of the lateral pterygoid muscles. Mandibular elevation results from the collective concentric action of the bilateral masseter, temporalis, and medial pterygoid muscles. The bilateral superior lateral pterygoid muscles eccentrically control the TM discs as the mandibular condyles relocate into the mandibular fossa with mandibular elevation.[2,32]

The other mandibular motions of protrusion, retrusion, and lateral deviation are produced by the same muscles that elevate and depress the mandible, but in different sequences. Mandibular protrusion is produced by the bilateral action of the masseter, medial pterygoid, and lateral pterygoid muscles,[6,32] Retrusion is generated through the bilateral action of the posterior fibers of the temporalis muscles, with assistance from the anterior portion of the digastric muscle.[32] Lateral deviation of the mandible is produced by the unilateral action of a selected set of these muscles. The medial and lateral pterygoid muscles each deviate the mandible to the opposite side.[32] The temporalis muscle can deviate the mandible to the same side. Although the temporalis and lateral pterygoid muscles on the left appear to create opposite motions of the mandible, concomitant contractions of the right lateral pterygoid and right temporalis muscles function as a force couple. The lateral pterygoid muscle is attached to the medial pole of the condyle and pulls the condyle forward. The temporalis muscle on the ipsilateral side is attached to the coronoid process and pulls it posteriorly. Together these muscles effectively spin the condyle to create deviation of the mandible to the left. Because the temporalis muscle is also an elevator of the mandible, this combination of muscular activity is particularly useful in chewing.

Patient Application 6–1

Jill Smith is a 48-year-old insurance actuarial who is a single parent to three young children. She is complaining of jaw pain with mouth opening, as well as intermittent headaches and ear pain. Jill's medical history is unremarkable except for a history of serious hay fever since childhood. After questions about a history of trauma, she recalled a biking accident when she was 12 years old where she flew forward off the seat of her bike, hitting her chin hard enough on the handle bars that she had to have a couple of stitches.

Upon examination, Jill demonstrates decreased active ROM with mandibular depression, protrusion, and left lateral excursion. Passive mobility of the right TM joint is limited in distraction and anterior translation, as well as lateral slide. Her mandible deflects to the right with mandibular depression. Palpation of the mandibular condyles reveals a larger space on the left than the right during active mandibular depression and protrusion. These findings indicate that the right mandibular condyle and disc are not translating as far anteriorly as the left mandibular condyle and disc. The asymmetry may be due either to hypomobility on the right or to hypermobility on the left, but the differential (asymmetrical) activity or stretch on muscles and capsuloligamentous structures may be the source of Jill's jaw pain.

Relationship to the Cervical Spine and Posture

The cervical spine and TM joint are intimately connected. A biomechanical relationship exists between the position of the head, the cervical spine, and the dentofacial structures.[31,37] The proximal and distal attachments of the primary and secondary TM muscles show both direct and indirect connections to the cervical spine, throat, clavicle, and scapula, which implies that dysfunction in these areas (e.g., postural deviations) can affect the TM joint, or visa versa. Head and neck position may affect tension in the cervical muscles, which may in turn influence the position or function of the mandible.[37–39] Correct posture minimizes the forces produced by the cervical spine extensors as well as the other cervical muscles necessary to support the weight of the

head. Over time, improper posture can lead to adaptive shortening or lengthening of the muscles around the head, cervical spine, and upper quarter (including all structures of the upper trunk from the scapulae superiorly). Improper posture may result in an alteration in ROM, the ability to produce muscular forces, and joint morphology. Many of the symptoms associated with TM joint dysfunction are similar to the symptoms associated with primary cervical spine impairment. Therefore, the cervical spine and upper quarter should be thoroughly examined when clients present with TM joint complaints.[37,39]

Expanded Concepts 6–1

TM/Cervical Joint Interrelationships

Pain often occurs concurrently in the head, neck, and jaw as the result of faulty posture. According to Kendall, forward head posture involves mechanical changes that stimulate the nociceptors that produce pain.[40,41] Forward head posture consists of extension of the occiput and the cervical spine, leading to compensatory flexion of the cervicothoracic junction and upper thoracic spine to achieve a level head position (Fig. 6–18). In a forward headposition, the cervical extensors are in a shortened position and will adaptively shorten over time, whereas the anterior cervical muscles are in an elongated position and develop stretch weakness.[40] Concomittantly, the occiput is extended on the atlas (C1), creating adaptive shortening of the suboccipital tissues, including the anterior atlantoaxial and atlanto-occipital ligaments, the posterior belly of the digastric muscles, the stylohyoid muscles, the upper fibers of the upper trapezius, and the semispinalis capitis and splenius capitis muscles.[42] The forces necessary to maintain the head against gravity with faulty cervical posture and forward head result in muscle imbalances and altered movement patterns. Such alterations typically lessen the capacity of particular structures to meet the thresholds for adaptive responses to physical stresses.

Increased tension from shortening of the suboccipital tissues may lead to headaches that originate in the suboccipital area, limitation in active ROM of the upper cervical spine, and TM joint dysfunction. Furthermore, pain in the TM region may be referred

from the cervical region.[41,43] Thus, cervical posture should be normalized to successfully treat dysfunction in the TM joint complex.[43] Andrade and colleagues found that individuals with TM dysfunction report higher perceptions of pain with palpation of the cervical muscles.[44]

FIGURE 6-18

Poor cervical posture, such as forward head shown here, increases the physical demands on the suboccipital structures, contributing to TM joint dysfunction.



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Patient Application 6–2

In addition to her TM joint asymmetry, Jill Smith is seen upon postural examination to have a forward head (see Fig. 6–18) with rounded shoulders, winging scapulae, increased thoracic kyphosis, increased lumbar lordosis, hyperextended knees, and

overpronated feet. The noted elevation of her right shoulder may imply tightness of the suboccipital and cervical musculature, consistent with her limited active ROM when performing mandibular depression, protrusion, and left lateral excursion. Jill reports tenderness with palpation of the muscles at the base of her head, the right side of her face, and under her chin. Tenderness and muscle guarding may be related to the stresses placed on the tissues from improper positioning.[41]

Dentition

Occlusion, or contact of the teeth, is intimately involved in the function of the TM joint because, in part, chewing is one of its functions. Although the teeth are together for approximately 15 minutes of each day, the presence and position of the teeth are critical to normal TM joint function. Contact of the upper and lower teeth limits motion of the TM joint during empty mouth movements. The complexities of the TM joint and the interrelated issues with the teeth underscore the necessity of the comprehensive management of TM joint disorders. Normal adult dentition includes 32 teeth divided into four quadrants. The only teeth we will refer to by name are the upper and lower central incisors. These are the two central teeth of the maxilla and the two central teeth of the mandible.[8,45] When the central incisors are in firm approximation, the position is called **maximal intercuspation** or the **occlusal position**.[46] This position is not the normal resting position of the mandible. Rather, there is typically 1.5 to 5 mm of **"freeway space"** between the upper and lower incisors.[47] By maintaining this freeway space, the intra-articular pressure within the TM joint is decreased, the stress on the articular structures is reduced, and the tissues of the area are able to rest and repair.[8]

The American Academy of Orofacial Pain and others have identified occlusion as a causative factor in TM disorders.[1] However, the literature has been contradictory and inconclusive. Lack of uniformity in research methods and techniques has been

suggested as the source of these discrepancies. Peck noted that occlusion is not necessarily a predictor of TM disorders as individuals with and without TM disorders demonstrate similar occlusal patterns.[8] He also noted that altered articular patterns of the TM joint could occur as the result of occlusal changes. Clinicians should be aware that dental occlusion and TM disorders are more than a biomechanical concern. The biobehavioral aspect must be included in treating patients with TM disorders.[8]

Sonnesen and colleagues studied a sample of 96 children with severe malocclusion prior to orthodontic intervention.[38] These investigators identified a direct correlation between the occurrence of muscle tenderness in the masticatory muscles and a long-face craniofacial morphology. They provided two possible explanations: (1) the muscle tenderness could be the result of functional overloading of weak mandibular elevator muscles, or (2) the muscle tenderness may lead to temporary hypofunction of the masticatory muscles, resulting in decreased bite force. Additionally, the investigators confirmed an association in the studied sample between posture and TM dysfunction. Clicking (joint noise), locking of the mandible, and asymmetric mandibular depression were noted in children with a marked forward inclination of the cervical column and an increased craniocervical angle.[38,39]

Life Span and Clinical Considerations

TM joint dysfunction is a vague term that encompasses numerous clinical problems that involve the masticatory system. Mechanical stress is the most critical factor in the multifactorial etiology.[18,38] Dysfunction of either the muscles or the joint structure is generally at fault. Most clients with TM dysfunction will not fit into a specific category of

dysfunction classification, which creates a clinical challenge.[48] Additionally, only 20% to 30% of individuals with internal derangement of the TM joint develop symptomatic joints.[17] These symptoms may progress or resolve spontaneously.[11] De Paiva Bertoli and colleagues identified the most common signs of TM dysfunction in children with headaches as (from most to least frequent): joint pain, muscular pain, joint noise, and mandibular deviation.[49] Pathomechanics of this joint may result from faulty posture, an isolated traumatic event such as a fall, cervical whiplash, or chronic inflammation of the TM joint resulting from indirect trauma. As with many disorders, the exact pathomechanics are often unknown. Although treatment of TM dysfunction is beyond the scope of this text, collaboration between dentists, orofacial pain specialists, physical therapists, and counselors provides the best functional outcomes.[39]

Determining whether a client has TM dysfunction requires an evidence-based diagnostic process. This process should include a thorough clinical assessment performed by a TM clinician, classification of patients according to the *Research Diagnostic Criteria for TM Disorders,*[48] and MRI, the diagnostic gold standard. Because of the complexity associated with TM joint dysfunction, clinicians should seek advanced education before attempting to manage this patient clientele. Common etiologies of TM joint dysfunction include aging, inflammatory conditions, capsular fibrosis, osseous mobility, articular disc displacement, and degenerative conditions.

Age-Related Changes in the TM Joint

The aging process affects the joints of the human body and the TM joint is no exception. However, degenerative changes are not always the result of the normal aging process and may occur from a preexisting dysfunction. Furthermore, degenerative changes do

not necessarily indicate disability. Rowe and Kahn described successful aging as "multidimensional, encompassing the avoidance of disease and disability, the maintenance of high physical and cognitive function, and sustained engagement in social and productive activities."[50] With progression through life, tissues become less supple, less elastic, and less able to withstand maximal forces. Pathology or biomechanical dysfunction will not necessarily result from these changes.[50] Alexiou and colleagues conducted a retrospective study of computer data from 71 individuals aged 20 to 75 with characteristics of degenerative arthritis on diagnostic imaging.[51] The authors noted flattening, resorption, and sclerosis in the condylar head of the mandible in 56%, 43%, and 25% of the patients, respectively. They concluded that osteoarthritis of the TM joint is an age-related degenerative disease in patients over the age of 40.[51] Nannmark and colleagues examined cadaver mandibular condyles from 37 TM joints of people ages 55 to 99.[52] The investigators reported structural changes in 38% of the mandibular condyles examined. No signs of inflammatory cell infiltration were found, suggesting that the observed changes were secondary to biomechanical stresses rather than the result of inflammation. Twenty-two (59%) of the articular discs had perforations, roughness, or were thinned. However, only three (8%) of the discs were in an anterior position, and each of these was perforated. The investigators concluded that osteoarthritis may be expected in 14% to 40% of adults, with increased frequency with age in men and women.[52]

Clinical Considerations

De Leeuw and colleagues performed radiography and MRI studies of 46 former patients 30 years after the diagnosis of osteoarthritis and internal derangement of the TM joint.[53] **Internal derangement** is described as the abnormal position and function of the disc

and articulating surfaces.[17] The investigators incorporated into their study 22 age-matched controls without known TM joint dysfunction.[53] Signs noted on the radiographs were more common and severe in the former patient group. MRI findings revealed a higher percentage of osteoarthritis and internal derangement in the dysfunctional TM joints as well as in the contralateral TM joints. However, degenerative changes in the contralateral TM joints appeared to have been asymptomatic, with only 25% reporting any symptoms and none of those seeking intervention. MRI evidence of the controls revealed infrequent osteoarthritis and internal derangement.[53]

The work conducted by Nannmark and colleagues and by de Leeuw and colleagues indicates that TM joint degeneration does not necessarily occur with aging and that degenerative changes evident on radiograph or MRI are not necessarily associated with symptoms or dysfunction.[52,53]

Respiratory Dysfunction and TM Joint

Children with chronic respiratory allergies may develop TM joint dysfunction as a result of dysfunctional growth and developmental patterns.[39] For example, a child with allergies who has difficulty breathing through the nose will often hyperextend the cervical spine, assuming the forward head position, to increase the diameter of the upper respiratory tract and improve ventilation.[39,54] This posture changes the dynamics of the TM joint. The upper and lower teeth contact each other and may affect the resting position of the TM joint. Thus, the muscles surrounding the TM joint complex expend greater energy to maintain this posture. Blocked nasal passages impede inhalation and promote the use of accessory muscles of respiration (scalene and sternocleidomastoid muscles) to assist with breathing. Use of accessory muscles may lead to a forward head

posture.[55] Over time, a forward head posture contributes to a cycle of increasing musculoskeletal dysfunction,[56] including repeated episodes of TM inflammation that can result in fibrosis of the TM joint capsule.[35]

Inflammatory Conditions

Inflammatory conditions of the TM joint include **capsulitis** and **synovitis.** Capsulitis involves inflammation of the joint capsule, and synovitis is characterized by fluctuating edema caused by effusion within the synovial membrane of the TM joint. Rheumatoid arthritis is the most common cause of synovial membrane inflammation, but gout, psoriatic arthritis, ankylosing spondylitis, systemic lupus erythematosus, juvenile chronic arthritis, and calcium pyrophosphate dehydrate deposition may also contribute to inflammation of the synovia.[17] Individuals with inflammatory conditions experience pain and inflammation within the joint complex, which may diminish mandibular depression.[6] Unresolved inflammation can lead to adhesions that restrict the movement of the disc and limit the function of the TM joint.[11]

Rheumatoid arthritis is a chronic systemic condition with articular and extra-articular involvement. The primary symptoms of rheumatoid arthritis include pain, stiffness, edema, and warmth. This autoimmune disorder targets the joint capsule, ligamentous structures, and synovial lining of the joint complex, resulting in joint instability, joint deformity, or ankylosis.[57] Multiple bilateral joints are typically involved with this disease. A detailed discussion of rheumatoid arthritis is beyond the scope of this text; however, clinicians should be aware that rheumatoid arthritis often affects the TM joint.[57]

Capsular Fibrosis

Unresolved or chronic inflammation of the TM joint capsule stimulates overproduction of fibrous connective tissue, which creates **capsular fibrosis** of the TM joint complex.[6] The resultant fibrosis causes progressive damage and loss of tissue function.[6] A thorough client history is the key to identifying this condition. The clinician should listen for reports of repeated episodes of capsulitis. Circumstances leading to chronic capsulitis with progression to capsular fibrosis may include prolonged periods of immobilization, direct or indirect trauma, or arthritis.[6] Physical examination will reveal limited or altered osteokinematic motions, suggestive of a decrease in translatory motion on the involved side. Active motion of the TM joint capsule will typically elicit pain.[6]



Patient Application 6–3

Jill Smith's musculoskeletal complaints and limited active ROM of the mandible may be attributed to capsular fibrosis, as well as to her faulty posture. Most likely she has had repeated episodes of acute capsulitis of the right TM joint, with her initial episode following her childhood bicycle accident. The trauma to her face from hitting her chin on the handlebars of her bicycle likely caused compression forces to both TM areas, resulting in an inflammatory process with edema and pain. Her faulty posture may have developed as a result of her long-standing allergies and is exacerbated by her job that requires extended time in front of a computer screen in what by her report sounds like an ergonomically poor workspace. The stress of being a single working mother may have exacerbated the inflammation without the opportunity for resolution, leading to a chronic and progressive capsular fibrosis.[31]

Osseous Mobility Conditions

Osseous mobility disorders of the TM joint complex include **joint hypermobility** and **dislocation.** Many similarities are noted in the client history and clinical findings for these two conditions. Hypermobility, or excessive motion, of the TM joint is a common

phenomenon found in both symptomatic and nonsymptomatic populations. The hypermobility is a result of laxity of the joint capsules, tendons, and ligaments.[6,58] Joint hypermobility may be a generalized connective tissue disorder that involves all joints of the body, including the TM joints.[36,59] Individuals seen clinically for TM joint hypermobility typically report that the jaw "goes out of place," produces noises, or "catches" when the mouth is in the fully opened position. Physical examination of clients with TM joint hypermobility reveals an increased indentation posterior to the lateral pole. Joint noises occur at the end of mandibular depression and at the beginning of mandibular elevation. These noises may be heard by the person; however, the clinician often can only palpate the clicking or crepitus associated with the noises. Hypermobility of one TM joint results in the deflection of the mandible toward the contralateral side with mandibular depression. In addition, mandibular depression will exceed 40 mm.[6] Yang and colleagues examined MRI films of 98 patients diagnosed with TM joint hypermobility during jaw opening.[36] They found pathological changes (hypertrophy, atrophy, or contracture) in 77% of the lateral pterygoid muscles, with changes more common in the superior portion of the muscle. The investigators often found anterior disc displacement with reduction in the patients who reported more symptoms than were reported by persons with normal mobility or with disc displacement without reduction.[36]

Many aspects of the client history and physical examination of individuals with dislocation of the TM joint are similar to those of individuals with hypermobility. However, with TM joint dislocation, full mandibular depression results in deflection (lateral deviation) of the jaw to the contralateral side of the involved TM joint, and inability to close the mouth. The individual may experience pain with this condition.

With dislocation, both the mandibular condyle and the disc have translated anteriorly beyond the articular crest of the tubercle of the temporal bone, thus “sticking” in the extreme end-range position.[6,11] Dislocation of the TM joint is usually temporary and resolves with joint mobilization.

Articular Disc Displacement

Articular disc displacement occurs when the articular disc subluxes beyond the articular eminence.[6,60] Two conditions can result: disc displacement with reduction and disc displacement without reduction.[6,36,60] Without intervention, disc displacement with reduction often advances to disc displacement without reduction.[6,11,36,60] Disc displacement is an internal derangement that may be identified by diagnostic imaging or during a physical examination.[11] MRI is the imaging modality of choice to identify disc displacement.[17]

Individuals exhibiting disc displacement with reduction experience “joint noise” at two intervals: during mandibular depression and during mandibular elevation. The joint noise is referred to as a **reciprocal click** and is the gold standard necessary to diagnose disc displacement with reduction.[6,11,61] At rest the articular disc is anterior to the mandibular condyle; therefore, the mandibular condyle is in contact with the retrodiscal tissue rather than with the disc. During mandibular depression, the condyle translates anteriorly and captures the inferior surface of the disc to obtain a normal relationship with the disc. When the condyle slips under the disc, an audible click is often present. Once the condyle-disc relationship is restored, motion continues normally through full mandibular depression. As the mandible elevates to close the mouth, the condyle translates posteriorly and slips out from under the disc, producing

a second audible click. The second click signifies that the condyle and disc have lost the normal condyle-disc relationship. When the click occurs early in opening and late in closing, the amount of anterior displacement of the disc is relatively limited. The later the click occurs in the opening phase, the more severe the disc dislocation.[6,61]

Individuals exhibiting disc displacement with reduction may remain in this state or progress rapidly, within months, to an acute condition of disc displacement without reduction. The posterior attachments to the disc become overstretched and unable to relocate the disc during mandibular depression, which results in the loss of the reciprocal clicks.[6,11,61] On MRI, Yang and colleagues discovered a strong correlation between abnormalities of the lateral pterygoid muscles and TM joints with disc displacements both with and without reduction of the disc.[36] The abnormalities of the lateral pterygoid muscle included hypertrophy, contracture, and atrophy of the superior and inferior bellies of the lateral pterygoid muscles of the involved TM joint. Recall that the lateral pterygoid muscle attaches to the anterior portion of the disc (see Fig. 6–11) and is normally active with mandibular elevation, presumably to eccentrically control the return to the resting position. Hypertrophy of this muscle indicates overactivity, which possibly leads to the excessive anterior translation of the articular disc.[36]

Whether acute or chronic, disc displacement without reduction indicates that the disc does not relocate onto the mandibular condyle. Thus, clients with acute disc displacement without reduction demonstrate limited mandibular motion as a result of the disc creating a mechanical obstruction to condylar motion rather than facilitating condylar translation. Individuals with disc displacement without reduction typically describe an inability to fully depress the mandible, as well as difficulty performing

functional movements involving the jaw, such as chewing, talking, or yawning.

Degenerative Conditions

Morales and Cornelius conducted imaging studies of the TM joint using MRI.[12] The authors noted that degenerative changes were one of the most common pathologies impacting the TM joint, second only to internal derangement, which was considered the most common.[12]

Two degenerative conditions may affect the TM joint: osteoarthritis and rheumatoid arthritis. Rheumatoid arthritis was discussed previously under inflammatory conditions. It is estimated that 80% to 90% of the population older than 60 years have some symptoms of osteoarthritis in the TM joint.[36] Osteoarthritis usually occurs unilaterally (unlike rheumatoid arthritis, which usually presents bilaterally). The primary cause of osteoarthritis is repeated minor trauma to the joint, particularly trauma that creates an impact between the articular surfaces.[11,62] The radiographic features of degenerative changes in the TM joint, similar to those elsewhere in the body, include joint space narrowing, erosion, osteophyte formation, sclerosis, and remodeling.[11] Loss of posterior teeth may also contribute to degenerative changes because simple occlusion of the remaining teeth alters the forces that occur between the TM joint forces.[6]



Foundational Concepts 6–1

Signs and Symptoms of TM Joint Dysfunction

Clinical manifestations of TM joint dysfunction can vary widely, depending on the extent of the condition and the presence of complicating factors. Signs and symptoms may include:

- Pain in the area of the jaw
- Edema around the mandibular condyle
- Increased or decreased active or passive ROM
- Popping or clicking noises
- Difficulty with functional activities (e.g., eating, talking) or parafunctional activities (e.g., clenching, nail biting, pencil chewing) of the mandible
- Catching or locking of the jaw
- Forward head posture



Patient Application 6–4

Jill Smith's symptoms may be attributed to a number of potential sources in isolation—trauma to the TM joint, poor cervical posture, forward head position—or some combination of these sources. Factors such as stress from work and family life also may play a role in her clinical presentation. Although the research literature does not explicitly draw a direct link between these variables, it suggests that these biomechanical variables may play a role in a patient's pain presentation. Therefore, clinical interventions aimed at improving the structural balance of the head, neck, and thorax are important augmentations to any direct intervention at the TM joint (Fig. 6–19). Task modification in her work environment and stress management strategies may be therapeutic adjuncts.

FIGURE 6-19

Corrected cervical posture restores the muscles of the cervical spine and TM joint to a more balanced length-tension relationship.



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Summary

- The TM joints are unique both structurally and functionally. The magnitude and frequency of mandibular movement, the daily resistance encountered during mastication, the physical stress imposed by sustained sitting and standing postures, and the chronic adaptation of muscles around the TM joint complex make this joint particularly vulnerable to problems.
- The influence of the cervical spine and posture upon the TM joint must always be recognized. The typical changes associated with aging must be considered also.
- Intervention for clients with TM disorders presents many clinical challenges, and

practitioners with interest in this clientele should seek advanced education beyond entry-level in this specialty area.

- As we proceed in subsequent chapters to examine the joint complexes of the appendicular skeleton, evidence will indicate that each complex has its own unique features. We will not see again, however, the complexity of intra-articular and discal motions that can be observed at the TM joints.

Study Questions

1. Describe the articulating surfaces of the TM joint.
2. Although the TM joint is classified as a synovial joint, its articular surfaces are covered with fibrocartilage rather than hyaline cartilage. Discuss the significance of this difference.
3. What is the significance of the differing thicknesses and vascularity of the disc?
4. How do the superior and inferior laminae of the retrodiscal area differ?
5. Describe the motions in the upper and lower joints within the TM joint during mouth opening and closing.
6. What limits posterior motion of the condyle? How is the motion limited?
7. What are the consequences of a left TM joint that does not translate?
8. What are the consequences of a right disc that does not rotate freely over the

condyle?

9. Describe the control of the disc in moving from an open mouth to a closed mouth position.

10. What is the potential impact of the posture of the cervical spine on the function of the TM joint complex?

11. Discuss the effects of aging on the TM joint.

12. Compare and contrast the functional presentations of hypo- and hypermobile TM joint complexes.

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Chapter 7: The Shoulder Complex

Paula M. Ludewig; John Borstad

Anatomy Overview

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MUSCLE ACTIONS OF THE GLENOHUMERAL JOINT		
TABLE KEY: Primary movers Secondary movers		
Sagittal Plane	Flexion	Extension
	Deltoid (anterior) Pectoralis major (clavicular head) Biceps brachii Coracobrachialis	Deltoid (posterior) Latissimus dorsi Triceps brachii (long head) Pectoralis major (sternal head) Teres major
Frontal Plane	Abduction	Adduction
	Supraspinatus Deltoid (middle)	Pectoralis major Latissimus dorsi Teres major Coracobrachialis

Transverse Plane	Medial rotation	Lateral rotation
	Pectoralis major Subscapularis Teres major Latissimus dorsi Deltoid (anterior)	Infraspinatus Teres minor Deltoid (posterior)

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MUSCLE ACTIONS OF THE SCAPULOTHORACIC JOINT	
TABLE KEY: Primary movers Secondary movers	
Protraction	Retraction
Serratus anterior Pectoralis major Pectoralis minor	Middle trapezius Rhomboids
Elevation	Depression
Upper trapezius Levator scapulae Rhomboids	Lower trapezius Latissimus dorsi Pectoralis minor
Upward rotation	Downward rotation

Serratus anterior Upper trapezius Lower trapezius	Rhomboids Latissimus dorsi Levator scapulae Pectoralis minor

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SHOULDER MUSCLE ATTACHMENTS		
Muscles	**Proximal Attachment**	**Distal Attachment**
Pectoralis major	Clavicular head: anterior surface of medial half of clavicle Sternocostal head: anterior surface of sternum, superior six costal cartilages, aponeurosis of external oblique muscle	Lateral lip of intertubercular (bicipital) groove and crest of greater tubercle of humerus
Pectoralis minor	3rd–5th ribs near their costal cartilages	Medial border and superior surface of coracoid process of scapula
Serratus anterior	External surfaces of lateral parts of 1st–8th ribs	Anterior surface of medial border of

		scapula
Trapezius	Medial third of superior nuchal line, external occipital protuberance, nuchal ligament, spinous processes of C7–T12 vertebrae	Lateral third of clavicle, acromion, and spine of scapula.
Latissimus dorsi	Spinous processes of inferior 6 thoracic vertebrae, thoracolumbar fascia, iliac crest, and inferior 3 or 4 ribs	Floor of intertubercular sulcus of humerus
Levator scapulae	Transverse processes of C1–C4 vertebrae	Superior aspect of medial border of the scapula
Rhomboid minor	Nuchal ligament; spinous processes of C7 and T1 vertebrae	Smooth triangular area at medial end of scapular spine
Rhomboid major	Spinous processes of T2–T5 vertebrae	Medial border of scapula from level of spine to inferior angle
Deltoid	Lateral 1/3 of clavicle; acromion and spine of scapula	Deltoid tuberosity of humerus
Supraspinatus	Supraspinous fossa of scapula	Superior facet of greater tubercle of humerus
Infraspinatus	Infraspinous fossa of scapula	Middle facet of greater tubercle of humerus
Teres minor	Middle aspect of lateral border of scapula	Inferior facet of greater tubercle of humerus
Teres major	Posterior surface of inferior angle of scapula	Medial lip of intertubercular

		(bicipital) groove of humerus
Subscapularis	Subscapular fossa (most of anterior surface of scapula)	Lesser tubercle of humerus
Biceps brachii	Short head: tip of coracoid process of scapula Long head: supraglenoid tubercle of scapula	Tuberosity of radius
Coracobrachialis	Tip of coracoid process of scapula	Middle third of medial surface of humerus
Triceps brachii	Long head: infraglenoid tubercle of scapula	Proximal end of olecranon of ulna

Introduction

The shoulder complex, composed of the clavicle, scapula, and humerus, is an intricately designed combination of three joints that links the upper extremity to the thorax. The articular structures of the shoulder complex are designed primarily for mobility, allowing us to move and position the hand through a wide range of space. The **glenohumeral (GH) joint,** which links the humerus and scapula, has greater mobility than any other joint in the body. Although the components of the shoulder complex constitute half the mass of the entire upper limb, they are connected to the axial skeleton by a single joint, the **sternoclavicular (SC) joint.**[1] As a result, muscle forces serve as a primary mechanism for securing the shoulder girdle to the thorax and providing a stable base of support for upper extremity movements.

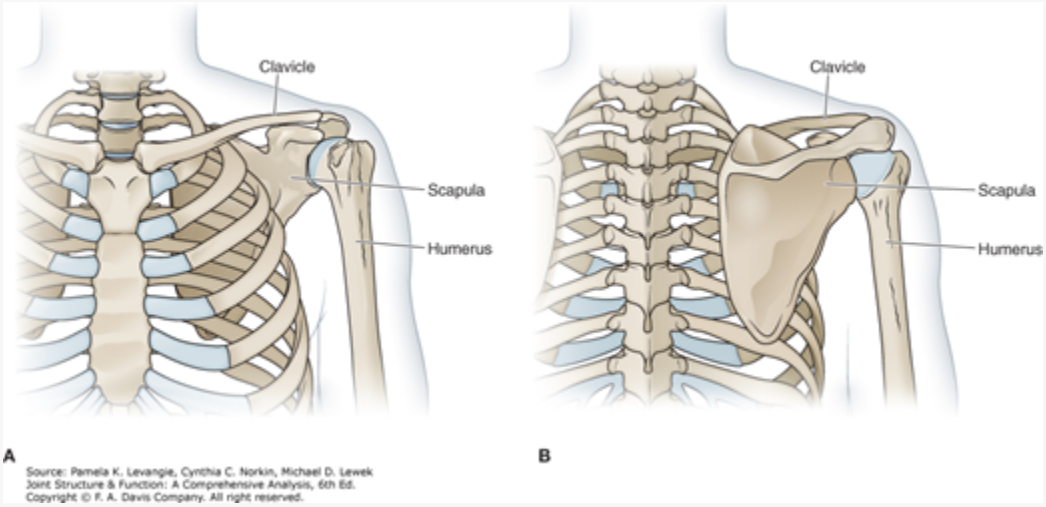
The contradictory requirements on the shoulder complex for both mobility and stability are met through active forces, or **dynamic stabilization,** a concept for which the shoulder complex is considered a classic example. In essence, dynamic stability exists when a moving segment or set of segments is not limited very much by passive forces such as articular surface configuration, capsule, or ligaments, but instead relies heavily on active forces or dynamic muscular control. Dynamic stabilization results in a wide range of mobility for the shoulder complex and provides adequate stability when the complex is functioning normally. However, the competing mobility and stability demands on the shoulder girdle and the intricate structural and functional design mediating the compromise between structure and function make the shoulder complex highly susceptible to dysfunction and instability.

The osseous segments of the shoulder complex are the clavicle, scapula, and humerus (Fig. 7–1). These three segments are joined by three interdependent linkages: the sternoclavicular joint, the **acromioclavicular (AC) joint,** and the glenohumeral joint. The articulation between the scapula and the thorax is often described as the **scapulothoracic (ST) "joint,"** although it does not have the characteristics of a fibrous, cartilaginous, or synovial joint. Instead, scapula motion on the thorax is a function of sternoclavicular, acromioclavicular, or combined sternoclavicular and acromioclavicular joint motions. The scapulothoracic joint is frequently described in the literature as a "functional joint." An additional functional joint that is often considered part of the shoulder complex is the **subacromial** (or **suprahumeral) "joint."** This functional joint is formed by movement of the head of the humerus below the coracoacromial arch. Although the movement at this functional joint plays an important role in shoulder function and dysfunction, we will refer to it as the

subacromial space and consider it a component of the glenohumeral joint rather than a separate joint.

FIGURE 7-1

A. An anterior view of the three components of the shoulder complex: the humerus, the scapula, and the clavicle. **B.** A posterior view of the three components.



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The joints that compose the shoulder complex can, in combination with trunk motion, contribute as much as 180° of elevation to the upper extremity, although typical range of motion (ROM) for most individuals is less. **Elevation** of the upper extremity refers to the combination of scapular, clavicular, and humeral motion that occurs when the arm is raised either *forward* or *to the side,* including sagittal plane flexion, frontal plane abduction, and all the motions in between. Motion of the scapula on the thorax normally contributes about one-third of the total shoulder complex motion necessary for arm elevation, whereas the glenohumeral joint contributes about two-thirds of the total motion. Although the integrated function of all three joints is of primary interest,

each of the articulations and components of the shoulder complex must be examined individually before their integrated dynamic function can be appreciated.

Shoulder Complex Joints and Structures

Sternoclavicular Joint Structure

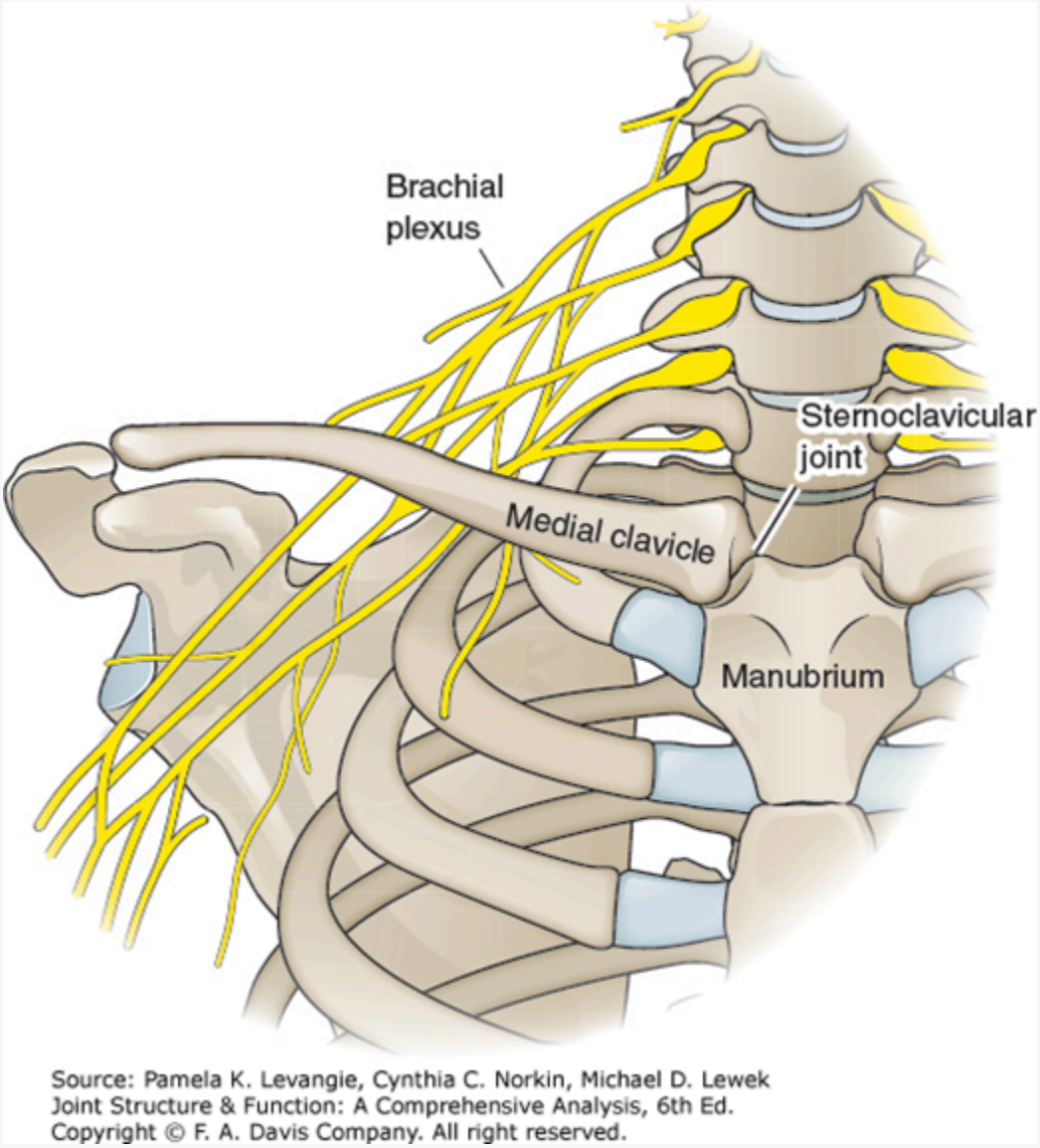
The sternoclavicular joint is the only structural attachment between the axial skeleton and the shoulder and upper extremity. The sternoclavicular joint is classified as a synovial joint with three rotary and three translatory degrees of freedom.

Sternoclavicular Articulation

The sternoclavicular articulation (Fig. 7–2) consists of two shallow, saddle-shaped surfaces but is often considered a plane (rather than saddle) synovial joint because of the subtlety of the saddle configuration and minimal joint congruency. The small articular surface of the clavicle is located anteroinferiorly, whereas the sternal articular surface of the clavicle covers nearly the width of the manubrial facet on the sternum.[2] The cartilage of the first rib is also part of the SC articulation. The superior and posterior portions of the medial clavicle do not contact the sternum but serve as attachments for the accessory joint structures: the **sternoclavicular joint disc, the joint capsule,** and the **interclavicular ligament.** At rest, the sternoclavicular joint space is wedge-shaped and open superiorly. Movements of the clavicle in relation to the manubrium result in contact area changes between the clavicle, the sternoclavicular joint disc, and the costal cartilage.

FIGURE 7-2

The sternoclavicular joint is the articulation of the reciprocally convex and concave (saddle-shaped) surfaces of the medial clavicle with the manubrium of the sternum and the first costal cartilage; it is considered either a saddle or a plane synovial joint.



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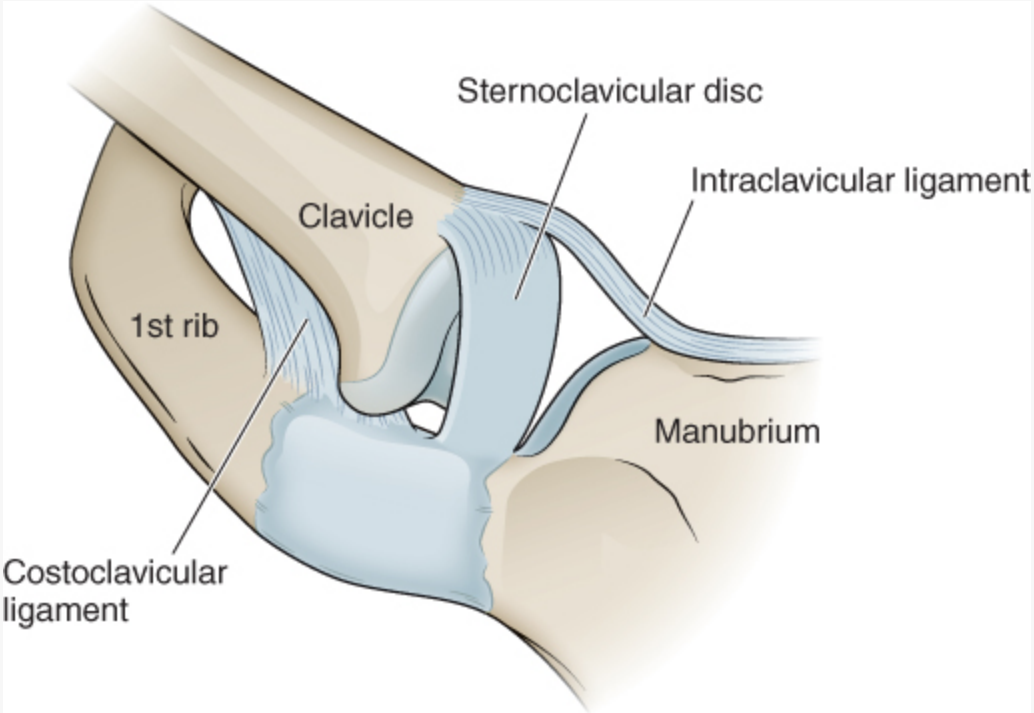
Sternoclavicular Accessory Joint Structures

The sternoclavicular joint has a fibrocartilage disc that increases congruence between

the articulating surfaces and dissipates joint loads. The upper portion of the disc is attached to the posterosuperior clavicle and the lower portion is attached to the manubrium and first costal cartilage, as well as to the anterior and posterior portions of the fibrous sternoclavicular capsule.[3] As shown in **Figure 7–3**, the disc transects the sternoclavicular joint space diagonally, dividing the joint into two separate cavities.[1] The disc improves SC stability by increasing joint congruence and by absorbing forces transmitted along the clavicle from its lateral end to the sternoclavicular joint. In Figure 7–3, note that the diagonal attachments of the sternoclavicular disc will limit medial translation of the clavicle, which might otherwise cause the medial articular surface of the clavicle to override the shallow manubrial facet. The disc also has a substantial area of contact with the medial clavicle which helps dissipate medially directed forces, protecting the small manubrial facet from high concentrations of pressure. Although one might think that medially directed forces on the clavicle are infrequent and minimal, we shall see that this is not the case when we examine the function of the acromioclavicular joint, the **upper trapezius muscle,** and the **coracoclavicular ligament.**

FIGURE 7-3

The sternoclavicular disc transects the joint into two separate joint cavities (shown with the clavicle pulled away slightly to facilitate visualization).



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During arm and shoulder motion, the disc acts like a pivot point for the medial end of the clavicle, but its role is different depending on the motion. With **clavicular elevation** and **depression,** the medial articular surface of the clavicle rolls and slides on the relatively stationary disc, with the upper attachment of the disc serving as a pivot point. With **clavicular protraction** and **retraction,** the sternoclavicular disc and medial articular surface of the clavicle roll and slide together on the manubrial facet, with the lower attachment of the disc serving as a pivot point.[1] The disc, therefore, is considered part of the manubrium during elevation and depression and part of the clavicle during protraction and retraction.

Expanded Concepts 7–1

Three-Compartment Sternoclavicular Joint

Anatomic examination of the sternoclavicular articulation has led to the proposal that there are three, rather than two, functional units of the sternoclavicular joint: a lateral compartment between the disc and clavicle for elevation and depression, a medial compartment between the disc and manubrium for protraction and retraction, and a costoclavicular joint for anterior and posterior long axis rotation. Anterior and posterior rotation are thought to occur between a portion of the disc over the first rib and a "conus" on the anteroinferior edge of the articular surface of the medial clavicle.[4]

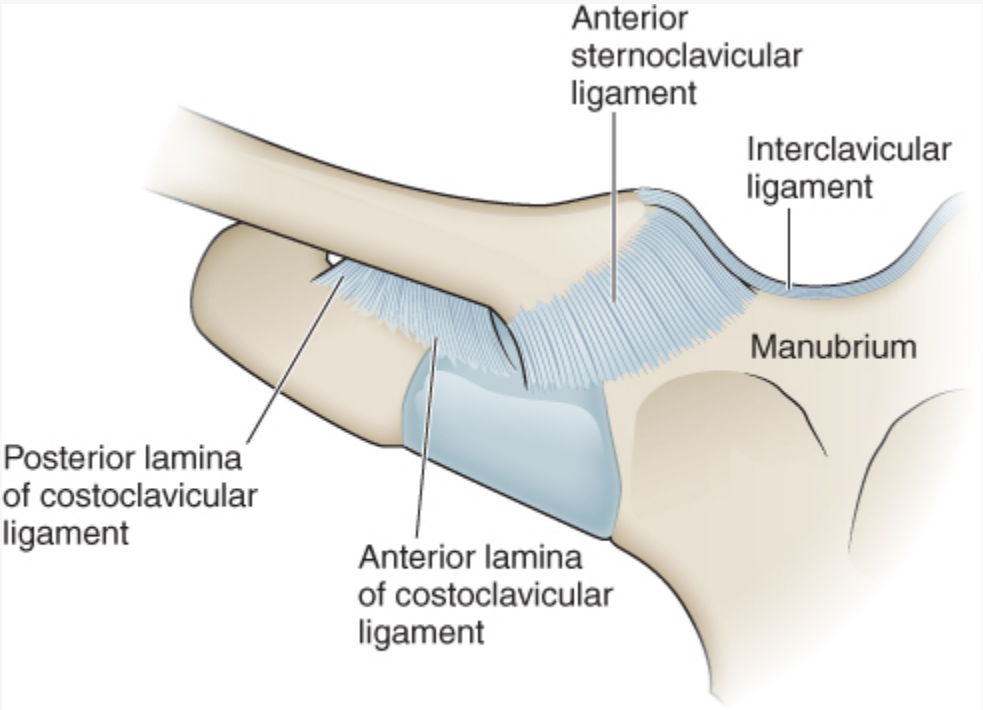
Sternoclavicular Capsule and Ligaments

The sternoclavicular joint is surrounded by a fairly strong fibrous capsule but depends on three ligament complexes for the majority of its support: the **anterior** and **posterior sternoclavicular ligaments,** the bilaminar **costoclavicular ligament,** and the interclavicular ligament (**Fig. 7–4**). The anterior and posterior sternoclavicular ligaments reinforce the capsule and function primarily to check anterior and posterior translation of the medial clavicle, with the thick posterior capsule providing the primary restraint to both translations.[5] The costoclavicular ligament is a very strong ligament between the clavicle and first rib that has two segments, or *laminae.* The anterior portion is directed laterally from the first rib to the clavicle, whereas the posterior portion is directed medially from the rib to the clavicle.[3,6] Both laminae limit lateral clavicle elevation and may contribute to roll and glide kinematics of the medial clavicle on the disc.[7] The costoclavicular ligament also absorbs and transmits the superiorly directed forces applied to the clavicle by the sternocleidomastoid and sternohyoid muscles, whereas the posterior lamina will resist medial translation of the clavicle. The interclavicular ligament limits excessive depression of the lateral clavicle and superior gliding of the medial clavicle on the manubrium.[8,9] The limitation of clavicular

depression by the interclavicular ligament is critical to protecting structures such as the brachial plexus and subclavian artery which pass under the clavicle and over the first rib (see Fig. 7–2).

FIGURE 7-4

The sternoclavicular joint ligaments. The axes for sternoclavicular joint motion appear to occur at the location of the costoclavicular ligament.



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Acromioclavicular Joint Structure

The acromioclavicular joint attaches the scapula to the lateral clavicle. It is generally described as a plane synovial joint with three rotational and three translational degrees of freedom. The primary function of the acromioclavicular joint is to allow the scapula

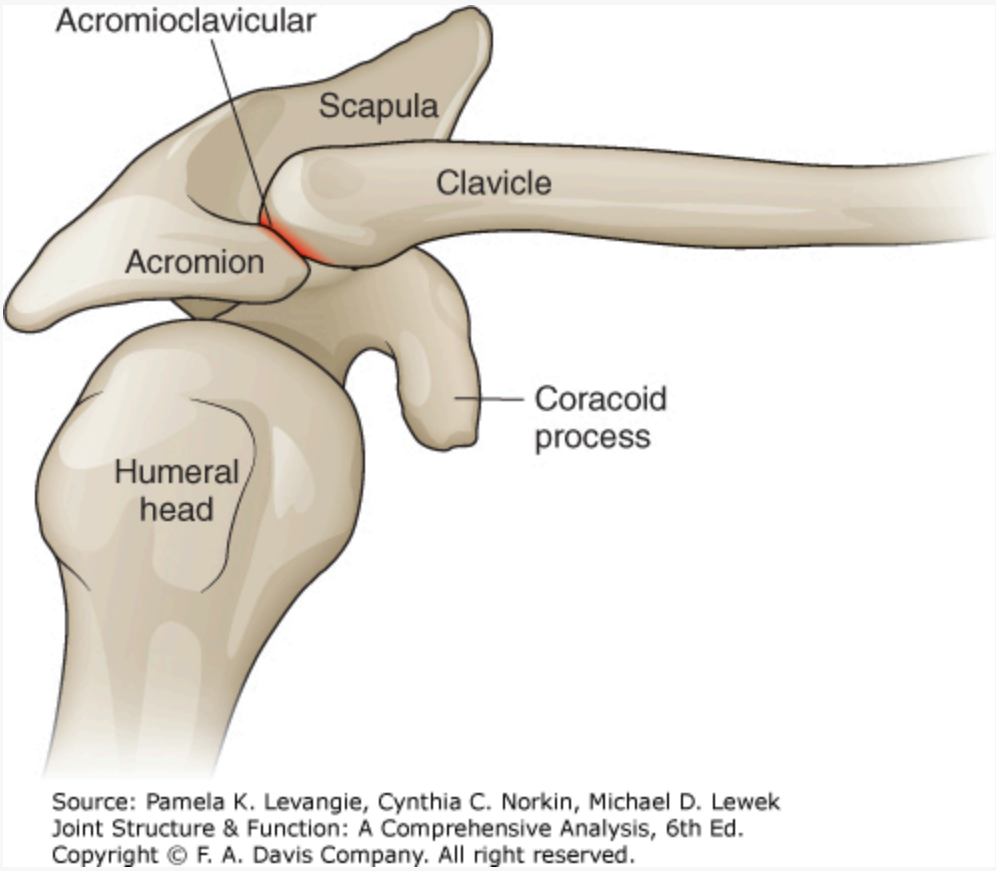
to rotate in three dimensions during arm movement. This function satisfies three objectives: increasing upper extremity motion; positioning the glenoid beneath the humeral head; and helping maximize scapula contact with the thorax. The acromioclavicular joint also helps transmit forces from the upper extremity to the clavicle.

Acromioclavicular Articulation

The acromioclavicular joint is the articulation between the lateral end of the clavicle and a small facet on the acromion of the scapula (Fig. 7–5). The articular facets are considered to be incongruent and vary in configuration from flat to concave-convex.[6] The vertical orientation of the articulating surfaces may make the joint susceptible to the degenerative effects of shear forces.[10]

FIGURE 7-5

The acromioclavicular joint is the articulation of the lateral clavicle with the acromion on the scapula, forming an incongruent plane synovial joint.



Acromioclavicular Accessory Joint Structures

Initially, the AC joint is a fibrocartilaginous union between the clavicle and acromion. With upper extremity use over time a joint space develops that may leave a “meniscal homologue” within the joint.[11] This fibrocartilage remnant, or disc, may vary in size between individuals, within an individual as they age, and between the shoulders of the same individual.

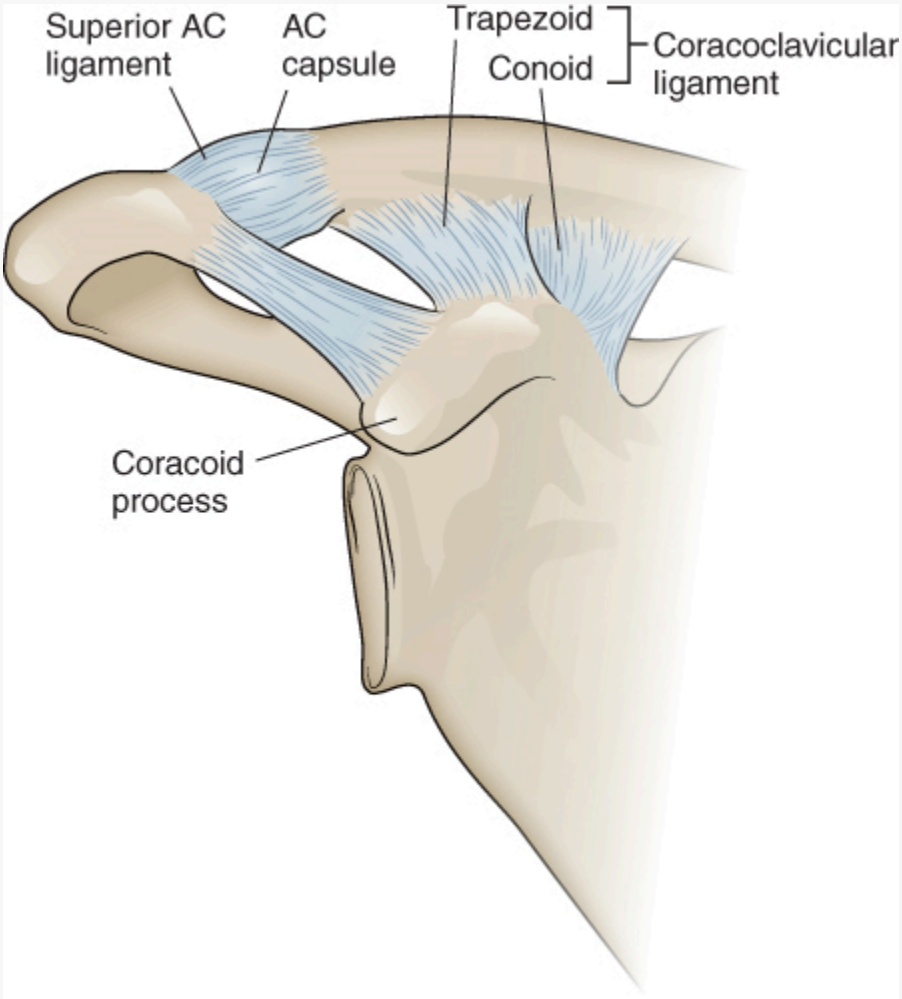
Acromioclavicular Capsule and Ligaments

The capsule of the acromioclavicular joint is weak and maintains integrity of the joint through the reinforcement of the **superior** and **inferior acromioclavicular ligaments** and the coracoclavicular ligaments (Fig. 7–6). The acromioclavicular ligaments assist the

capsule in apposing the articular surfaces; the superior acromioclavicular ligament is the main restraint to movement caused by anterior forces applied to the lateral clavicle. 12,13 The fibers of the superior acromioclavicular ligament are reinforced by aponeurotic fibers of the **trapezius** and **deltoid muscles,** making the superior ligament stronger than the inferior capsule and ligament.

FIGURE 7-6

The acromioclavicular joint capsule and ligaments, including the coracoclavicular ligament with its conoid and trapezoid portions.



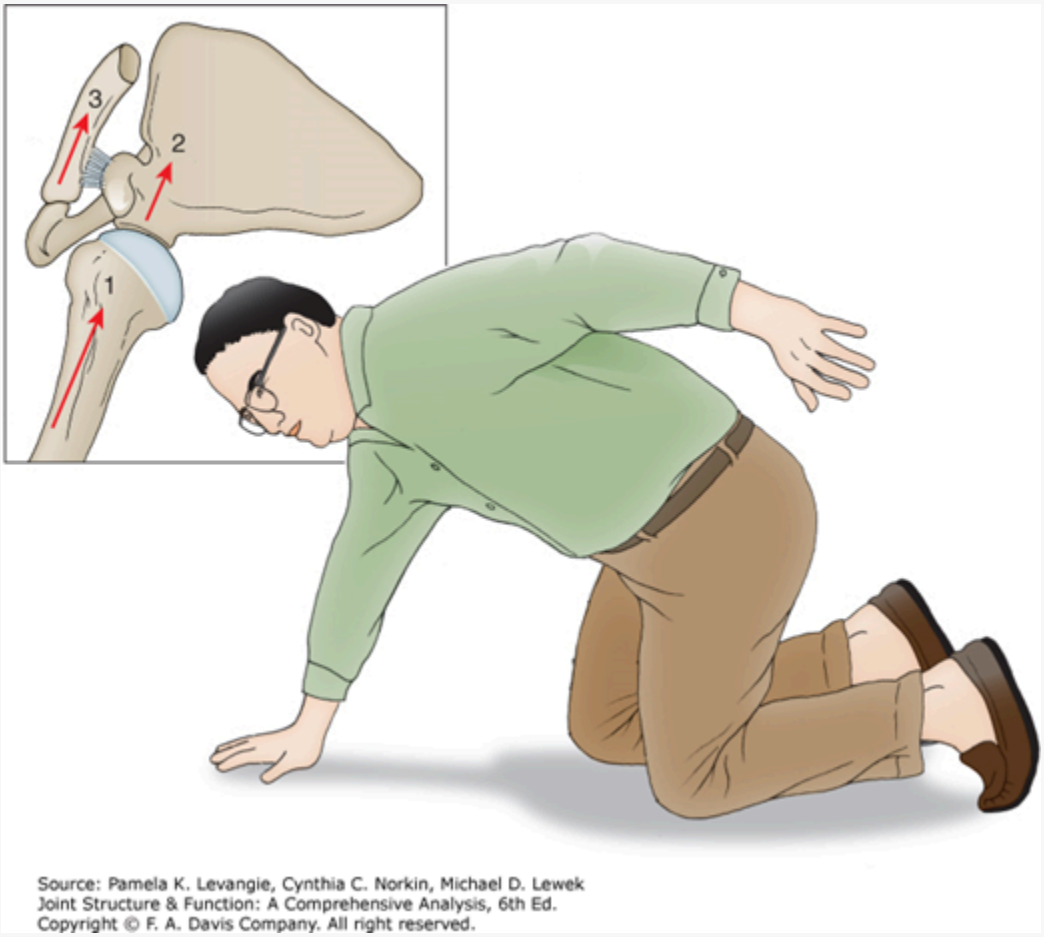
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The coracoclavicular ligament, although not directly reinforcing the acromioclavicular joint, firmly unites the clavicle and scapula to provide much of the joint's superior, inferior, and rotational stability.[12] This ligament is divided into a medial **conoid ligament** and a lateral **trapezoid ligament.** The conoid ligament is more triangular and vertically oriented, whereas the trapezoid ligament is quadrilateral in shape and oriented nearly horizontal.[6] The two portions are separated by adipose tissue and a large bursa.[9] Both ligaments attach to the undersurface of the clavicle and their influence on shoulder biomechanics will be described in the section on Integrated Function of the Shoulder Complex. Although the acromioclavicular capsule and ligaments can resist small rotary and translatory forces at the acromioclavicular joint, restraint of larger translations and rotations is credited to the coracoclavicular ligament.

The conoid portion of the coracoclavicular ligament provides the primary restraint to inferior translation of the acromion relative to the lateral clavicle.[13] The trapezoid portion of the ligament provides more restraint to posterior translations of the lateral clavicle relative to the acromion.[14,15] Both portions of the ligament limit upward rotation of the scapula at the acromioclavicular joint. When medially directed forces on the humerus are transferred to the glenoid fossa of the scapula, medial displacement of the scapula on the clavicle is prevented by the coracoclavicular ligament complex, in particular the horizontal trapezoid portion. Such medial forces are transferred by the ligament to the clavicle and then to the strong sternoclavicular joint (**Fig. 7–7**). In addition, one of the most critical functions of the coracoclavicular ligament relative to shoulder function is to couple posterior clavicle rotation and scapula upward rotation during arm elevation.

FIGURE 7-7

When a person bears weight (or falls) on an arm, a medially directed force up the humerus (1) is transferred to the glenoid fossa of the scapula (2) then to the clavicle via the coracoclavicular ligament (3) and finally to the stable sternoclavicular joint.



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Scapulothoracic Joint Structure

Scapulothoracic Articulation

The scapulothoracic “joint” is formed by the articulation of the anterior surface of the scapula with the thorax. It is not a true anatomic joint because it is not a union of bony segments by fibrous, cartilaginous, or synovial tissues. By contrast, the articulation of

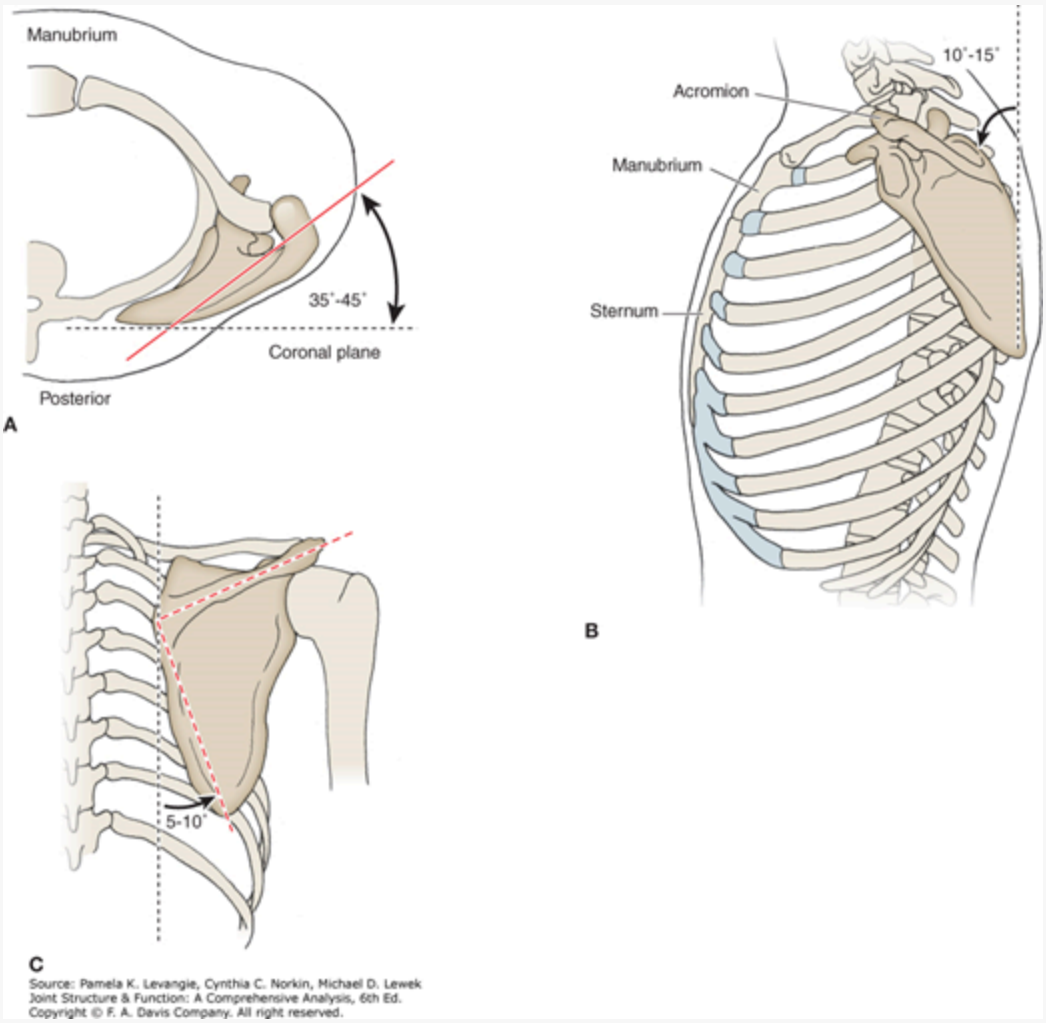
the scapula with the thorax depends on the integrity of the anatomic acromioclavicular and sternoclavicular joints. The sternoclavicular and acromioclavicular joints are interdependent with scapulothoracic motion because the scapula is attached by its acromion process to the lateral end of the clavicle through the acromioclavicular joint; the clavicle, in turn, is attached to the axial skeleton at the sternoclavicular joint. Any movement of the scapula on the thorax must result in movement at the acromioclavicular joint, the sternoclavicular joint, or both. This makes the functional scapulothoracic joint part of a true closed chain with the acromioclavicular and sternoclavicular joints and the thorax. Observation and measurement of individual sternoclavicular and acromioclavicular joint motions are more difficult than observing or measuring motions of the scapula on the thorax. Consequently, scapulothoracic position and motions are described and measured far more frequently than are the sternoclavicular and acromioclavicular joint motions upon which scapulothoracic motions are dependent.

The scapula rests on the posterior thorax approximately 5 cm from the midline between the second through seventh ribs. The resting position of the scapula on the thorax is shown in Figure 7–8. The scapula is internally rotated 35° to 45° from the coronal plane (Fig. 7–8A), is tilted anteriorly approximately 10° to 15° from vertical (Fig. 7–8B), and is upwardly rotated 5° to 10° from vertical.[16,17] This magnitude of upward rotation is described about a “longitudinal” reference axis perpendicular to the axis running from the root of the scapular spine to the acromioclavicular joint (Fig. 7–8C). If the vertebral (medial) border of the scapula is used as the reference “longitudinal” axis, the magnitude of upward rotation at rest is usually described as 2° to 3° from vertical.[18] Although these “normal” values for the resting scapula are cited, substantial individual

variability exists in scapular rest position, even among healthy subjects.[19]

FIGURE 7-8

The resting position of the scapula on the thorax. **A.** This superior view shows the scapula resting in an internally rotated position 35° to 45° anterior to the coronal plane. **B.** This side view shows that the scapula at rest is anterior tilted approximately 10° to 15° to the vertical plane. **C.** This posterior view shows that the “longitudinal” axis of the scapula (90° to an axis through the spine) at rest is upwardly rotated 5° to 10° from vertical.



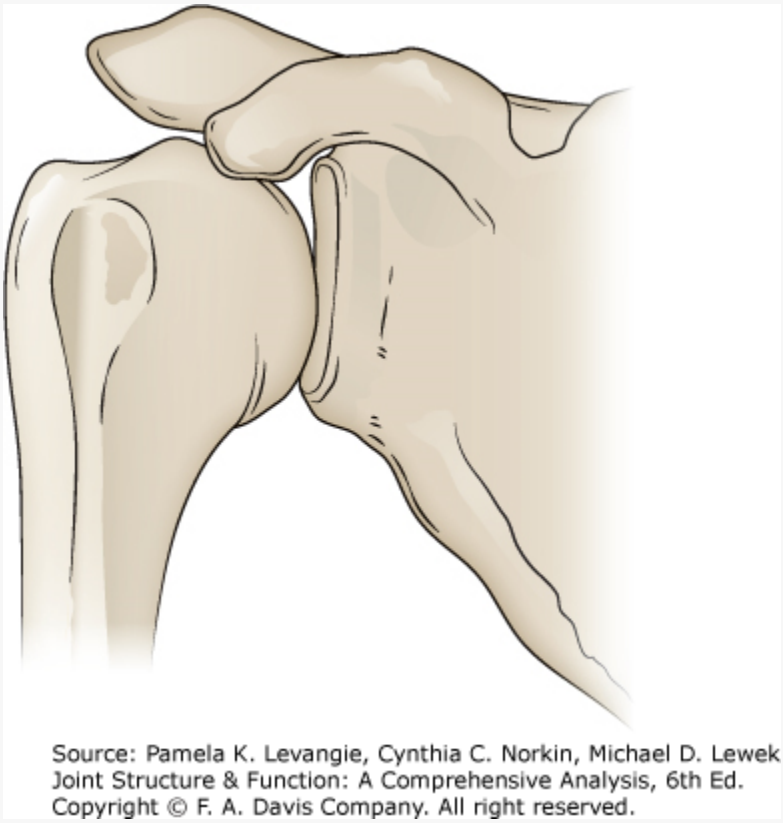
Glenohumeral Joint Structure

The glenohumeral joint is a ball-and-socket synovial joint with three rotary and three translatory degrees of freedom. It has a capsule as well as several associated ligaments and bursae. The articulation is composed of the large head of the humerus and the smaller glenoid fossa (Fig. 7–9). Because the glenoid fossa of the scapula is the proximal segment of the glenohumeral joint, any motions of the scapula will influence glenohumeral joint function. The glenohumeral joint has sacrificed articular congruency to increase the mobility of the upper extremity, allowing flexible

positioning of the hand. As such, the reduced stability results in increased susceptibility to degenerative changes, instability, and derangement.

FIGURE 7-9

The glenohumeral joint.



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Proximal Articular Surface

The glenoid fossa of the scapula serves as the proximal articular surface for the glenohumeral joint. The orientation of the shallow concavity of the glenoid fossa in relation to the thorax varies with the resting position of the scapula on the thorax and with motion at the sternoclavicular and acromioclavicular joints. In addition, the orientation of the glenoid fossa may vary with the form of the scapula itself. The glenoid fossa may be tilted slightly upward or downward when the arm is at the side, although

representations most commonly show a slight upward tilt.[19–21] Similarly, the fossa does not always lie in a plane perpendicular to the plane of the scapula; it may be anteverted or retroverted up to 10°, with 6° to 7° of retroversion most typical.[20] With anteversion, the glenoid fossa faces slightly anterior in relation to the plane or body of the scapula and, with retroversion, slightly posterior.

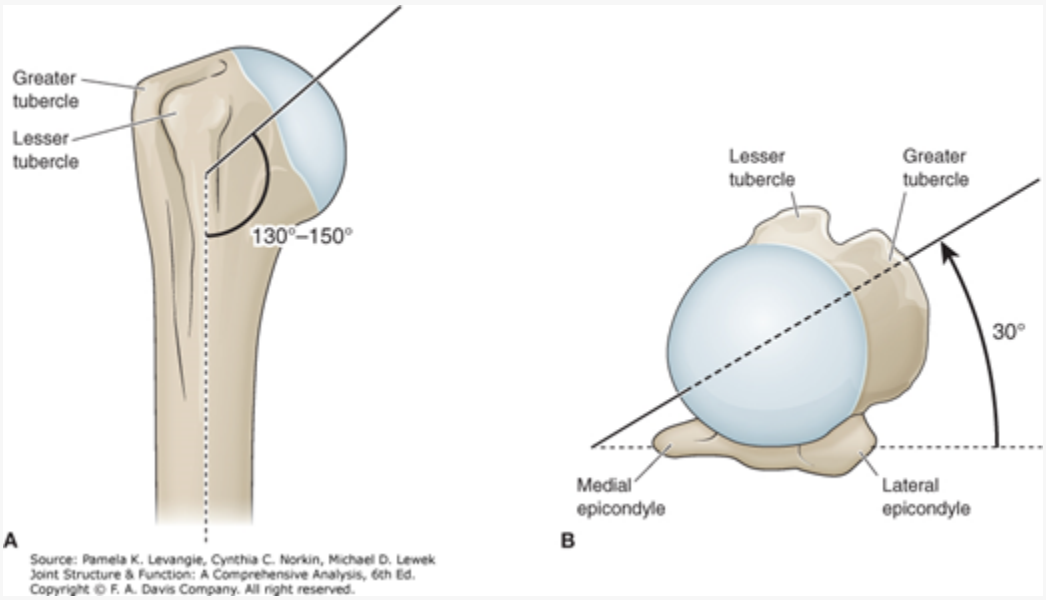
Distal Articular Surface

The humeral head is the distal articular surface of the glenohumeral joint and has an articular surface area that is larger than that of the glenoid articular surface, forming one-third to one-half of a sphere. In anatomical position, the head faces medially, superiorly, and posteriorly with regard to the shaft of the humerus and the humeral condyles. The **angle of inclination** is formed by an axis through the humeral head and neck in relation to a longitudinal axis through the shaft of the humerus and is normally between 130° to 150° in the frontal plane (Fig. 7–10A). The **angle of torsion** is formed by an axis through the humeral head and neck in relation to an axis through the humeral condyles. This transverse plane angle varies but is approximately 30° posterior (Fig. 7–10B). The posterior orientation of the humeral head with regard to the humeral condyles is also called **posterior torsion, retrotorsion,** or **retroversion** of the humerus. When the arms hang dependently (passively) at the side, the two articular surfaces of the glenohumeral joint have little contact; the inferior surface of the humeral head rests on only a small inferior portion of the glenoid fossa (see Fig. 7–9).[22,23]

FIGURE 7-10

A. The normal angle of inclination (the angle between the humeral

head and the shaft) varies between 130° and 150°. **B.** Viewing the humeral head from above and looking down the shaft, the humeral head is normally angled posteriorly approximately 30° with regard to an axis through the humeral condyles (angle of torsion).



Foundational Concepts 7–1

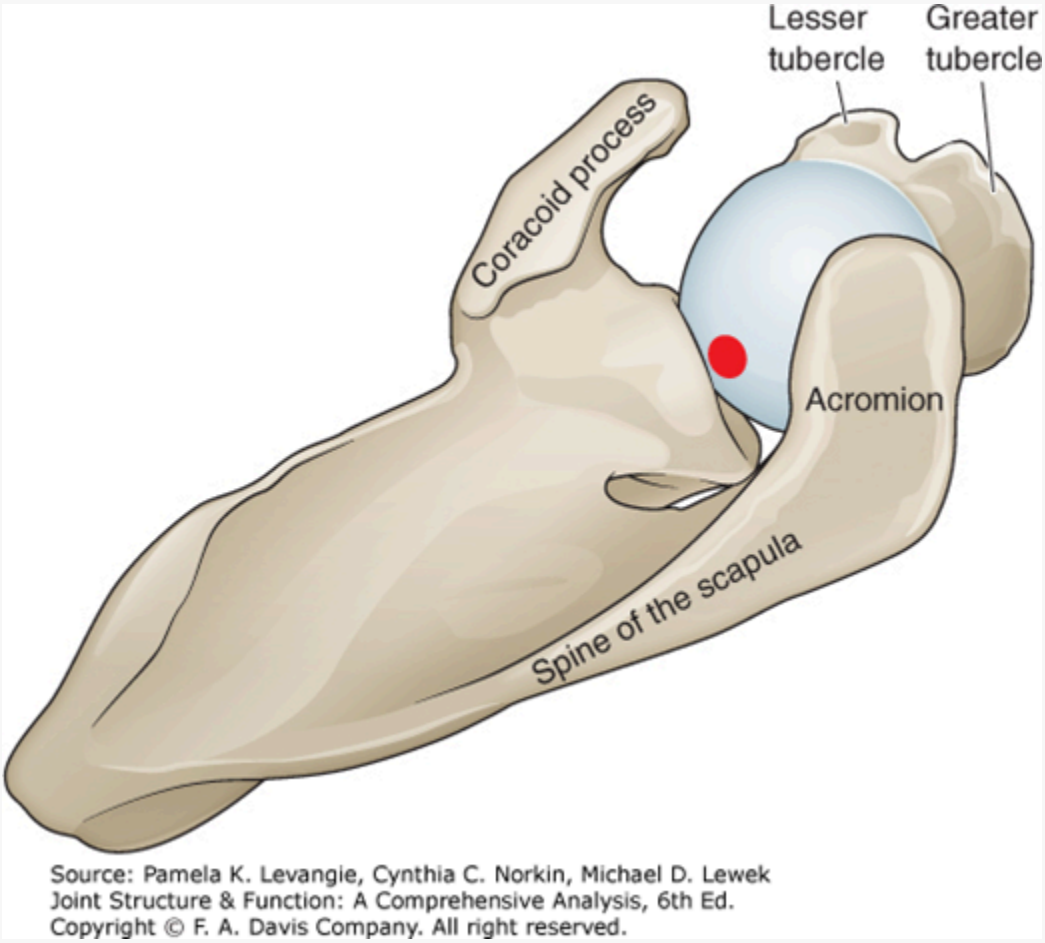
Humeral Retroversion

Because of the internally rotated resting position of the scapula on the thorax (see Fig. 7-8A), normal retroversion of the humeral head increases congruence of the glenohumeral joint by orienting the humeral head toward the articular surface of the glenoid fossa (**Fig. 7–11**). A larger humeral retroversion angle orients the humeral head more posteriorly on the glenoid surface when the humeral condyles are aligned in the coronal plane (anatomic neutral) (**Fig. 7–12**). Importantly, this increased humeral retroversion may result in increased range of lateral rotation of the humerus and reduced range of medial rotation. Increased glenohumeral lateral rotation and decreased glenohumeral medial rotation have been demonstrated in the dominant arm of throwing athletes, and evidence suggests that increased humeral retroversion may be one contributing mechanism for this ROM adaptation.[24,25] Although not as

common, decreased humeral retroversion (or anteversion) may increase medial rotation ROM and decrease lateral rotation ROM (Fig 7–13).

FIGURE 7-11

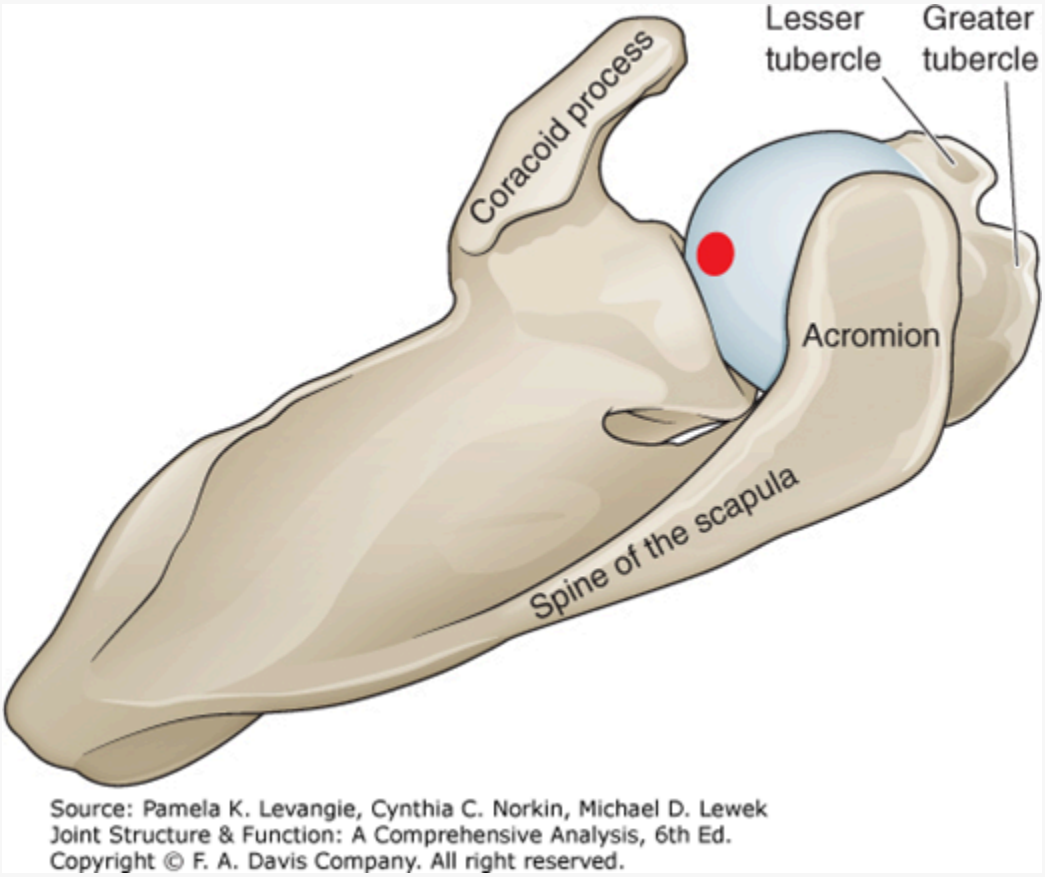
In a superior view of the scapula and humerus, the slightly retroverted angle of humeral torsion turns and effectively centers the humeral head on the glenoid fossa of the scapula given that the scapula is in its internally rotated on the thorax when the scapula is at rest and the humerus is in neutral position.



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FIGURE 7-12

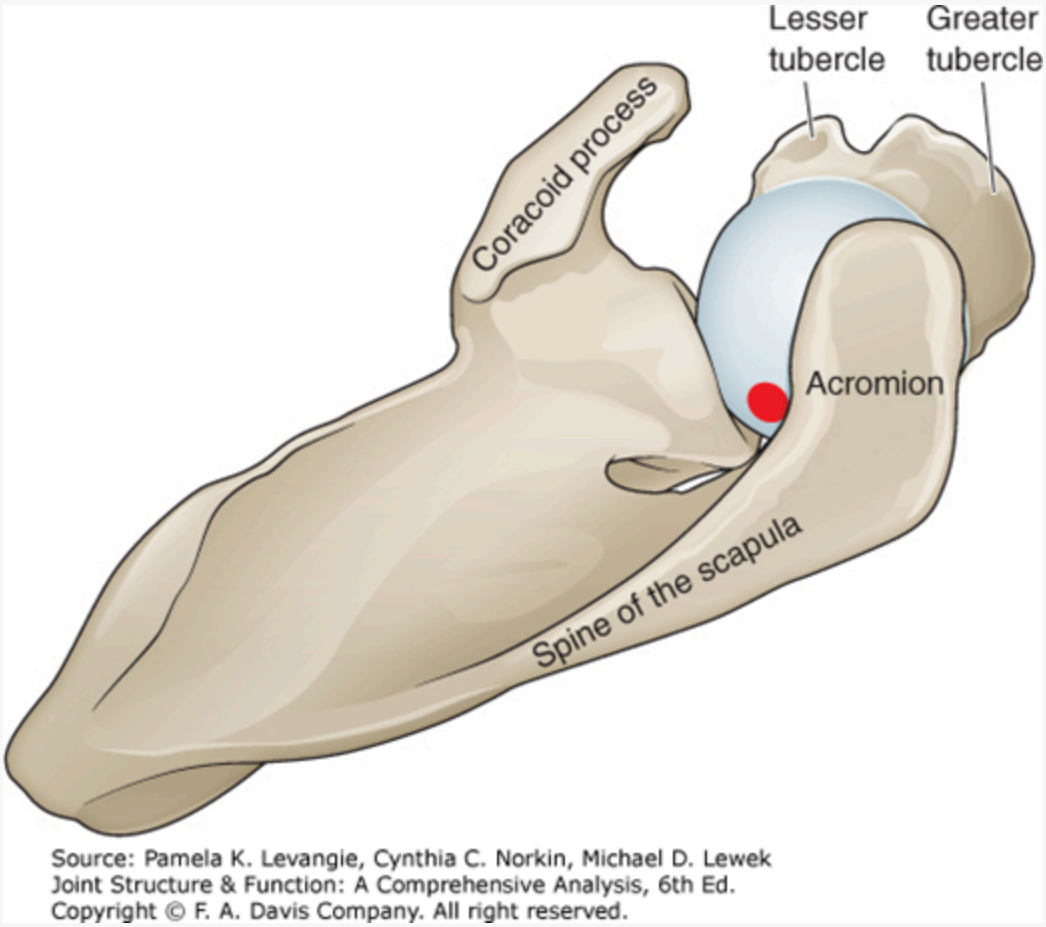
Retroversion of the humerus places the humeral head posteriorly relative to the glenoid fossa of the scapula when the scapula is at rest and the humerus is in neutral position.



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FIGURE 7-13

Reduced retroversion or anteversion of the humerus places the humeral head anteriorly with respect to the glenoid fossa of the scapula when the scapula is at rest and the humerus is in neutral position.



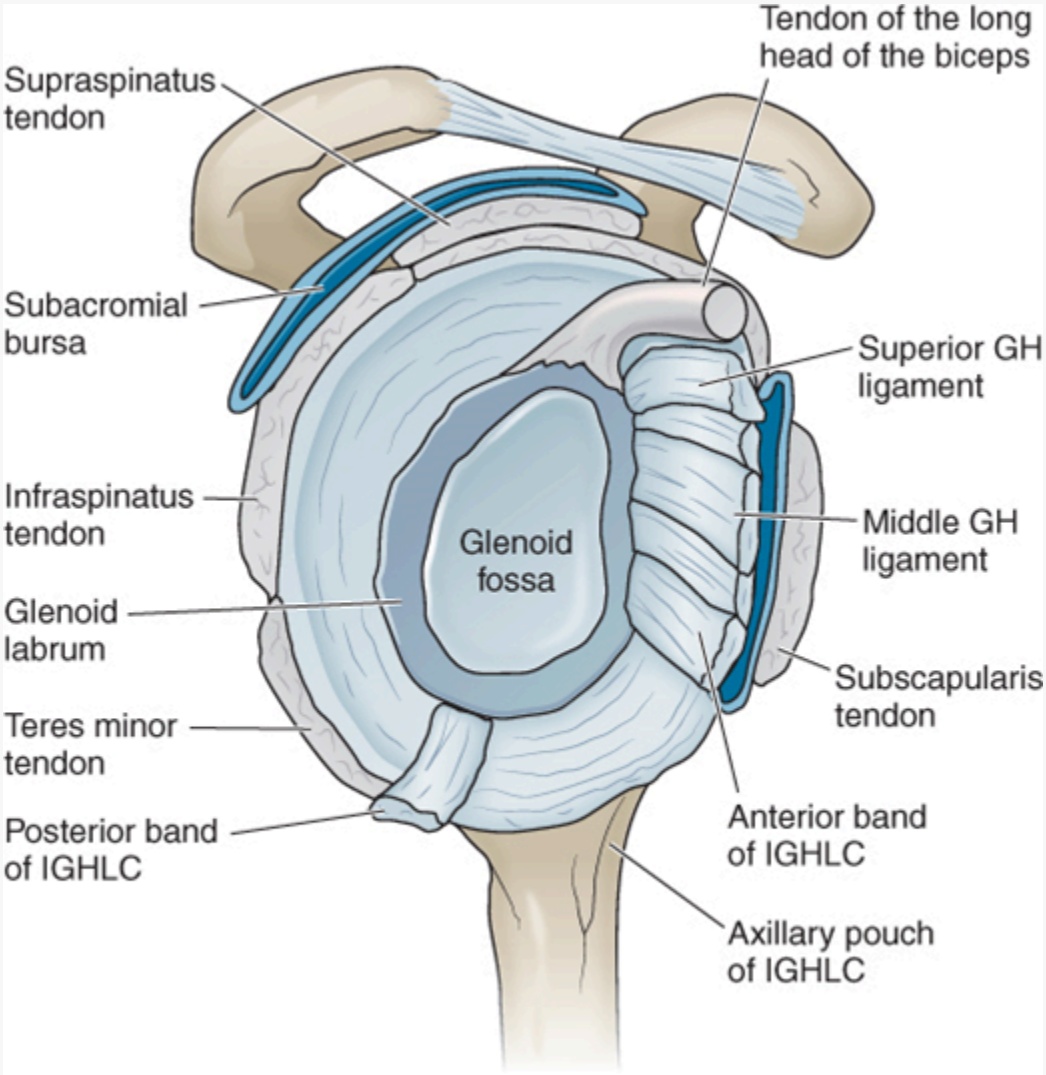
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Glenohumeral Accessory Structures

The total available articular surface of the glenoid fossa is enhanced by the **glenoid labrum.** This accessory structure surrounds and is attached to the periphery of the glenoid fossa (**Fig. 7–14** and **Fig. 7–15**), enhancing the depth or concavity of the fossa by approximately 50%.[26,27] The labrum also functions to resist humeral head translations, protect the bony edges of the glenoid fossa, minimize GH joint friction, and dissipate joint contact forces.[28] The glenoid labrum serves as the attachment site for the glenohumeral ligaments and the tendon of the **long head of the biceps brachii** (Fig. 7-14).[29]

FIGURE 7-14

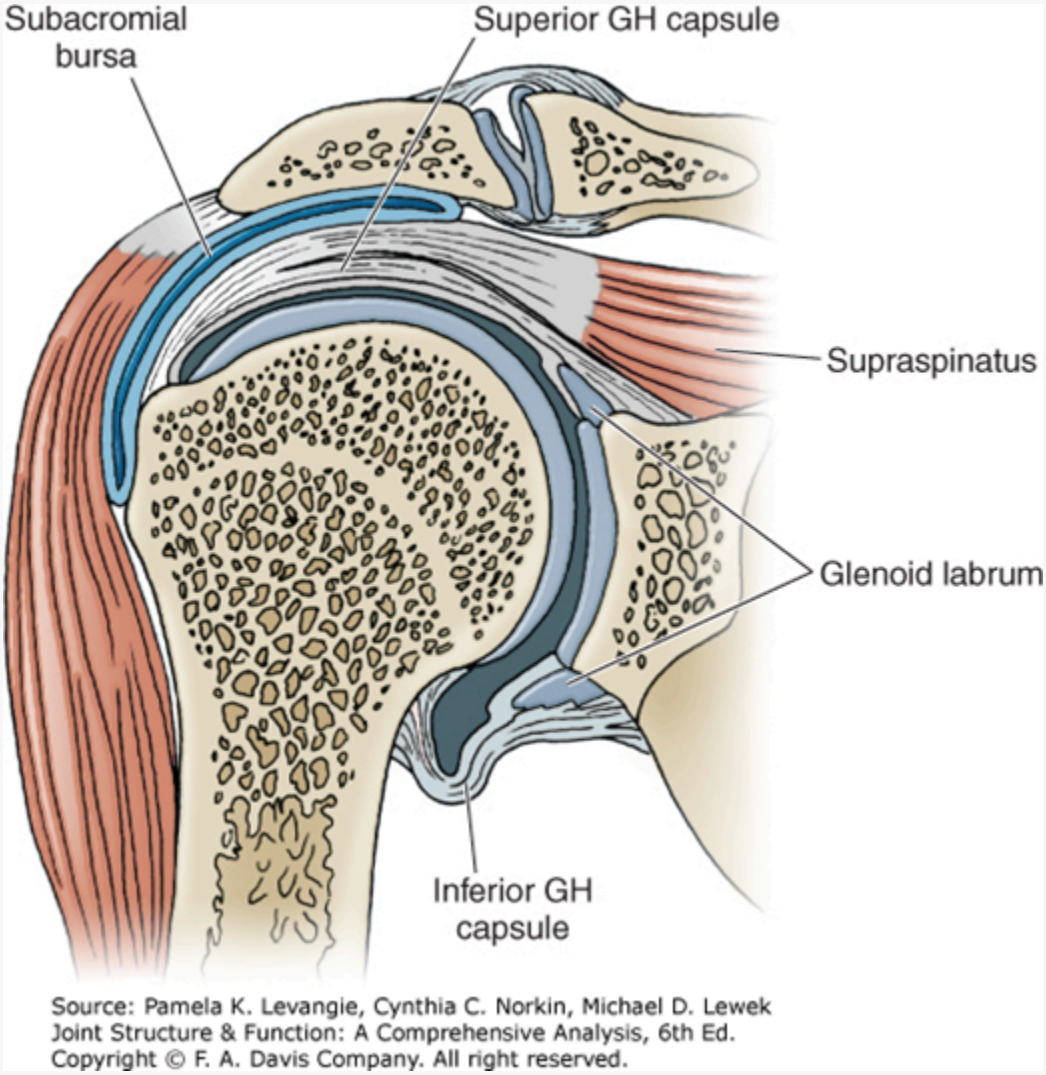
In a direct view into the glenoid fossa (humerus removed), it can be seen that the glenoid labrum increases the articular area of the glenoid fossa and serves as the attachment for the glenohumeral (GH) capsule and capsular ligaments, including the inferior glenohumeral ligament complex (IGHLC).



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FIGURE 7-15

When the arm is at rest at the side, the superior glenohumeral (GH) capsule is taut whereas the inferior capsule is slack.



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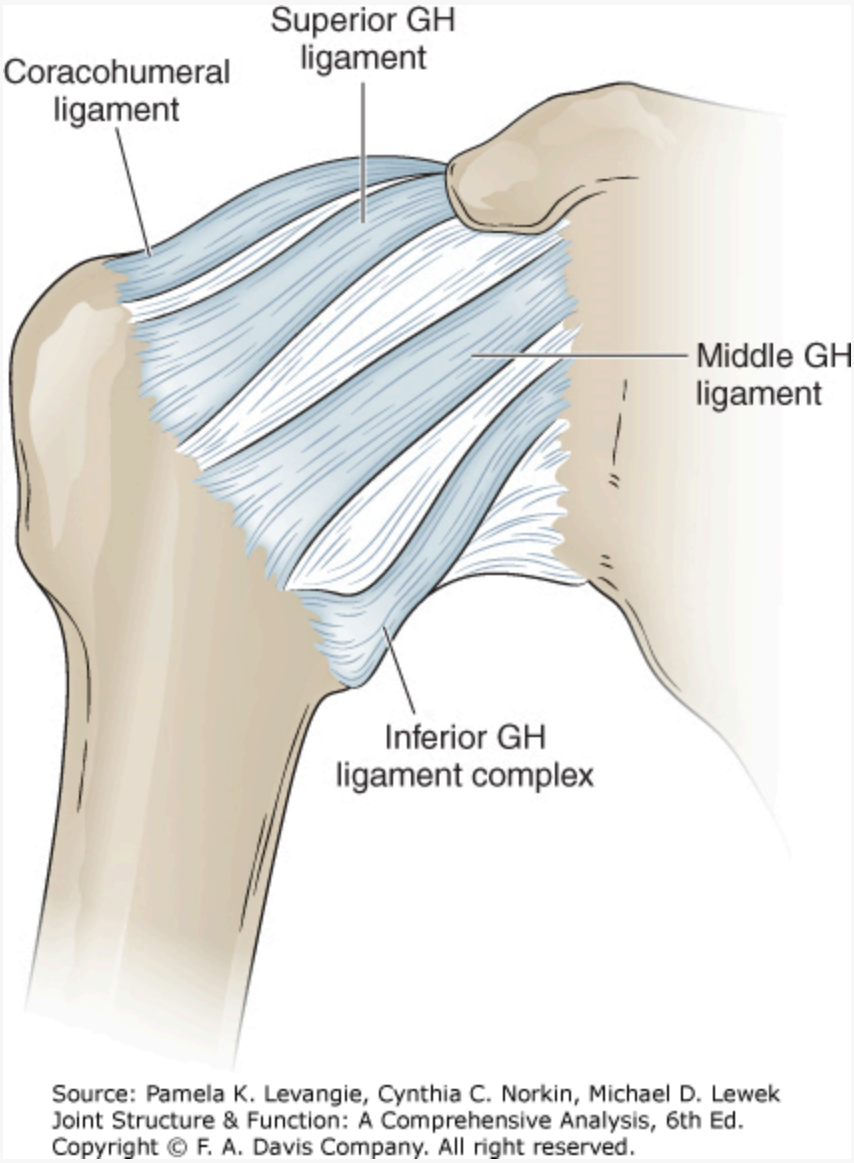
Glenohumeral Capsule and Ligaments

The glenohumeral joint is surrounded by a large, loose capsule that is taut superiorly and slack anteriorly and inferiorly with the arm dependent at the side (see Fig. 7–15). The capsule tightens when the humerus is abducted and laterally rotated, making this the close-packed position for the glenohumeral joint.[30] The capsular surface area is

twice that of the humeral head, and more than 2.5 cm of distraction of the head from the glenoid fossa is possible in the loose-packed position.[6] The relative laxity of the glenohumeral capsule is necessary for the large excursions of the joint but provides little stability without the reinforcement of ligaments and muscles. The capsule is reinforced by the **superior, middle,** and **inferior glenohumeral ligaments** and by the **coracohumeral ligament** (Fig. 7–16). The superior, middle, and inferior glenohumeral ligaments are thickened regions within the capsule tissue itself. In addition to these static capsular reinforcements, the **rotator cuff muscles** and their tendons provide dynamic reinforcement to the capsule through their anatomical proximity to the joint and because the tendons of the cuff muscles insert directly onto the glenohumeral capsule. Even with these reinforcements, the glenohumeral joint is vulnerable to dislocations, particularly in the anterior direction. The three capsular glenohumeral ligaments (superior, middle, and inferior) vary considerably in size, extent, and attachment sites between individuals.[31] Figure 7–14 shows the three ligaments as they appear on the interior surface of the joint capsule, as well as the tendons of the rotator cuff muscles (supraspinatus, infraspinatus, teres minor, and subscapularis).

FIGURE 7-16

The ligamentous reinforcements to the glenohumeral (GH) capsule include the superior, middle, and inferior glenohumeral ligaments, along with the coracohumeral ligament.

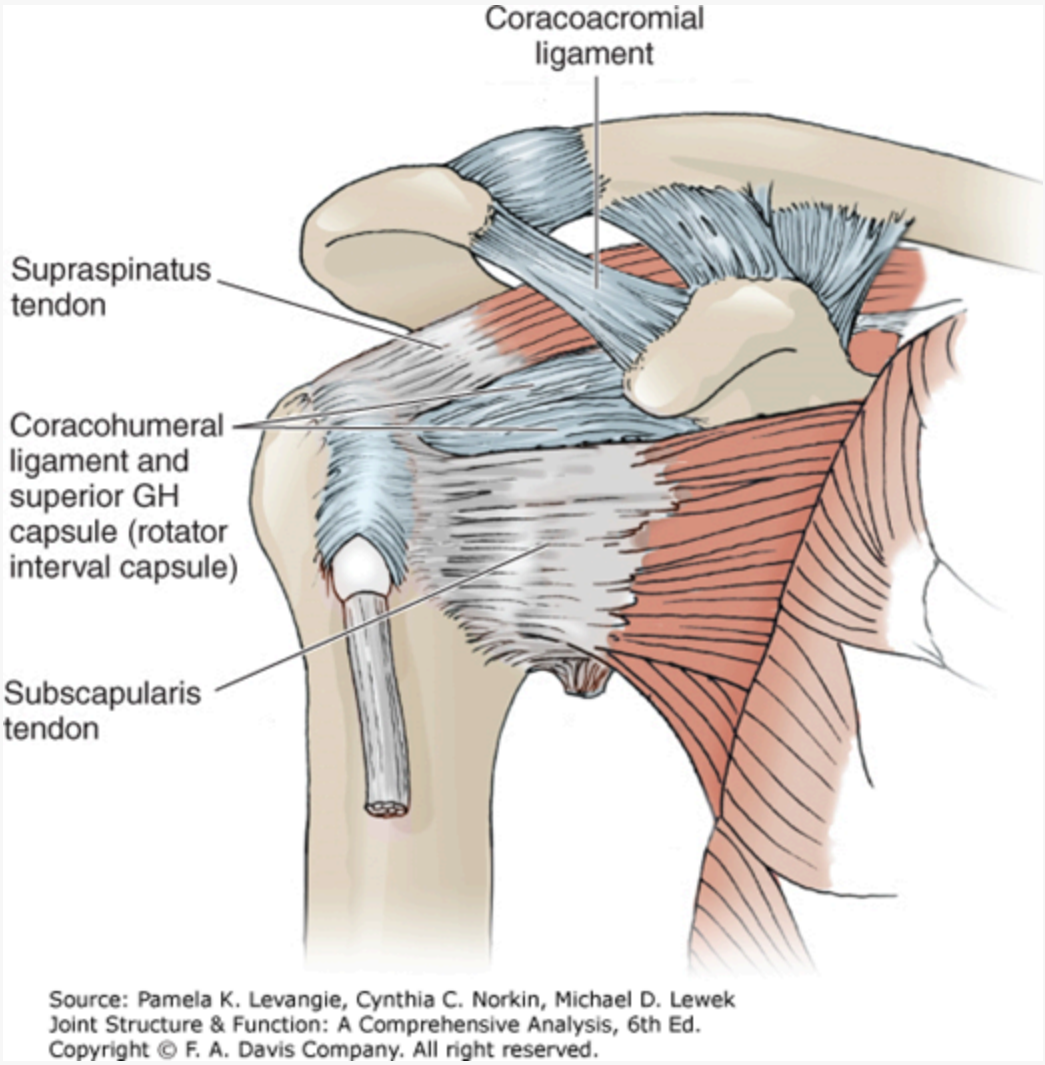


The superior glenohumeral ligament passes from the superior glenoid labrum to the upper neck of the humerus deep to the extracapsular coracohumeral ligament. Harryman and colleagues described the superior glenohumeral ligament, the superior capsule, and the coracohumeral ligament as interconnected structures that bridge the space between the **supraspinatus** and **subscapularis** muscle tendons and form the **rotator interval capsule** (RIC; Fig. 7–17).[32] The middle glenohumeral ligament runs

obliquely from the superior anterior labrum to the anterior aspect of the proximal humerus below the superior glenohumeral ligament attachment (see Figs. 7–14 and 7–16). Ide and colleagues found this ligament to be absent in up to 30% of subjects.[31] The inferior glenohumeral ligament is described as having three components and thus has been termed the **inferior GH ligament complex (IGHLC).**[33] The three components of the complex are the anterior and posterior ligament bands and the axillary pouch in between (see Fig. 7–14). The IGHLC shows position-dependent variability in function, as well as variations in viscoelastic behavior.[27,34]

FIGURE 7-17

The rotator interval capsule is made up of the superior glenohumeral (GH) capsule, superior glenohumeral ligament, and coracohumeral ligament. Together, these structures bridge the gap between the supraspinatus and subscapularis muscle tendons and form the rotator interval capsule.



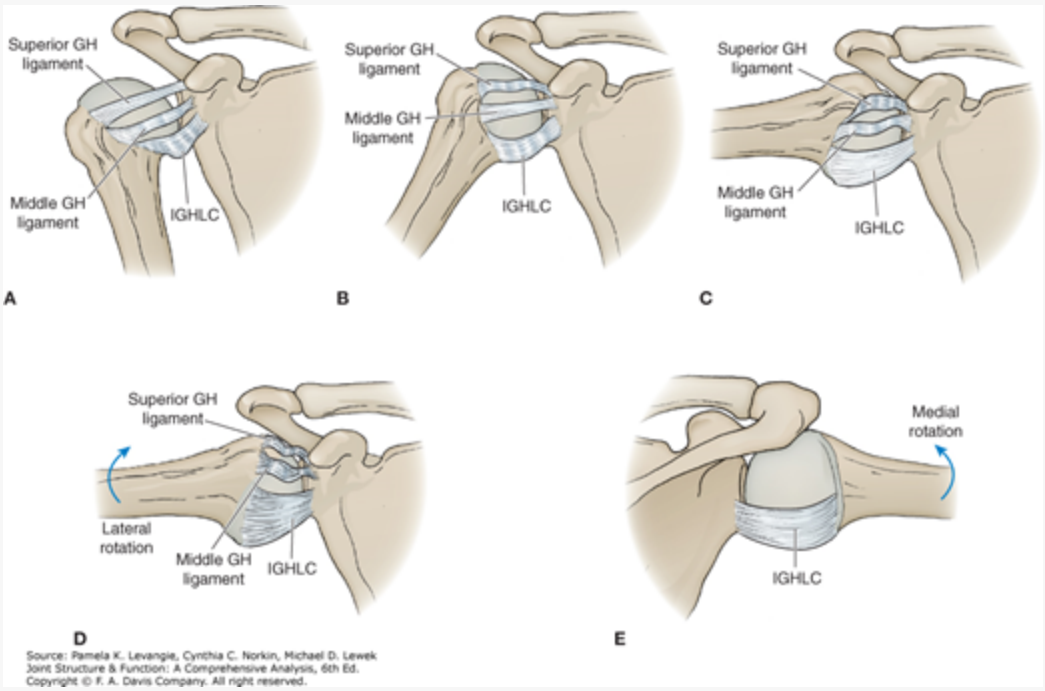
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Study of the movement restraint provided by the glenohumeral ligaments indicates that each ligament contributes differently to glenohumeral stability (Fig. 7–18). The superior glenohumeral ligament and its associated rotator interval capsule structures contribute most to anterior and inferior joint stability by limiting anterior and inferior translations of the humeral head when the arm is at the side (0° abduction; Fig. 7–18A). The middle glenohumeral ligament contributes primarily to anterior joint stability by limiting anterior humeral translation with the arm at the side and up to 60° of abduction (Fig. 7–18B).[35] With abduction beyond 45° or with combined abduction and rotation

(Fig. 7–18C), the IGHLC plays the major role of joint stabilization.[27,33,34,36,37] With abduction, the axillary (inferior capsule) slack is taken up, and the IGHLC resists inferior humeral head translation. If humeral lateral rotation is added (Fig. 7–18D), the anterior band of the IGHLC fans out anteriorly to provide anterior joint stability and resistance to anterior and inferior humeral head translation. If humeral medial rotation is added (Fig. 7–18E), the posterior band of the IGHLC fans out posteriorly and provides posterior joint stability and resistance to posterior and inferior humeral head translation.[38] In all positions of humeral abduction, lateral or medial rotation of the humerus tightens the capsule and glenohumeral ligaments and increases glenohumeral joint stabilization.[39]

FIGURE 7-18

The glenohumeral (GH) ligaments and inferior glenohumeral ligament complex (IGHLC) shown in **A.** an anterior view at rest; **B.** at 45° humeral abduction and neutral rotation; **C.** at 90° humeral abduction and neutral rotation; and **D.** at 90° humeral abduction and external rotation. **E.** In a posterior view, the IGHLC is shown at 90° humeral abduction and medial rotation.



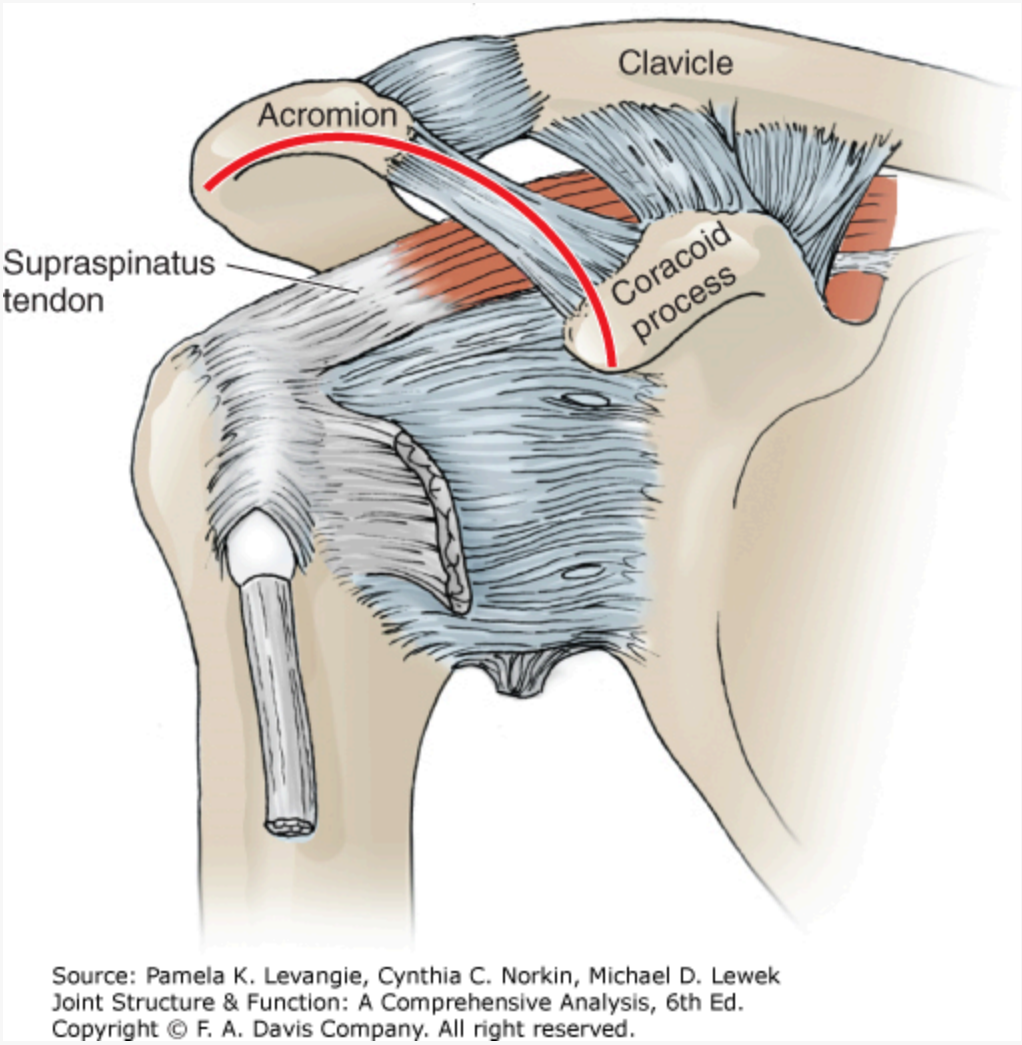
The coracohumeral ligament (see Figs. 7–16 and 7–17) originates from the base of the coracoid process and has two bands. The first band inserts into the edge of the supraspinatus tendon and onto the greater tubercle, joining the superior glenohumeral ligament. The second band inserts into the subscapularis and lesser tubercle.[32,40] The two bands form a tunnel through which the tendon of the long head of the biceps brachii passes.[41] As part of the rotator interval capsule, the coracohumeral ligament limits inferior translation of the humeral head in the dependent arm position. In addition, the coracohumeral ligament resists humeral lateral rotation with the arm adducted. The ligament may also assist in preventing superior translation of the humerus, especially when the dynamic stabilizing force of the rotator cuff muscles is impaired.[40]

Coracoacromial Arch and Bursae

The **coracoacromial** (or **suprahumeral**) **arch** is formed by the coracoid process, the acromial undersurface, the **coracoacromial ligament,** and the inferior surface of the acromioclavicular joint (Fig. 7–19). The coracoacromial arch forms an osteoligamentous vault over the humeral head and the region between the arch and the humeral head is called the *subacromial space.* The **subacromial bursa,** the rotator cuff tendons, and a portion of the tendon of the long head of the biceps brachii lie within the subacromial space and are protected superiorly from direct trauma by the coracoacromial arch. Such trauma could occur through simple daily tasks such as carrying a heavy bag over the shoulder. The arch also acts as a physical barrier to superior translatory forces acting on the humeral head, preventing it from dislocating superiorly. Although beneficial to joint stability, contact of the humeral head with the undersurface of the arch can simultaneously cause painful impingement or mechanical abrasion of the structures within the subacromial space. The supraspinatus tendon is particularly vulnerable because of its location beneath all of the potentially impinging structures except the coracoid process (see Fig. 7–19).

FIGURE 7-19

The coracoacromial (suprahumeral) arch is formed by the coracoid process anteriorly, the acromion and acromioclavicular joint posteriorly, and the coracoacromial ligament superiorly. Together, these structures form an osteoligamentous arch over the humeral head.



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Several bursae are associated with the shoulder complex in general and with the glenohumeral joint specifically, reflecting adaptation to frictional forces between anatomical structures. Although all bursae at the shoulder contribute to function, the most important are the subacromial and subdeltoid bursae. These bursae separate the supraspinatus tendon and head of the humerus from the acromion, coracoid process, coracoacromial ligament, and deltoid muscle. These bursae may be separate entitites but are commonly continuous with each other and are collectively known as the subacromial bursa (see Fig. 7–15). The subacromial bursa reduces friction to permit

smooth gliding between the humerus and supraspinatus tendon and the surrounding structures. Subacromial bursitis is a common finding on imaging, most commonly assumed to be secondary to inflammation or degeneration of the supraspinatus tendon.[11] In the absence of inflammation, bursae are merely layers of synovial tissue in contact with each other with a very thin layer of fluid in between. However, when inflamed, the subacromial bursa may decrease the subacromial space available for rotator cuff tendon clearance.

The subacromial space, also referred to as the **suprahumeral space** or **supraspinatus outlet,** has been quantified by measuring the superior-to-inferior **acromiohumeral interval** on radiographs. This interval averages 10 mm in healthy subjects with the arm adducted at the side and decreases to about 5 mm during elevation of the arm.[42,43] As the acromiohumeral interval decreases, it must accommodate the soft tissue structures within it, as well as the articular cartilage and the capsuloligamentous structures. For this reason, Flatow and colleagues suggested that even during normal motion into humeral elevation, there is some contact of the rotator cuff with the coracoacromial arch.[43] However, recent three dimensional (3D) imaging determined that anatomical approximation of the supraspinatus tendon with the acromion typically occurs at humeral elevation angles below 60°, whereas the smallest acromiohumeral interval is between the acromion and the greater tuberosity of the humerus at approximately 90° of elevation.[44,45] By 90° the supraspinatus tendon rotated medial to the undersurface of the acromion. Subsequently, normal decreases in the acromiohumeral distance during arm elevation may not impact the rotator cuff for most people at elevation angles higher than 60° because the tendon is no longer between the two bony prominences.

When the subacromial space decreases even more than what has been measured in healthy subjects during lower ranges of arm elevation, there is possibility of mechanical impingement or compression of the rotator cuff tendons and the subacromial bursa. The space can decrease by anatomical factors such as changes in the shape, slope, or lateral projection of the acromion, acromial bone spurs, acromioclavicular joint osteophytes, a large coracoacromial ligament, or a disproportionately large humeral head.[46,47] Abnormal scapular or humeral motions can also functionally reduce the size of the subacromial space. Inadequate posterior tilting of the scapula or inadequate upward rotation of the scapula during arm elevation or excessive superior or anterior translation of the humeral head on the glenoid fossa are believed to bring the humeral head in closer proximity to the acromion or coracoacromial ligament, increasing the risk of impingement.[17,48-51] Finally, repetitive impingement may lead to inflammation, fibrosis, and thickening of the soft tissues, further reducing the subacromial space during arm elevation. Abnormal anatomic factors and motion abnormalities have been identified in persons with impingement, as has a decrease in the subacromial space during arm elevation.[17,47,51,52]

Shoulder Joint Function

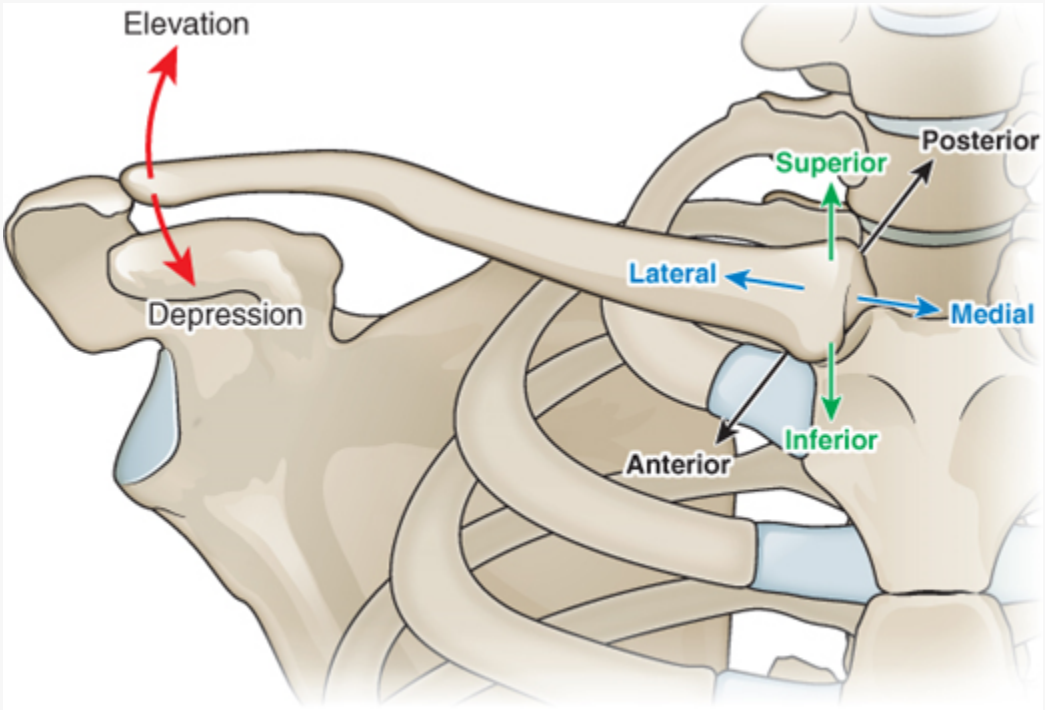
Sternoclavicular Motion

The three rotational degrees of freedom at the sternoclavicular joint are most commonly described as elevation/depression, protraction/retraction, and **anterior/posterior rotation** of the clavicle. Elevation/depression (Fig. 7–20) and protraction/retraction (Fig. 7–21) are named according to the movement of the *lateral* end of the clavicle. Anterior/posterior rotations are long-axis rolling motions of the entire clavicle

(Fig. 7–22) and are named for motion of a point located on the anterior clavicle. Three degrees of translatory motion at the sternoclavicular joint can also occur, although clavicle translations are very small in magnitude in a healthy joint. Translations of the medial clavicle on the sternum are usually defined as occurring in anterior/posterior, medial/lateral, and superior/inferior directions (see Figs. 7–20 and 7–21).

FIGURE 7-20

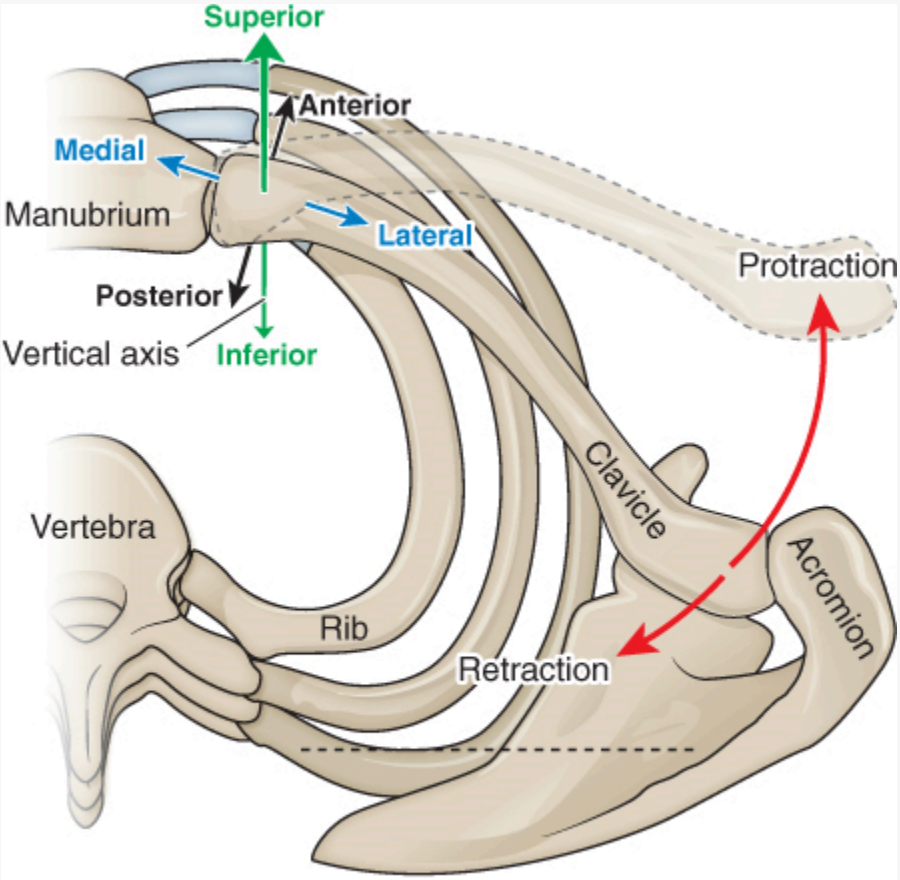
Elevation and depression of the clavicle are named by what is happening to the *lateral* clavicle during rotation at the sternoclavicular joint around an anteroposterior (A-P) axis. Translatory motions of the medial clavicle may occur in a medial/lateral, superior/inferior, or anterior/posterior direction.



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FIGURE 7-21

Protraction and retraction of the clavicle are named by what is happening to the *lateral* clavicle during rotation at the sternoclavicular joint around a vertical axis (superior view). Translatory motions of the medial clavicle may occur in a medial/lateral, superior/inferior, or anterior/posterior direction.

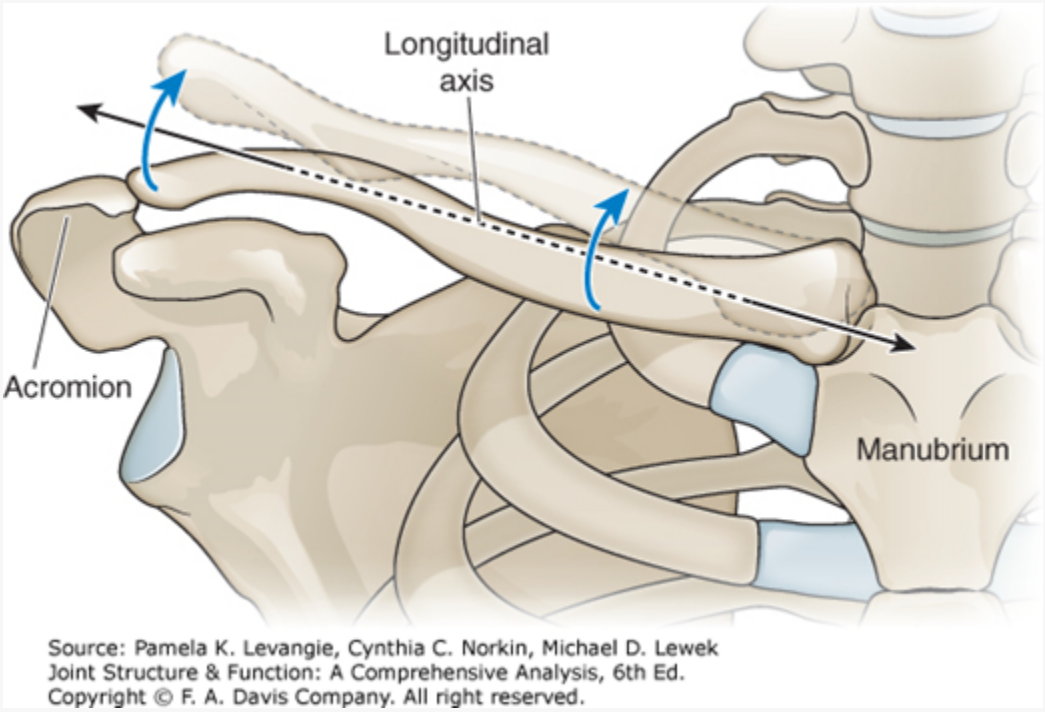


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FIGURE 7-22

Clavicular rotation at the sternoclavicular joint occurs as a spin of the

entire clavicle around a long axis that has a medial/lateral orientation. As the clavicle posteriorly rotates, the lateral end of the clavicle flips up; anterior rotation is a return to resting position.



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Elevation and Depression

The clavicular motions of elevation and depression occur around an approximately anteroposterior (A-P) axis (see Fig. 7–20) between the somewhat convex clavicular surface and the concave surface formed by the sternum and first costal cartilage. The sternoclavicular joint axis is described as lying lateral to the joint at the costoclavicular ligament. The location of this functional (rather than anatomic) axis relatively far from the joint is associated with a larger intra-articular motion of the medial clavicle. With clavicle elevation, the lateral end of the clavicle moves upward as the medial clavicle articular surface rolls superiorly and slides inferiorly on the sternum and first rib. During depression, the lateral clavicle moves downward as the medial clavicle articular surface

rolls inferiorly and slides superiorly. The range of clavicular elevation has been reported as up to 48°, whereas passive depression from neutral is limited to less than 15°.[53] The full available range of clavicle elevation is generally not used during functional arm elevation.[54,55]

Protraction and Retraction

Clavicle protraction and retraction occur at the sternoclavicular joint around an approximately vertical axis that also appears to be located at the costoclavicular ligament (see Fig. 7–21). With protraction, the lateral clavicle moves anteriorly in the transverse plane, while with retraction the lateral clavicle moves posteriorly. The configuration of joint surfaces in this plane is the opposite of that for elevation/depression, with a concave medial end of the clavicle meeting a sternum surface that is convex. During protraction, the medial clavicle is expected to roll and glide anteriorly on the sternum and first costal cartilage with the opposite joint kinematics occurring during retraction. Ap proximately 15° to 20° of protraction and 30° of retraction are available.[16,53,55,56]

Anterior and Posterior Rotation

Anterior/posterior, or long axis, rotation of the clavicle (see Fig. 7–22) occurs as a spin between the saddle-shaped surfaces and inferior conus of the medial clavicle and manubriocostal facet and disc. Unlike many joints, which can rotate in either direction from their resting position, the clavicle rotates primarily posteriorly from neutral, bringing the inferior surface of the clavicle to face anteriorly. This has also been referred to as backward or upward rotation rather than posterior rotation.[1] From this posteriorly rotated position, the clavicle can then rotate anteriorly to return to neutral. Available

anterior rotation past neutral is limited to less than 10°.[1] The range of available clavicular posterior rotation may be as much as 50°.[53] The axis of rotation runs longitudinally through the clavicle, intersecting the sternoclavicular and acromioclavicular joints.

Sternoclavicular Stability

The bony morphology, capsule, ligaments, and disc of the sternoclavicular joint combine to create an articulation that contributes to the great mobility of the arm while remaining a stable link to the axial skeleton. Although the sternoclavicular joint is considered incongruent, it does not typically undergo the degree of degenerative change common to the other joints of the shoulder complex.[10,11] Strong force-dissipating structures such as the sternoclavicular disc and the costoclavicular ligament minimize articular stresses and limit intra-articular motion that could lead to subluxation or dislocation. Reported dislocations of the sternoclavicular joint represent only 1% of joint dislocations in the body and only 3% of shoulder girdle dislocations, although subluxations may be more frequent.[57]

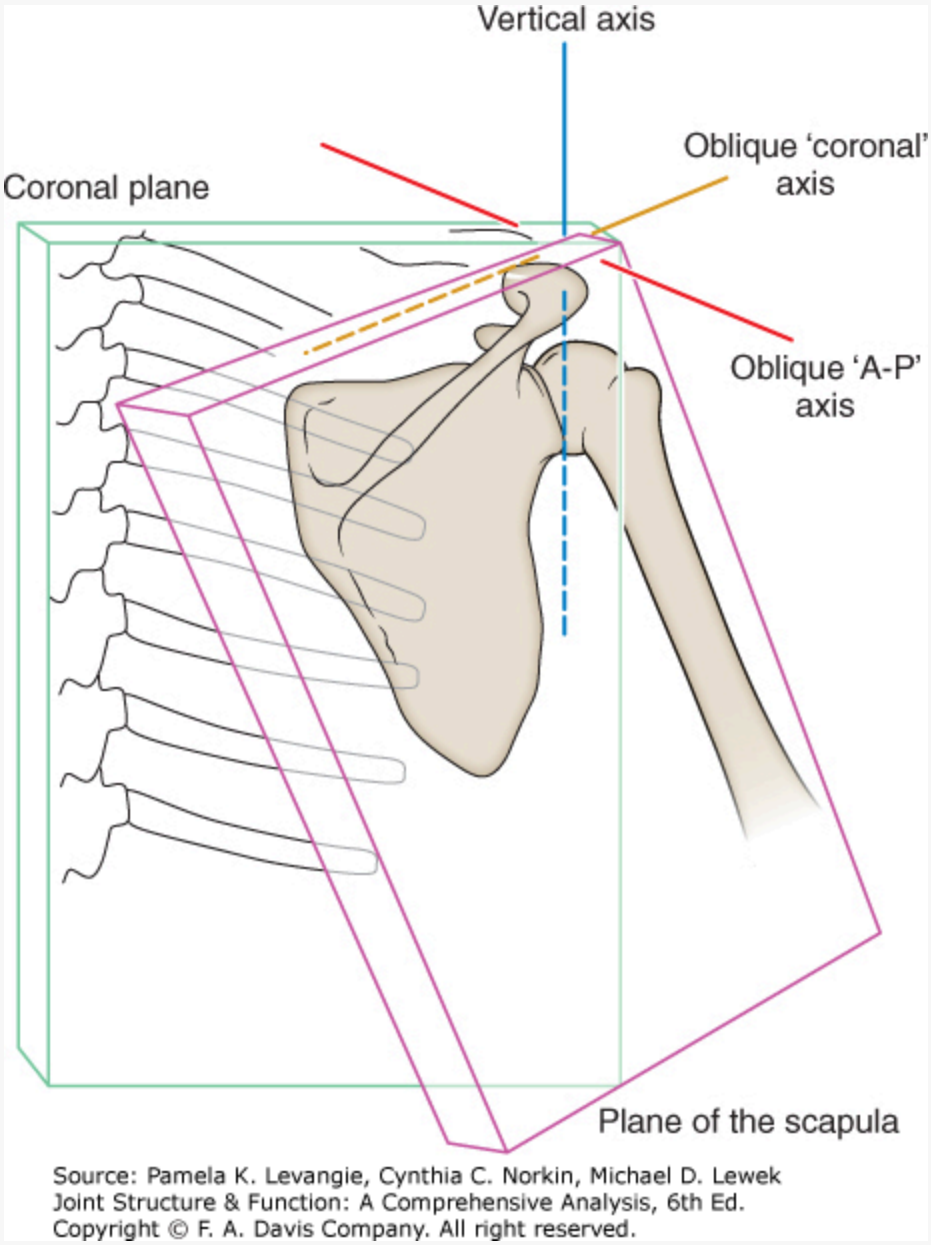
Acromioclavicular Kinematics

The small articular facets of the acromioclavicular joint vary widely across individuals and afford limited motion, which has led to difficulty in clearly determining axes of motion and joint movements. The primary rotations at the acromioclavicular joint are **internal/external rotation, anterior/posterior tilting,** and **upward/downward rotation.** These motions occur around axes that are oriented relative to the **plane of the scapula** rather than to the cardinal planes. The plane of the scapula is where the scapula sits at rest, 30° to 45° in front of the coronal plane (see Fig. 7–9A). Although internal/external

rotation of the scapula at the AC joint occurs around a nearly vertical axis, anterior/posterior tilting occurs around an oblique “coronal” axis, and upward/downward rotation around an oblique “A-P” axis (Fig. 7–23). Acromioclavicular joint motion also influences, and is influenced by, long-axis rotation of the clavicle. In addition, small magnitude translations at the acromioclavicular joint can occur. Acromioclavicular translations are usually described as anterior/posterior, medial/lateral, and superior/inferior.

FIGURE 7-23

The acromioclavicular rotary axes of motion are oriented in relation to the plane of the scapula rather than in relation to the cardinal planes, resulting in coronal and anteroposterior (A-P) axes that are oblique to their cardinal orientation.



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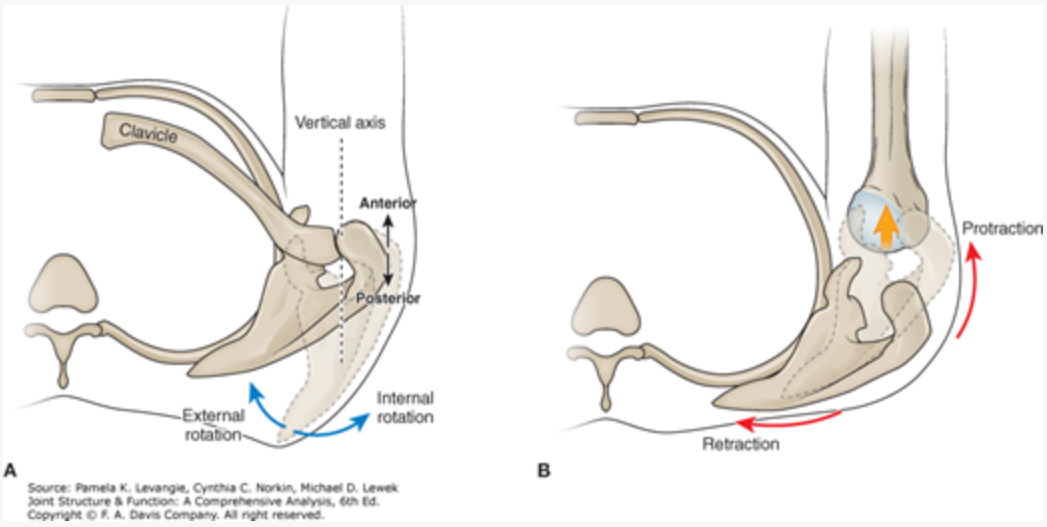
Internal and External Rotation

Internal/external rotation of the scapula relative to the clavicle occurs around an approximately vertical axis through the acromioclavicular joint (Fig. 7–24). Internal and external rotation at the acromioclavicular joint can be visualized best as orienting the glenoid fossa of the scapula anteromedially and posterolaterally, respectively (Fig.

7–24A). These motions occur in part to maintain contact of the scapula with the horizontal curvature of the thorax as the clavicle protracts and retracts, sliding the scapula around the thorax in scapular internal and external rotation (Fig. 7–24B). These motions also “aim” the glenoid fossa toward the plane of humeral elevation, which is an important function for maintaining congruency and stability between the humeral head and scapula, and for maximizing the function of glenohumeral muscles, capsule, and ligaments. The available ROM at the acromioclavicular joint is difficult to measure. Dempster provided a range of 30° for combined internal and external rotation ROM in cadaveric acromioclavicular joints separated from the thorax.[1] Smaller values (20° to 35°) have been reported in vivo during arm motions, although up to 60° may be possible with full-range motions reaching forward and across the body.[16,53,55,56]

FIGURE 7-24

A. A superior view of scapular internal and external rotation at the acromioclavicular (AC) joint. Although the rotation motions are drawn at the vertebral border, the motions are named with the glenoid fossa of the scapula as the reference. The acromion also has small amounts of anterior and posterior translatory motions that can occur. **B.** Protraction and retraction of the scapula require internal and external rotation at the AC joint for the scapula to follow the convex thorax and orient the glenoid fossa with the plane of elevation (e.g., anteriorly with flexion of the arm).



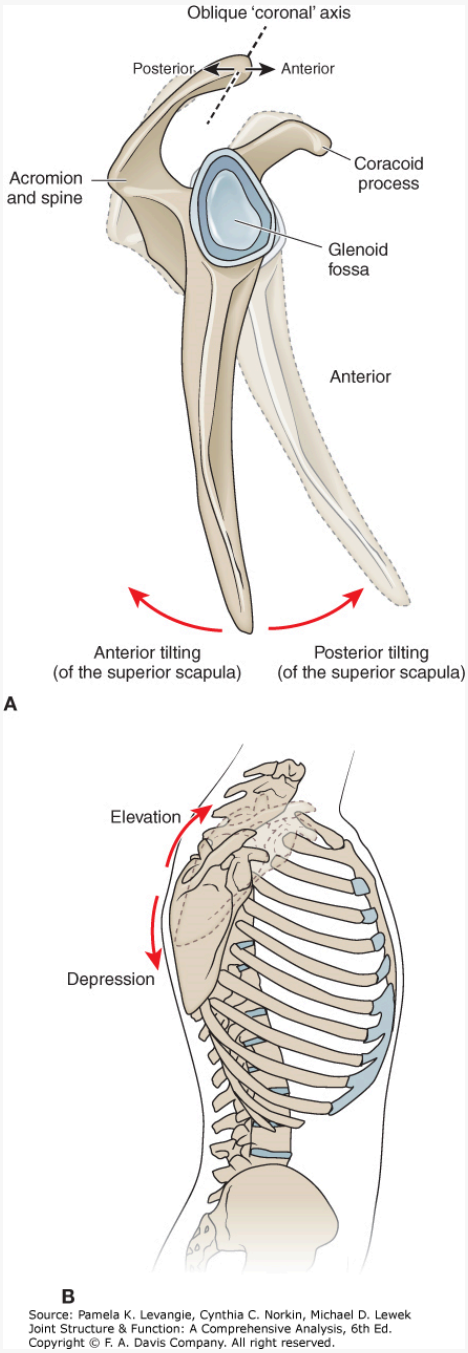
Anterior and Posterior Tilting

Anterior/posterior tilting of the scapula relative to the clavicle occurs around an oblique "coronal" axis through the joint (Fig. 7–25). Anterior tilting results in the acromion moving forward and the inferior angle of the scapula moving backward (Fig. 7–25A). During posterior tilting the acromion moves backward and the inferior angle moves forward. Scapular tilting, like scapula internal/external rotation, occurs to maintain the contact of the scapula with the contour of the thorax and to orient the glenoid fossa relative to the humeral head. As the scapula moves upward or downward on the rib cage in elevation or depression, tilting may occur to maximize contact between the anterior scapula and the curvature of the ribs (Fig. 7–25B). However, the scapula does not always follow the curvature of the thorax precisely. For example, during normal flexion or abduction of the arm, the scapula posteriorly tilts on the thorax as the scapula is upwardly rotating. Available passive motion into anterior/posterior tilting at the acromioclavicular joint is 60° in cadaveric acromioclavicular joint specimens separated from the thorax.[1] The magnitude of anterior/posterior tilting during in vivo

arm elevation is approximately 20°, although up to 40° or more may be possible in the full range from maximum flexion to extension.[16,58]

FIGURE 7-25

A. A lateral view of scapular anterior and posterior tilting at the acromioclavicular joint. Although the directional arrows are drawn at the inferior angle, the motions are named with the *superior* aspect of the scapula as the reference. The acromion also has small amounts of anterior and posterior translatory motions that can occur. **B.** Elevation and depression of the scapula can include anterior and posterior tilting, respectively, for the scapula to approximate the convex thorax.



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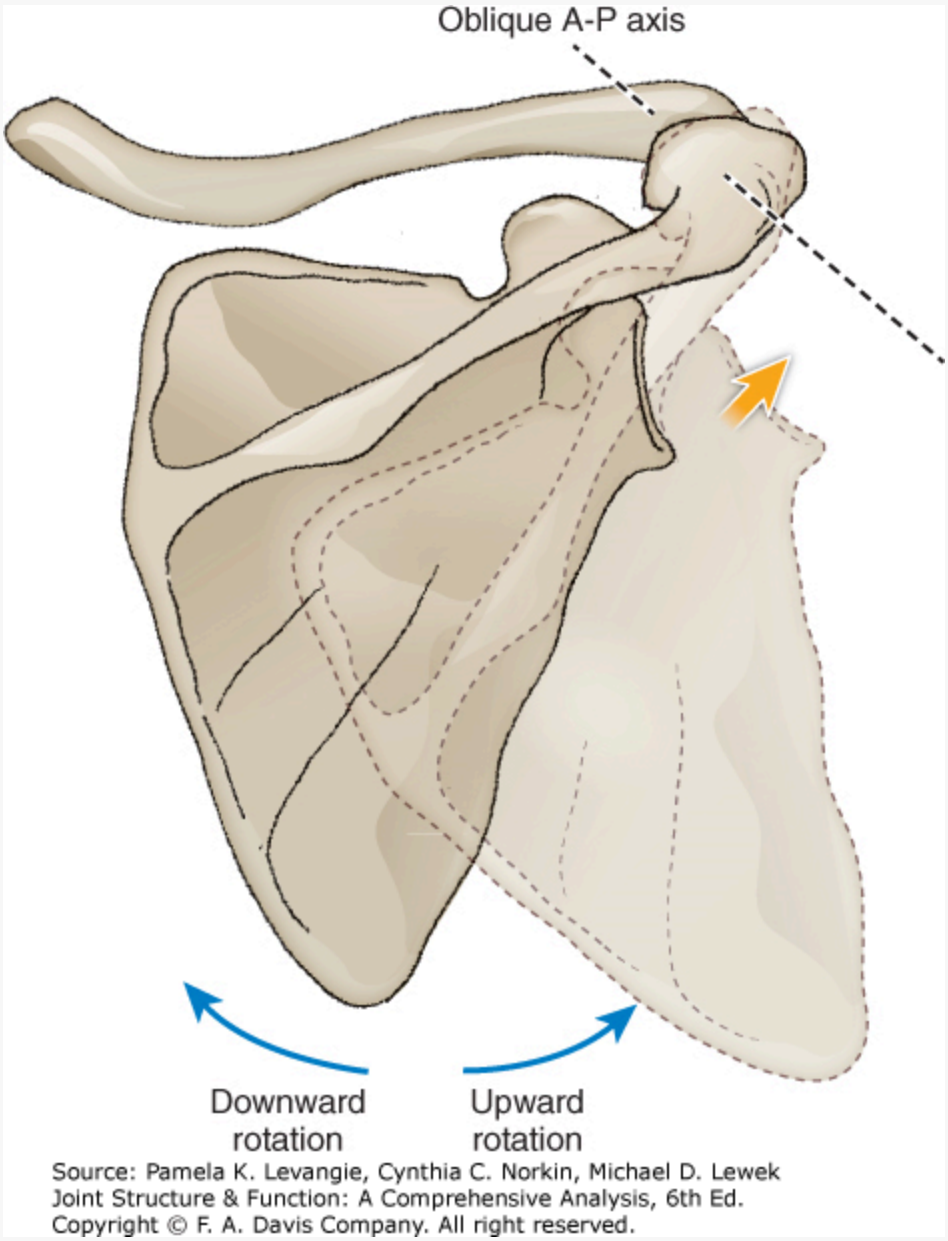
Upward and Downward Rotation

Upward/downward rotation of the scapula relative to the clavicle occurs about an oblique “A-P” axis that is roughly perpendicular to the plane of the scapula, passing midway between the joint surfaces (Fig. 7–26). Upward rotation tilts the glenoid fossa

upward and moves the inferior angle laterally, while downward rotation does the opposite. The amount of available passive motion into upward/downward rotation occurring directly at the acromioclavicular joint is limited by the coracoclavicular ligament. For isolated upward rotation at the acromioclavicular joint, the coracoid process and superior border of the scapula need to move downward (inferiorly) relative to the clavicle, a motion that is restricted by tension in the coracoclavicular ligaments (see Fig. 7–17). The amount of available upward rotation is also dependent in part on rotation of the clavicle around its long axis. Because of the attachment of the coracoclavicular ligaments to the undersurface and posterior edge of the clavicle, posterior rotation of the clavicle releases tension on the coracoclavicular ligaments and "opens up" the acromioclavicular joint, allowing upward rotation to occur.[54] Conway describes 30° of upward rotation and 17° of downward rotation actively at the acromioclavicular joint in vivo.[53] Others have described 15° to 16° of upward rotation occurring during active humeral abduction.[16,58]

FIGURE 7-26

Upward/downward rotation of the scapula at the acromioclavicular joint occurs around an approximately A-P (perpendicular to the plane of the scapula) axis. Although the directional arrows are drawn at the inferior angle, the motions are named with the glenoid fossa of the scapula as the reference, with the glenoid facing upwardly in upward rotation.



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Acromioclavicular Stability

Unlike the sternoclavicular joint, the acromioclavicular joint is not very stable, making it susceptible to both trauma and degenerative changes. Trauma to the acromioclavicular joint most often occurs in the first three decades of life, during either contact sports or a fall on the shoulder with the arm adducted. Most often the result of

high inferior forces on the acromion, trauma results in acromioclavicular joint disruption ranging from sprains and subluxations to dislocations.

Expanded Concepts 7–2

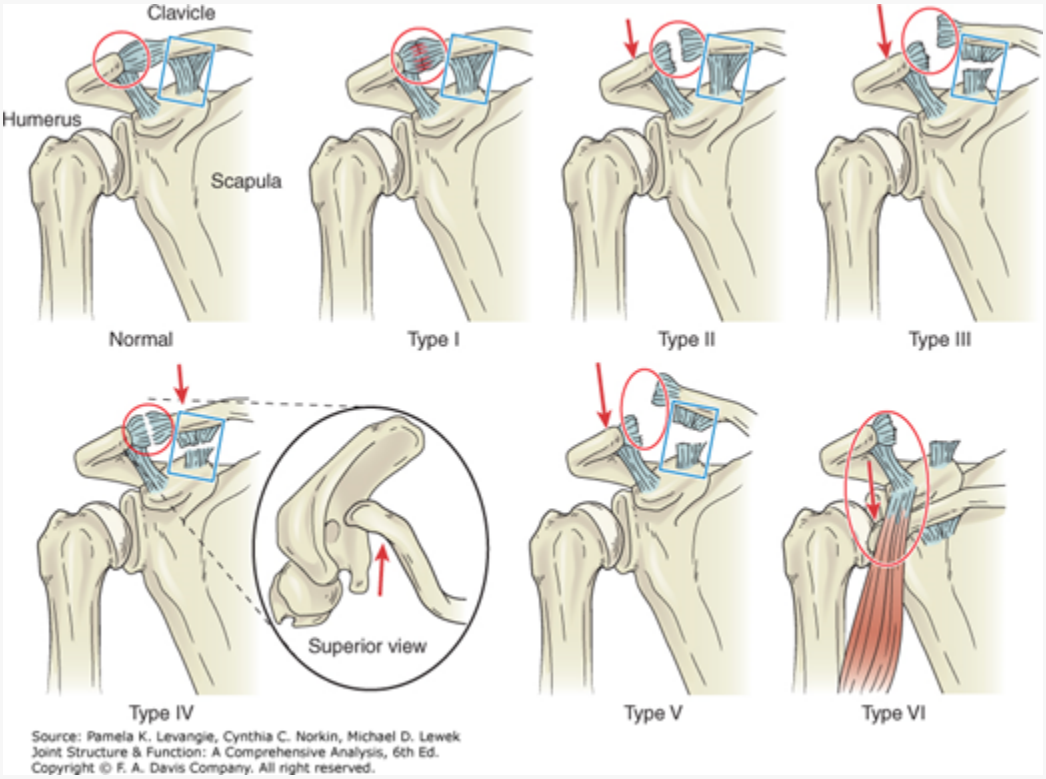
Classifying Acromioclavicular Dislocations

The acromioclavicular joint is susceptible to traumatic injury through accidents or contact sports. These acromioclavicular dislocations are graded by the relationship of the displacement between the acromion and clavicle and by the extent of the displacement (Fig. 7–27). Various classification schemes exist; most commonly, type I injuries consist of a sprain to the acromioclavicular ligaments only, type II injuries consist of ruptured acromioclavicular ligaments and sprained coracoclavicular ligaments, and type III injuries result in the rupture of both sets of ligaments, with a 25% to 100% increase in coracoclavicular space. Types II and III acromioclavicular dislocations involve inferior displacement of the acromion in relation to the clavicle due to loss of support from the coracoclavicular ligaments. Type IV injuries have a posteriorly displaced lateral clavicle, often pressing into the trapezius posteriorly, with complete rupture of both the acromioclavicular and coracoclavicular ligaments. Type V injuries also involve an inferior displacement of the acromion and complete rupture of both sets of ligaments, but unlike type III, they exhibit between three and five times greater coracoclavicular space than normal. Type VI injuries have an inferiorly displaced clavicle in relation to the acromion, with complete ligament rupture and displacement of the lateral clavicle into a subacromial or subcoracoid position. Types IV, V, and VI are much rarer and each requires surgical management.[59] scapular malposition and dyskinesis following type III injuries, a finding in nearly 60% of subjects in a recent analysis.[61] These reports suggest that more aggressive treatment and rehabilitation may be warranted following acromioclavicular joint injuries.

FIGURE 7-27

Classification of acromioclavicular (AC) dislocations based on changes to the AC joint (circled) and degree of disruption to the

coracoclavicular ligaments (rectangle). **Type I:** sprain to the acromioclavicular ligaments only. **Type II:** ruptured acromioclavicular ligaments and sprained coracoclavicular ligaments with slight inferior displacement of acromion. **Type III:** rupture of both sets of ligaments and inferior displacement of acromion. **Type IV:** complete ligamentous disruption and posteriorly displaced lateral clavicle. **Type V:** substantial inferior displacement of the acromion and complete rupture of both sets of ligaments. **Type VI:** complete ligament rupture and displacement of the lateral clavicle into a subacromial or subcoracoid position.



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Patient Application 7–1

Acromioclavicular Pathology

Kelsey Rigby is a 28-year-old woman who is complaining of right shoulder pain. She reports a prior right shoulder acromioclavicular joint separation from 9 years ago that was treated by immobilization of the arm in a sling. This treatment is consistent with an injury ranging from type I to type III in severity, which is frequently not surgically stabilized. With a type I injury, she may have healed well and may have normal acromioclavicular joint function, leading the examiner to look elsewhere for the cause of her pain. If the past injury was a type III injury, she may still have substantial instability or disruption of the claviculo-scapular linkage. The position and prominence of the lateral clavicle on the right in comparison with her left (uninvolved) side can provide some insight into the injury. A prominent lateral clavicle with a step down to the acromion (step sign) would be consistent with inferior scapular (or superior clavicular) positioning related to a past type II or III injury. Although this injury is commonly not surgically stabilized because many surgeons believe it unnecessary, follow-up studies indicate rates of residual symptoms in persons with past acromioclavicular joint injuries from 36% in type I injuries to 69% in type III injuries.[60] One residual problem is

Scapulothoracic Kinematics

The motions of the scapula from the resting position include three rotations that are similarly named as those that occur at the acromioclavicular joint. These are upward/downward rotation, internal/external rotation, and anterior/posterior tilting. Acromioclavicular motions occur between the lateral clavicle and acromion while scapulothoracic motions take place between the scapula and the thorax. Scapulothoracic movements occur either from sternoclavicular joint motions, acromioclavicular joint motions, or often, a combination of both. The scapula on the thorax is also often described as having translatory motions of scapulothoracic **elevation/depression** and **protraction/retraction.** These scapulothoracic motions are often described as if they occur independently. However, the linkage of the scapula to the acromioclavicular and sternoclavicular joints actually prevents scapulothoracic

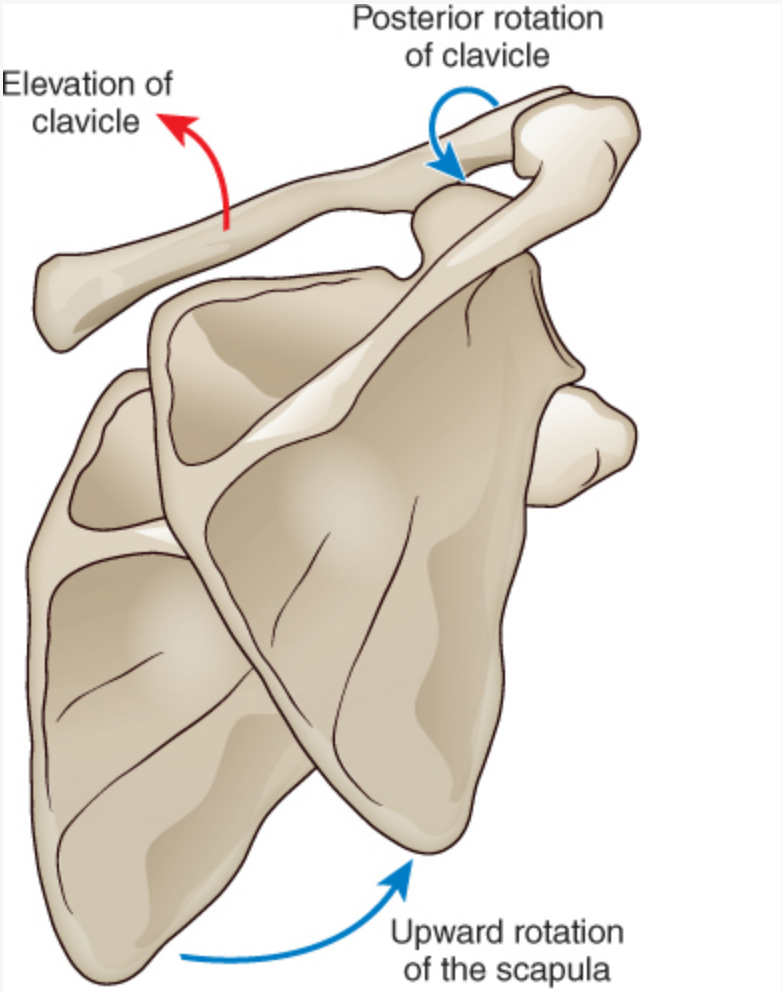
motions from occurring in isolation or as true translatory motions.

Upward and Downward Rotation

Upward rotation of the scapula on the thorax (Fig. 7–28) is the principal motion of the scapula observed during active elevation of the arm and plays a significant role in increasing the arm's range of elevation overhead. Approximately 50° to 60° of upward rotation of the scapula on the thorax is typically available. Because scapulothoracic motions are combined joint motions, differing proportions of upward/downward rotation of the scapula on the thorax are contributed by clavicular posterior/anterior rotation and elevation/depression at the sternoclavicular joint, and by upward/downward rotation of the scapula at the acromioclavicular joint.[62]

FIGURE 7-28

Full upward rotation of the scapula is produced somewhat by elevation at the sternoclavicular joint, and more so by clavicular posterior rotation, and upward rotation at the acromioclavicular joint.



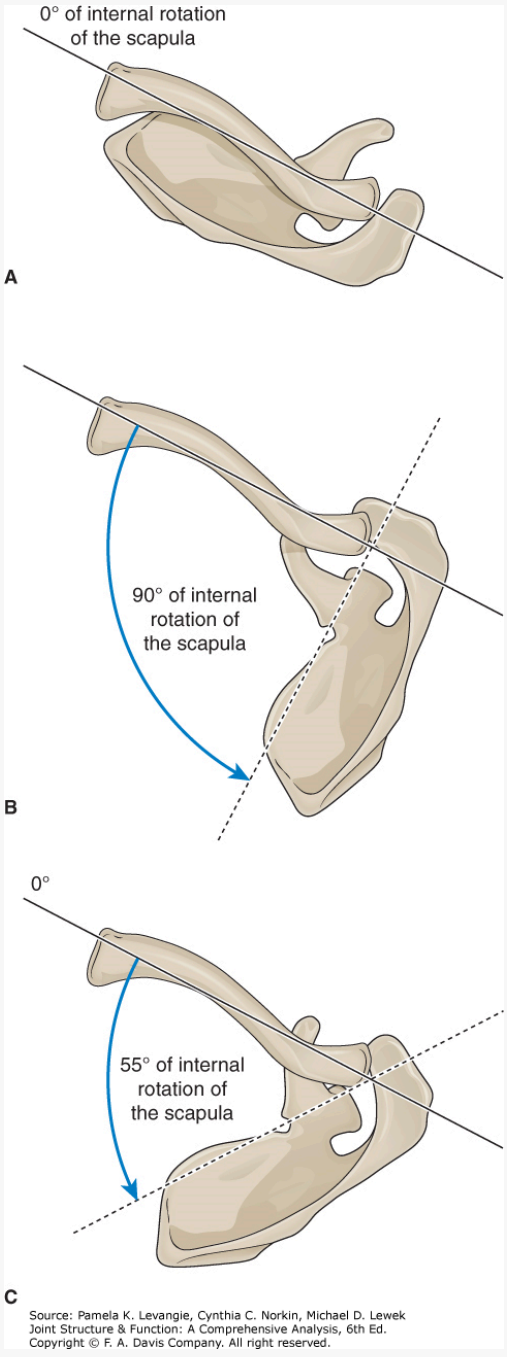
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Because the axes of the sternoclavicular and scapulothoracic joints are not parallel, the relationships between sternoclavicular and acromioclavicular joint motions and scapulothoracic motion may be challenging to visualize. However, considering the orientation of the sternoclavicular and scapulothoracic joint axes in two hypothetical configurations may assist in visualizing these relationships (Fig. 7–29). First, if the long axis of the clavicle were parallel to the plane of the scapula (Fig. 7–29A) such that acromioclavicular joint internal rotation was 0° (scapula in resting position), scapulothoracic upward rotation would directly couple with *sternoclavicular joint*

elevation.

FIGURE 7-29

Coupling of rotation of the clavicle at the sternoclavicular (SC) joint with scapular motion on the thorax. **A.** Theoretical acromioclavicular (AC) joint internal rotation of 0° (clavicular plane and scapular plane are aligned) would result in direct coupling of clavicular elevation at the SC joint and scapular upward rotation. **B.** Theoretical AC joint internal rotation of 90° would result in direct coupling of clavicular posterior rotation at the SC joint with scapular upward rotation. **C.** A typical acromioclavicular joint internal rotation of approximately 55° (~two-thirds of the way toward 90°) means that two-thirds of any posterior rotation motion of the clavicle will translate into upward rotation of the scapula, whereas only one-third of any clavicular elevation motion will translate into upward rotation of the scapula.



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In other words, sternoclavicular elevation (rise of the lateral end of the clavicle) would result in scapulothoracic upward rotation, and vice versa. Second, if the long axis of the clavicle were perpendicular to the plane of the scapula such that acromioclavicular joint internal rotation was 90° (Fig. 7–29B), scapulothoracic upward rotation would

directly couple with *posterior rotation of the clavicle at the sternoclavicular joint.* These two hypothetical configurations suggest scapulothoracic upward rotation can be related to both clavicular elevation and posterior rotation of the clavicle, both occurring at the sternoclavicular joint. The average angle of acromioclavicular joint internal rotation is approximately 60° (Fig. 7–29C), or about two-thirds of a 90° acromioclavicular joint internal rotation position.[16,62] Consequently, two-thirds of the motion of *clavicular posterior rotation at the sternoclavicular joint* will translate into or couple with scapulothoracic upward rotation, while only one-third of *clavicular elevation at the sternoclavicular joint* will result in scapulothoracic upward rotation.[62]



Expanded Concepts 7–3

Terminology

Upward and downward rotation of the scapula are defined by upward and downward movement of the glenoid fossa, respectively. However, the inferior angle of the scapula may also be used as the referent, with upward and downward rotation described as movement of the inferior angle away from the vertebral column (upward rotation) or movement of the inferior angle toward the vertebral column (downward rotation). Some also refer to upward/downward rotation of the scapula (regardless of reference point on the scapula) as *abduction/adduction* or *lateral/medial rotation,* respectively.[1,63]

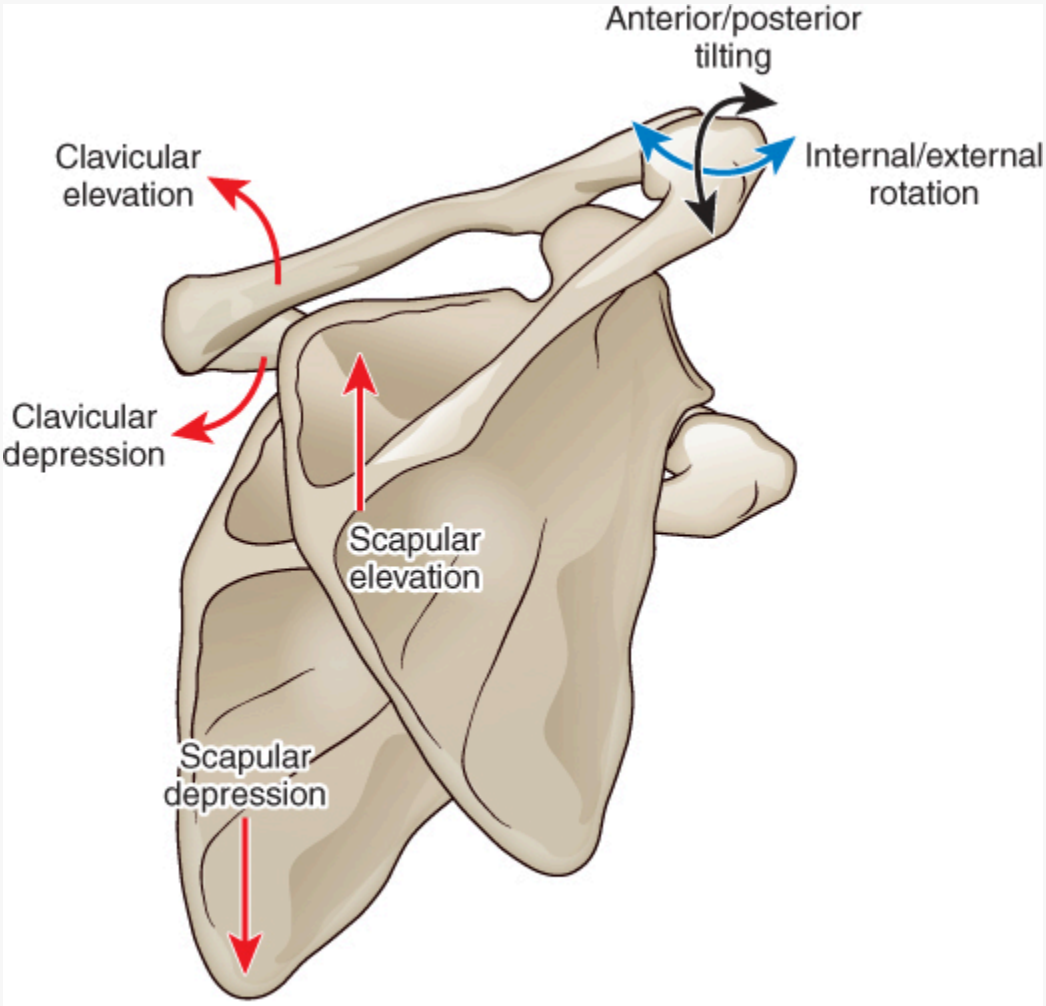
Elevation and Depression

Scapulothoracic elevation and depression can be performed by shrugging the shoulder up and depressing the shoulder downward, respectively. Elevation and depression of the scapula on the thorax are sometimes described as translatory motions in which the scapula moves upward (cephalad) or downward (caudal) along the rib cage from its resting position. Scapular elevation, however, actually occurs as elevation of the clavicle around an A-P axis at the sternoclavicular joint and may include subtle

adjustments in anterior/posterior tilting and internal/external rotation at the acromioclavicular joint to keep the scapula in contact with the thorax, (Fig. 7–30).

FIGURE 7-30

Elevation and depression of the scapula are produced by elevation/depression of the clavicle at the sternoclavicular joint and by rotations (anterior/posterior tilting and internal/external rotation) at the acromioclavicular joint.



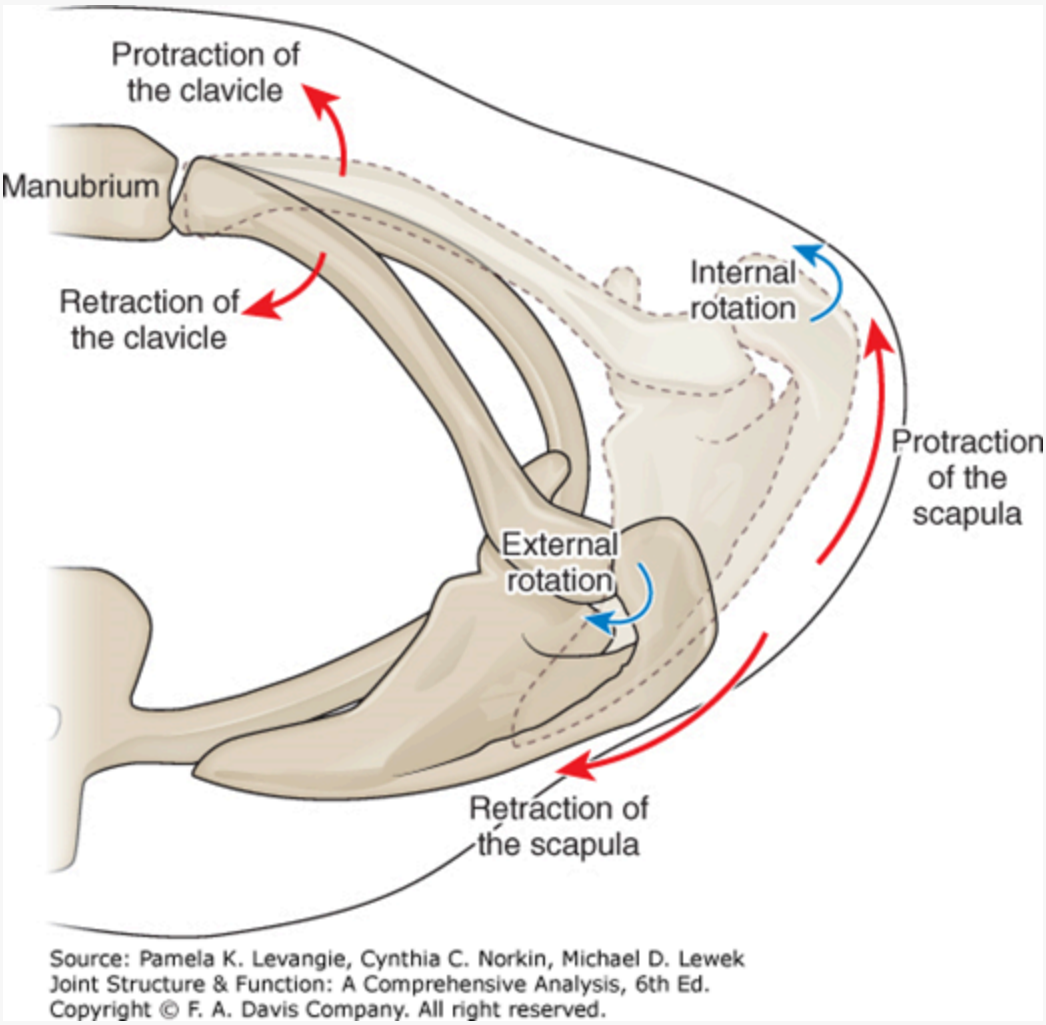
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Protraction and Retraction

Protraction and retraction of the scapula on the thorax are often described as translatory motions of the scapula away from and toward the vertebral column, respectively. These theoretical translatory motions have also been termed *scapular abduction* and *adduction.* However, if protraction or abduction of the scapula on the thorax occurred as a pure translatory movement, the scapula would move directly away from the vertebral column, and the glenoid fossa would face laterally. Only the vertebral border of the scapula would remain in contact with the rib cage. Full scapular protraction actually results in the glenoid fossa facing anteriorly, with the scapula in contact with the rib cage. The scapula protracts and retracts on the thorax through protraction and retraction of the clavicle at the sternoclavicular joint (vertical axis) and follows the contour of the ribs by rotating internally and externally at the acromioclavicular joint (Fig. 7–31).

FIGURE 7-31

Protraction and retraction of the scapula are produced by protraction/retraction of the clavicle at the sternoclavicular joint and by rotations (especially internal/external) at the acromioclavicular joint.



Internal and External Rotation

The scapulothoracic motions of internal and external rotation are less obvious on physical observation but are critical to movement of the scapula along the curved rib cage. Internal/external rotation of the scapula on the thorax should normally accompany protraction/retraction of the clavicle at the sternoclavicular joint (see Fig. 7–31). Approximately 15° to 16° of internal rotation occurs at the acromioclavicular joint during normal elevation of the arm.[16,58] A larger amount of internal rotation of the scapula on the thorax that is isolated to (or occurs excessively at) the acromioclavicular

joint results in prominence of the vertebral border of the scapula as a result of loss of contact with the thorax. This is often referred to clinically as **scapular winging** (Fig. 7–32). Excessive internal rotation may be indicative of pathology or poor neuromuscular control of the scapulothoracic muscles (particularly the **serratus anterior muscle**).

FIGURE 7-32

Scapular winging. Excessive internal rotation of the scapulae at the acromioclavicular joint causes prominence of the medial border of the scapula with attempted protraction of the scapula (or elevation of the arms).



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Anterior and Posterior Tilting

As is true for internal/external rotation, scapulothoracic anterior/posterior tilting is normally not very obvious on clinical observation and yet is critical to maintaining contact of the scapula against the curvature of the rib cage. Anterior/posterior tilting of the scapula on the thorax occurs predominately at the acromioclavicular joint around an oblique coronal axis that lies in the plane of the scapula.[16] Because of the differing sternoclavicular and acromioclavicular axis alignment described earlier (see Fig. 7–29), scapulothoracic anterior/posterior tilting and elevation/depression of the clavicle at the sternoclavicular joint can also couple.[62] If the clavicle was perpendicular to the axis for acromioclavicular internal rotation (see Fig 7–29B), clavicle elevation would couple with or result in anterior tilting of the scapula on the thorax, and vice versa. Given the approximately 60° typical internal rotation angle of the acromioclavicular joint (see Fig. 7–29C), two-thirds of the magnitude of clavicle elevation will couple or result in anterior tilting of the scapula on the thorax. Anterior tilting beyond resting values will result in prominence of the inferior angle of the scapula (**Fig. 7–33**). An anteriorly tilted scapula may occur with poor neuromuscular control or abnormal posture. Scapulothoracic posterior tilting can occur directly through posterior tilting at the acromioclavicular joint, or be coupled with posterior rotation of the clavicle at the sternoclavicular joint. If the clavicle were aligned parallel to the plane of the scapula at a 0° acromioclavicular internal rotation angle (see Fig. 7–29A), clavicle posterior rotation would couple with posterior tilting of the scapula on the thorax. Given the approximately 60° typical internal rotation angle of the acromioclavicular joint, only one-third of the magnitude of clavicle posterior rotation will couple with posterior tilting of the scapula on the thorax.

FIGURE 7-33

This scapula is anteriorly tilted with attempted flexion of the arm, causing the inferior angle of the scapula to lift off the thorax and become visually prominent. This condition typically occurs if the anterior deltoid and coracobrachialis are inadequately opposed by the serratus anterior, also referred to as “reverse action” as the deltoid and coracobrachialis move their unstabilized proximal attachments.



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Scapulothoracic Stability

Stability of the scapula on the thorax is provided by the structures that maintain integrity of the linked acromioclavicular and sternoclavicular joints. The muscles that attach to both the thorax and scapula maintain contact between these surfaces while

producing the movements of the scapula. In addition, stabilization is provided by the scapulothoracic musculature, which pulls or compresses the scapula to the thorax.[63]

The ultimate functions of scapular motion are to orient the glenoid fossa for optimal contact with the humeral head of the moving arm, to add range to elevation of the arm, and to provide a stable base for the controlled motions between the humeral head and glenoid fossa. The scapula, with its associated muscles and linkages, performs these mobility and stability functions so well in healthy shoulder function that it serves as a premier example of dynamic stabilization in the human body.



Patient Application 7–2

Anterior-Lateral Shoulder Pain

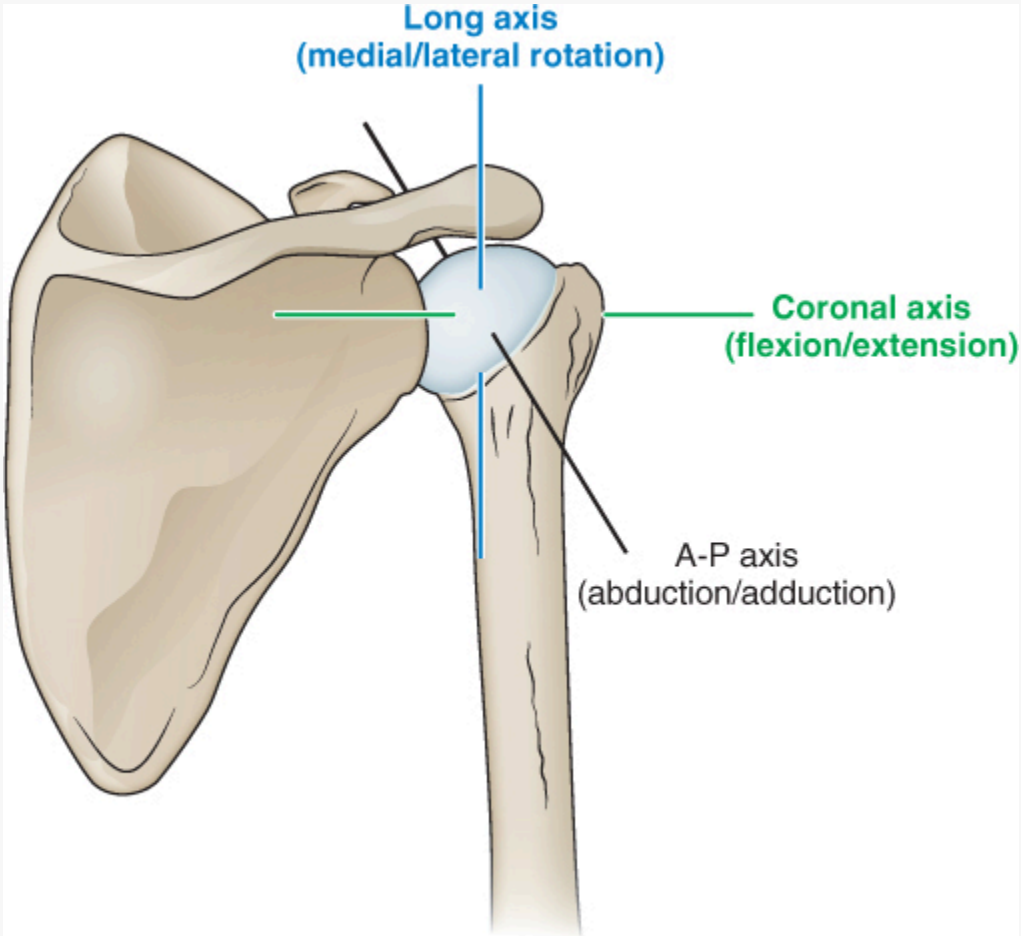
Delmar Fagin is a 57-year-old dentist reporting shoulder pain localized at the proximal lateral humerus. This location is consistent with pain originating from the rotator cuff tendons, the long head of the biceps tendon, or the subacromial bursa. His pain may be related to a rotator cuff or biceps tendinopathy and possible shoulder subacromial mechanical impingement due to repetitive work at low angles of humeral elevation. Repetitive mechanical impingement can create tendinopathy and progress to partial and full thickness rotator cuff tears.[47] In addition, he reports pain when sleeping on his right side. This is a common complaint of persons with pain originating from the subacromial space because additional compression of the subacromial tissues is experienced while lying on the affected side.

Glenohumeral Kinematics

The glenohumeral joint is usually described as having three rotational degrees of freedom: **flexion/extension, abduction/adduction,** and **medial/lateral rotation** (Fig. 7–34). The range of each of these motions occurring solely at the glenohumeral joint varies considerably.

FIGURE 7-34

Glenohumeral rotations. Flexion/extension occur around a coronal axis; abduction/adduction occur around an A-P axis; and medial/lateral rotation occur around a long axis.



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Flexion and Extension

Flexion and extension occur about a coronal axis passing through the center of the humeral head. The glenohumeral joint is often considered to have 120° of flexion and about 50° of extension.[64] However, more recent work measuring 3D glenohumeral

motion reported peak humeral flexion of only 97° in relation to the scapula in a small sample of subjects averaging 50 years of age.[65] This lesser value of glenohumeral motion is also supported by studies in young healthy subjects.[16] The higher values classically attributed to glenohumeral flexion may not have fully isolated glenohumeral motion from trunk and scapular motion.

Medial and Lateral Rotation

Medial and lateral rotation occur about a long axis parallel to the shaft of the humerus and passing though the center of the humeral head. The range of medial/lateral rotation of the humerus varies with position. With the arm at the side, medial and lateral rotation may be limited. Abducting the humerus to 90° frees the arc of rotation, with glenohumeral values of 130° or greater.[64] The restricted arc of medial/lateral rotation when the arm is at the side may be related to different alignment of the greater and lesser tubercles, which creates a mechanical block, or to different areas of capsular or muscular tightness when the arm is adducted versus abducted.

Abduction and Adduction

Abduction/adduction of the glenohumeral joint occurs around an A-P axis passing through the humeral head center. The maximum range of abduction at the glenohumeral joint is the topic of much disagreement. There is general consensus, however, that the range of abduction of the humerus in the frontal plane (whether the abduction is active or passive) will be diminished if the humerus is maintained in neutral or medial rotation. The restriction to abduction in medial or neutral rotation is commonly attributed to impingement of the greater tubercle on the coracoacromial arch. When the humerus is laterally rotated 35° to 40°, the greater tubercle will pass

under or behind the arch so that abduction can continue.[66,67]

The ROMs for abduction of the glenohumeral joint (if impact of the greater tubercle is avoided) are reported to be anywhere from 90° to 120°.[1,11,65,68,69] Inman and coworkers found active abduction to be limited to 90° when the scapula does not participate in the motion, but claim that 120° of motion was available passively.[54] Further increasing the variability between investigations, some studies examined the range of abduction in the traditional frontal plane, whereas others have investigated elevation in the plane of the scapula (30° to 45° anterior to the frontal plane). The available passive range for abduction in the scapular plane may be slightly greater than for abduction in the frontal plane. When the humerus is elevated in the plane of the scapula (referred to as *abduction in the plane of the scapula, scapular abduction,* or **scaption** in clinical jargon), there is presumably less restriction to motion because the capsule is less twisted than when the humerus is brought farther back into the frontal plane. An, however, found maximum elevation 10° to 37° anterior to the plane of the scapula rather than in that plane.[66] Although it has been proposed that abduction in the scapular plane does not require concomitant lateral rotation to achieve maximal range, this premise has also been disputed.[66,67] During active arm elevations in all planes of elevation, glenohumeral lateral rotation has been demonstrated.[16]

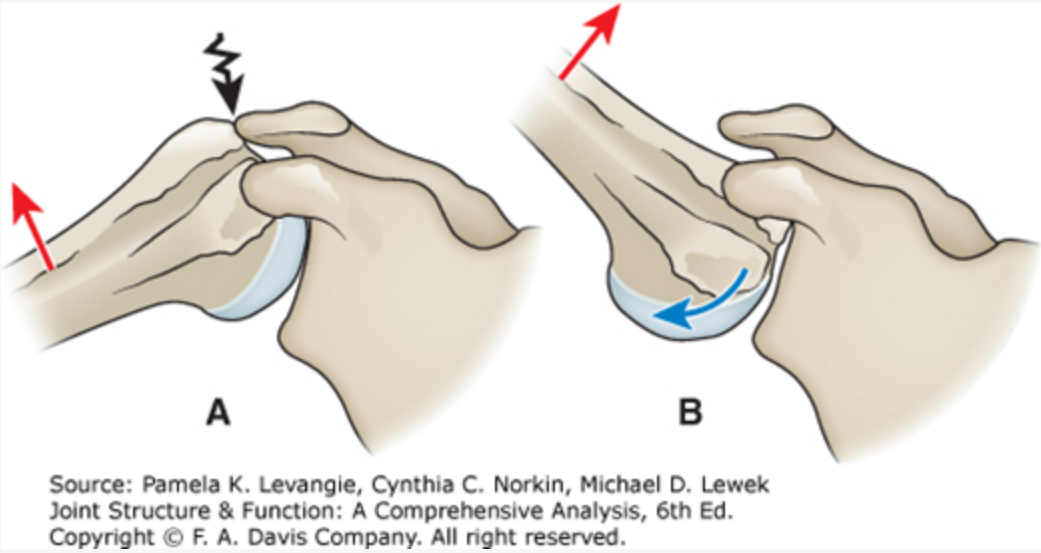
Intra-Articular Glenohumeral Motion

Full ROM of the glenohumeral joint is, to a reasonable degree, a function of the intra-articular movement of the incongruent articular surfaces. The convex humeral head is a substantially larger surface and may have a different radius of curvature than the shallow concave fossa. Given this incongruence, rotations of the joint around its three

axes do not occur as pure spin but have changing centers of rotation and shifting contact patterns within the joint. There is a somewhat surprising lack of consensus on the extent and direction of movement of the humeral head on the fossa.[27] However, elevation of the humerus requires that the articular surface of the humeral head slide inferiorly (caudally) in a direction opposite to movement of the shaft of the humerus. Failure of the humeral articular surface to slide downward in abduction of the humerus would cause superior (cephalad) rolling of the humeral head surface on the fossa (Fig. 7–35). The large humeral head would soon run out of glenoid surface, and the head of the humerus would impinge upon the overhanging coracoacromial arch (Fig. 7–35A). If, as it should, the articular surface of the head of the humerus slides inferiorly while the head rolls up the fossa, full ROM can be achieved (Fig. 7–35B).

FIGURE 7-35

A. Without downward sliding of the articular surface of the humeral head, the humeral head will roll up the glenoid fossa and impinge upon the coracoacromial arch. **B.** With downward sliding of the humeral head's articular surface as the humeral abducts, a full range of motion can occur.



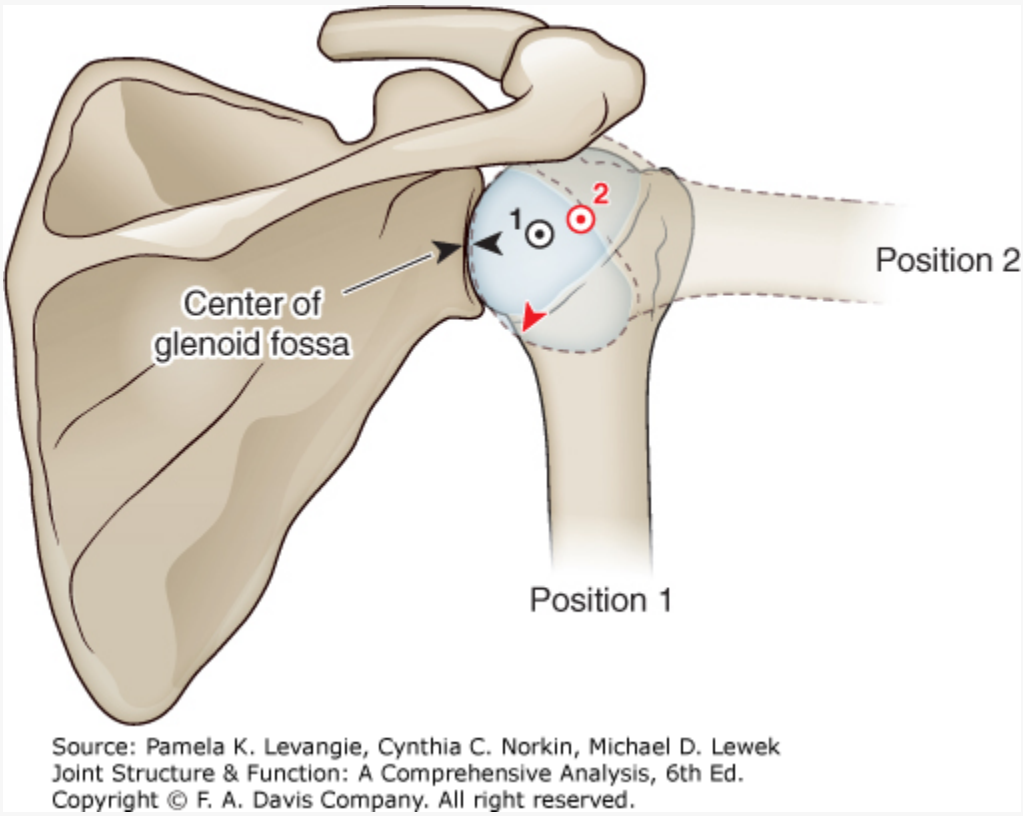
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There is consensus that inferior sliding of the humeral head's articular surface is necessary to minimize upward rolling; of the humeral head. However, the humeral head as a whole (referencing its center of rotation) is often reported to still move somewhat superiorly (translate upwardly) on the glenoid fossa in spite of the downward sliding (Fig. 7–36), although the magnitude of superior versus inferior humeral head center translation differs among investigators.[27,51,48,67,70,71] The humeral articular surface may also slide anteriorly or posteriorly and medially or laterally on the glenoid fossa. The humeral head's center is believed to move slightly superior (1–2 mm of translation) until about 60° of active elevation motion.[21,48,70,71] With further elevation, the humeral head's center is believed to remain relatively stable and centered on the glenoid fossa.

FIGURE 7-36

Slight superior translation of the center of the humeral head (from position 1 to position 2) may still occur during humeral abduction

despite inferior sliding of the head's articular surface.



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Less agreement exists regarding the anterior and posterior translations of the humeral head's center. Slight anterior positioning and translation (1–2 mm) has been reported during active elevation, including in abduction and scapular plane abduction.[51,70] Other investigators have reported slight anterior or posterior translations early in the range and opposite translations later in the range of active elevation, including during flexion.[51,48,71,72] All studies of active translations show smaller magnitudes of slide (less than 5 mm) during active motions than is available in a passive laxity examination, in which translations up to 20 mm have been reported.[73] These data support the premise that rotator cuff forces help to stabilize and center the humeral head on the glenoid fossa during active motion. Most investigators also agree that many variables determine

the patterns of movement of the humeral head on the glenoid fossa, including articular geometry, capsuloligamentous influences, influences of arm position, and muscle forces.

Much of the confusion surrounding reported motions of the humeral head on the glenoid fossa may be attributed to the differences in the point on the humerus that is being followed (combinations of rolling and sliding vs. translations), as well as to the small magnitudes of these motions and the limitations of currently available measurement systems.

 Expanded Concepts 7–4

The Role of the Capsule

The glenohumeral capsule and its associated glenohumeral ligaments provide stability to the glenohumeral joint by limiting anterior, inferior, or posterior humeral head translation on the glenoid fossa. The stabilizing function of these structures is minimal at less than 90° of humeral elevation when only the superior segment of the capsule is under any significant tension. At these lower elevation angles, the rotator cuff muscles and tendons actively stabilize the glenohumeral joint based on their orientation across the joint. Toward the end range of humeral motions, however, the capsule becomes tight passively, and this tension restricts glenohumeral translation. At end ranges, this tension begins to produce rather than restrict humeral head center translations.[74] With asymmetrical tightening of the capsule, this obligate translation occurs in a direction away from the tight and toward the looser capsular tissue.[74] For example, with increasing medial rotation, the posterior capsule becomes tight and produces anterior translation of the humeral head center that is not restricted by the relatively slack anterior capsule. A tight posterior capsule is, therefore, one potential mechanism for shoulder mechanical impingement, because it may produce increased anterior humeral head translation and minimize the subacromial space.[48]

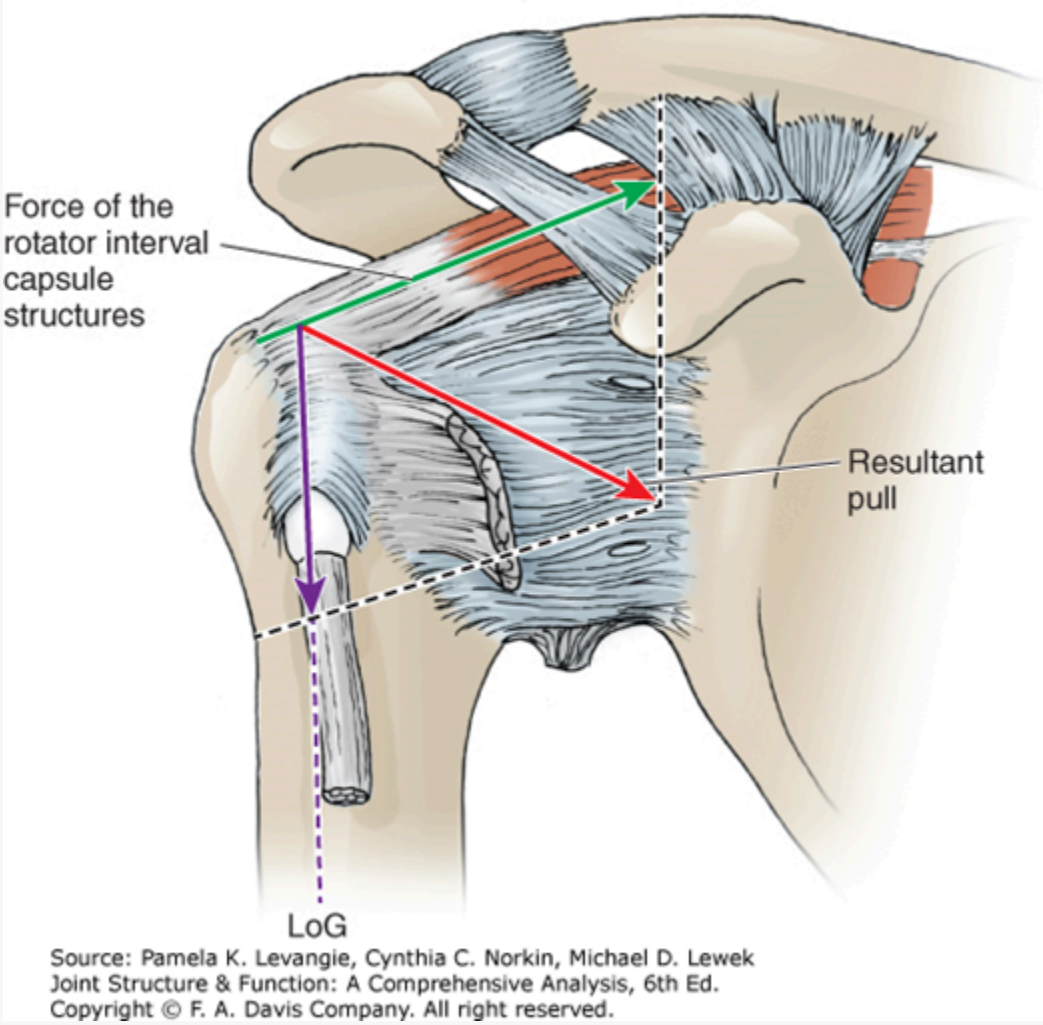
Static Stabilization of the Glenohumeral Joint in the Dependent Arm

Given the incongruence of the glenohumeral articular surfaces, bony geometry alone cannot maintain joint stability with the arm relaxed at the side, requiring the contribution of other mechanisms. With the humeral head resting on the fossa, gravity imparts a caudally directed translatory force on the humerus. To maintain equilibrium, a cranially directed force is needed and could be supplied by active contraction or passive tension of muscles, such as the deltoid, supraspinatus, or the long heads of the biceps brachii and **triceps brachii.** However, Basmajian and Bazant[19] and MacConaill and Basmajian[30] reported that all muscles of the shoulder complex are electrically silent in the relaxed, unloaded limb, even when the limb is tugged vigorously downward. The mechanism of joint stabilization, therefore, appears to be passive. The structures of the rotator interval capsule (superior capsule, superior glenohumeral ligament, and coracohumeral ligament) that are taut when the arm is at the side possess both the magnitude and orientation for this stabilization function.[32,34,36] The resultant vector formed from the gravity and rotator interval capsule vectors creates a force that compresses the humeral head into the lower portion of the glenoid fossa and prevents inferior humeral head translation (Fig. 7–37).

FIGURE 7-37

Mechanism for stabilization of the dependent arm. With the arm relaxed at the side, the downward pull of gravity on the arm (vector extended upward from the center of gravity of the upper extremity) is opposed by the passive tension in the rotator interval capsule. The resultant of these forces stabilizes the humeral head on the glenoid

fossa.



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In addition to passive tension from the rotator interval capsule, two other mechanisms help provide static stability to the glenohumeral joint with the arm at the side. In a healthy glenohumeral joint, the capsule is airtight and there is negative intra-articular pressure. This negative pressure creates a relative vacuum that resists the inferior humeral translation caused by the force of gravity.[34] Loss of intra-articular pressure, produced by venting the capsule or tears in the glenoid labrum, results in large increases in inferior humeral translations.[75,76] The degree of glenoid inclination also

influences glenohumeral joint stability with the arm in the dependent position.[37] A slight upward tilt of the glenoid fossa, either anatomically in the structure of the scapula or through scapular upward rotation, will produce a partial bony block against humeral inferior translation.

When passive forces are inadequate for glenohumeral joint stabilization, as may occur in the heavily loaded arm, the supraspinatus is recruited to provide active assistance.[19] This is not surprising, given that the supraspinatus tendon has attachments to the rotator interval capsule.[40] In fact, the role of the supraspinatus may be more critical than its activity as measured by electromyography (EMG) indicates because paralysis or dysfunction in the supraspinatus may lead to gradual inferior subluxation of the glenohumeral joint. Without the reinforcing passive tension of the intact supraspinatus muscle, the sustained load on the connective tissue structures of the rotator interval capsule apparently causes these structures to gradually stretch (become plastic), which results in a loss of joint stability. Inferior glenohumeral subluxation is commonly encountered in patients with diminished rotator cuff function caused by stroke or other brain injuries.

Dynamic Stabilization of the Glenohumeral Joint

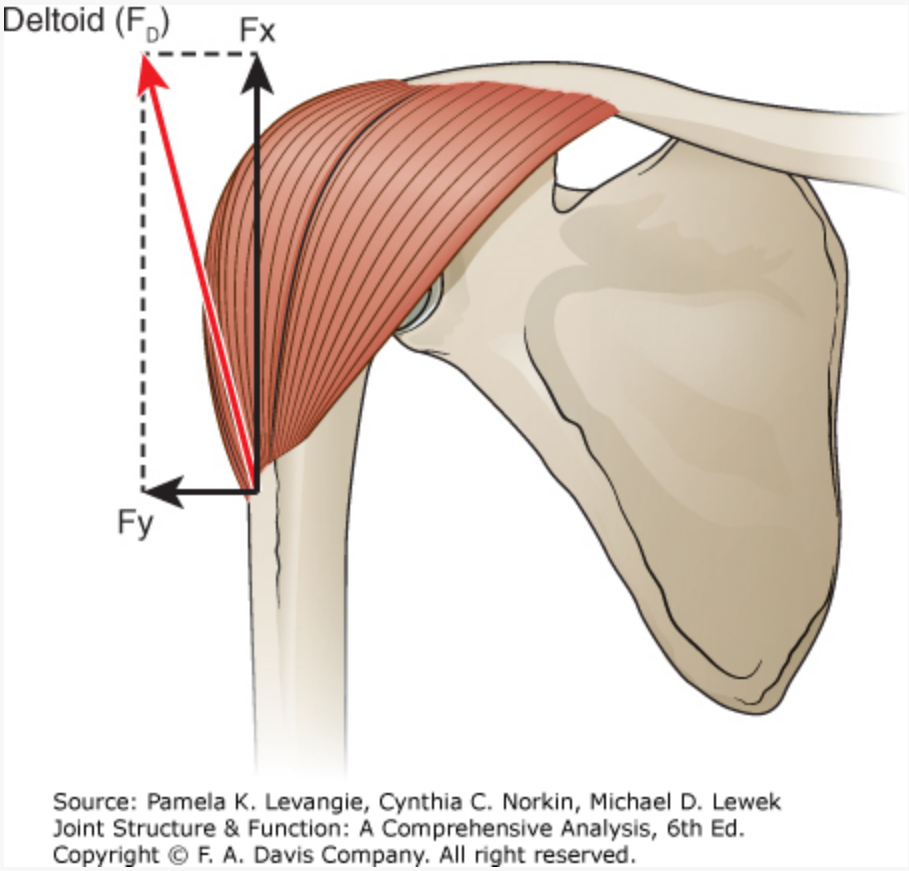
The Deltoid and Glenohumeral Stabilization

It is generally accepted that the deltoid muscle is a prime mover (along with the supraspinatus) for glenohumeral abduction. The anterior deltoid is also considered the prime mover in glenohumeral flexion. Both abduction and flexion are elevation activities with many biomechanical similarities. The segment or segments of the deltoid that participate in elevation will vary with role and function.[77,78] However, examination

of the resultant line of action of the deltoid muscle in abduction can be used to highlight the stabilization needs of the glenohumeral joint in elevation activities. Figure 7–38 shows the line of action of the deltoid muscle with the arm at the side. The force vectors of the three segments of the deltoid acting together coincide with the fibers of the middle deltoid. When the muscle force vector (F_D) is resolved into its parallel (Fx) and perpendicular (Fy) components in relation to the long axis of the humerus, the parallel component directed cephalad (superiorly) is by far the larger of the two components. That is, the majority of the force of contraction of the deltoid from this initial position causes the humerus and humeral head to translate superiorly; because of the line of action of the deltoid with the arm at the side, only a small moment arm in this position directly contributes to rotation (abduction) of the humerus.

FIGURE 7-38

The action line of all three segments of the deltoid follows the line of pull of the middle deltoid. The resultant pull of the three deltoid segments (F_D) resolves into a very large translatory component (Fx) and a small rotary component (Fy) so that an isolated contraction of the deltoid would cause the deltoid to produce more superior translation than rotation of the humerus.



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At many other joints, a force component parallel to the long bone (generally the larger component) has a stabilizing effect because the parallel component contributes to joint compression. However, when the arm is at the side, the glenoid fossa is not in line with the shaft of the humerus. Consequently, a force (Fx) applied parallel to the long axis of the bone in this position creates a shear force (approximately parallel to the contacting articular surfaces) rather than a stabilizing (compressive) effect. The large superiorly directed force of the deltoid, if unopposed, would cause the humeral head to impact the coracoacromial arch before much abduction had occurred. The rotary torque produced by the relatively small moment arm (and Fy component) of the deltoid will not be particularly effective until the translatory forces are in equilibrium. If the

humeral head migrated upward into the coracoacromial arch, the inferiorly directed contact force of the arch would offset the Fx component of the deltoid, theoretically permitting rotation of the humeral head to continue. However, pain from impinged structures in the subacromial space is likely to prevent much motion. The inferior pull of gravity (see Fig. 7–37) cannot offset the much larger superiorly directed Fx component of the deltoid; thus, the deltoid cannot independently abduct (elevate) the arm. Another force or set of forces must be introduced to work synergistically with the deltoid for the deltoid to work effectively to produce the desired rotation (elevation). This is one role of the rotator **(musculotendinous)** cuff.

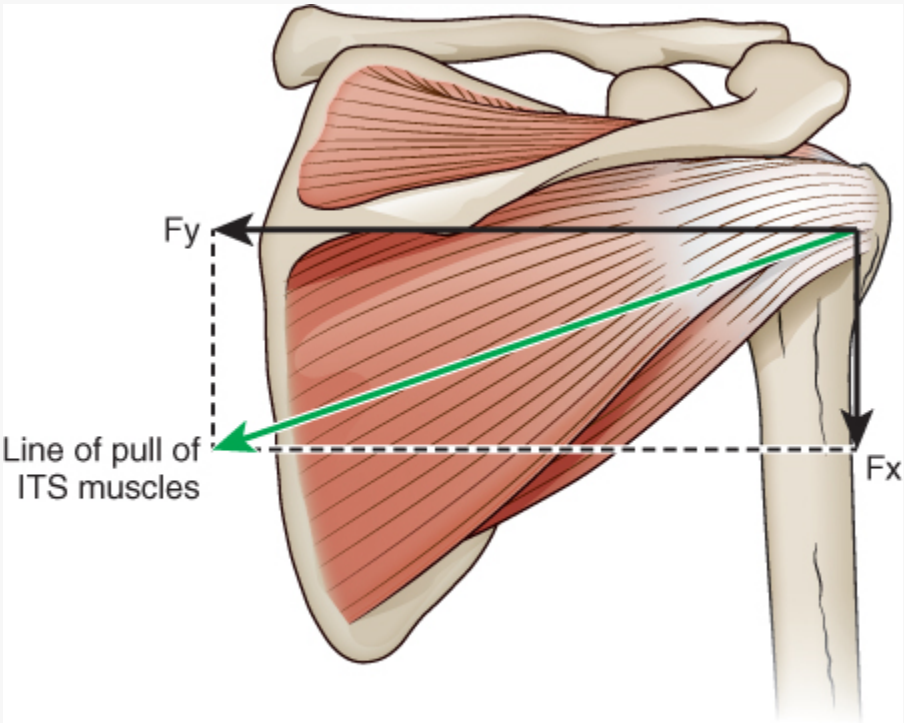
The Rotator Cuff and Glenohumeral Stabilization

The supraspinatus, **infraspinatus, teres minor,** and subscapularis muscles and tendons compose the rotator cuff. These muscles are considered to be part of a "cuff" because the inserting tendons of each muscle of the cuff blend with and reinforce the glenohumeral capsule both anteriorly (subscapularis) and posteriorly (supraspinatus, infraspinatus, and teres minor). Each of the rotator cuff muscles has lines of action that significantly contribute to the dynamic stabilization of the glenohumeral joint.

The force of the infraspinatus, teres minor, or subscapularis muscles taken together (as well as individually) resolves into its components such that the perpendicular force component (Fy) not only tends to cause at least some rotation of the humerus given its orientation to the long axis of the bone, but it also compresses the head into the glenoid fossa (Fig. 7–39). This is because the articular surface of the humerus lies nearly perpendicular to the shaft and provides a clear illustration of how muscle forces also produce translatory forces and do more than "rotate" a bone around a joint axis.

FIGURE 7-39

The combined action line of the infraspinatus, teres minor, and the anteriorly located subscapularis (ITS) muscles of the rotator cuff has a large Fy component that compresses the humeral head and a downwardly directed Fx component.



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Although the infraspinatus, teres minor, and subscapularis muscles of the rotator cuff are important glenohumeral joint compressors, equally (or perhaps even more) critical to the stabilizing function of these muscles is the inferior (caudal) translatory pull (Fx) of the muscles. The sum of the three downward (inferior) translatory components of these muscles of the rotator cuff nearly offsets the superior translatory force of the deltoid muscle. Sharkey and Marder showed that abduction without the infraspinatus, teres

minor, and subscapularis muscles resulted in substantial superiorly directed shifts in humeral position on the glenoid fossa in cadaver models.[79]

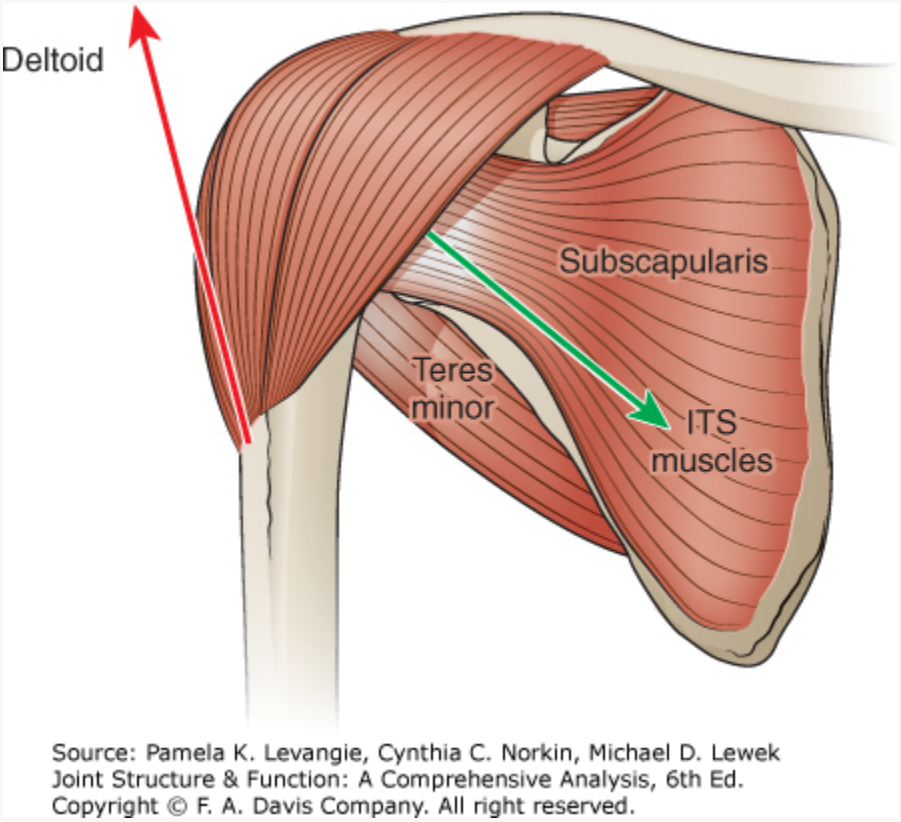
The teres minor and infraspinatus muscles, in addition to their stabilizing role, contribute to abduction of the arm by providing the lateral rotation that typically occurs with elevation of the humerus to help clear the greater tubercle from beneath the acromion. Although the weak adduction force of the teres minor muscle and the medial rotary force of the subscapularis muscle appear to contradict their role in elevation of the arm, Otis and colleagues found the effectiveness of these muscles in their contradictory functions to be diminished during abduction of the arm.[80] That is, the infraspinatus and subscapularis muscles add to the abduction torque, whereas the teres minor muscle adds to the lateral rotary torque. The medial and lateral rotary forces also help center the humeral head in an anterior/posterior direction, with increased anterior and posterior displacements evident when rotator cuff forces are reduced.[81] Saha referred to these cuff muscles as "steerers." [82]

The action of the deltoid and the combined actions of the infraspinatus, teres minor, and subscapularis muscles approximate a force couple (**Fig. 7–40**). The nearly equal and opposite superior/inferior forces for the deltoid and these three rotator cuff muscles acting on the humerus approximate an *almost* perfect rotation of the humeral head around a relatively stable axis of rotation with minimal translation.

FIGURE 7-40

The line of pull of the deltoid muscle and the combined pulls of the

posteriorly located infraspinatus and teres minor, along with the anteriorly located subscapularis (ITS muscles), approximate a force couple with nearly parallel lines of pull that produce almost perfect rotation of the humeral head around a stable AP axis with minimal translation.



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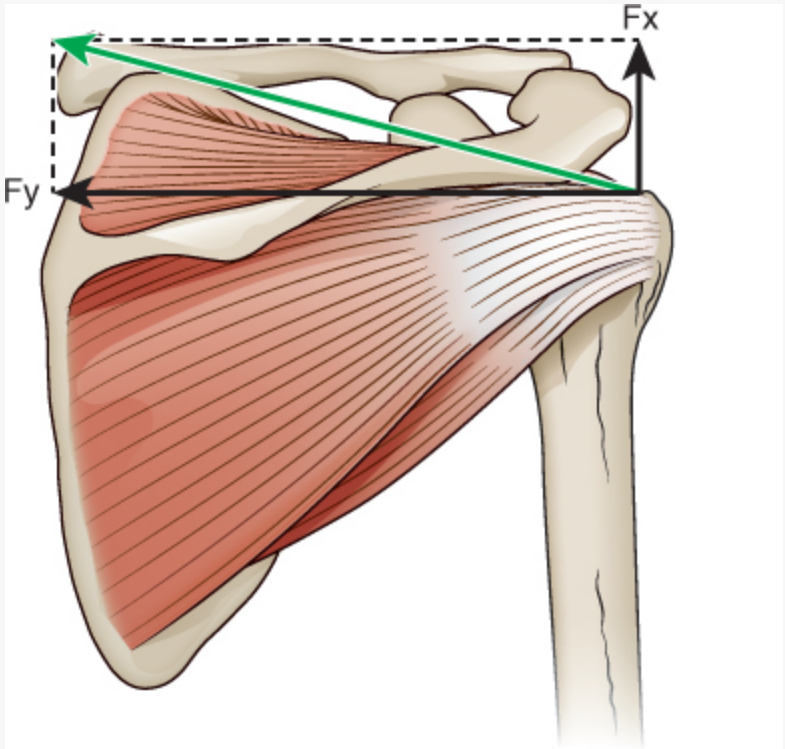
The Supraspinatus and Glenohumeral Stabilization

Although the supraspinatus muscle is part of the rotator cuff, the line of action of the supraspinatus muscle, unlike the action lines of the other three rotator cuff muscles, has a superior (cephalad) translatory component (Fig. 7–41), rather than the inferior (caudal) component found in the other muscles of the cuff (see Fig. 7–39). Given its line of pull, the supraspinatus cannot contribute to offsetting the upward dislocating action

of the deltoid.[83] The supraspinatus is still an effective stabilizer of the glenohumeral joint, however, because of its contribution to joint compression. Because the more superior location of the supraspinatus results in a line of action that lies farther from the glenohumeral joint axis than the action lines of the other rotator cuff muscles, the larger supraspinatus moment arm is capable of independently producing a full or nearly full range of glenohumeral joint abduction while simultaneously stabilizing the joint.[30] Gravity acts as a stabilizing synergist to the supraspinatus by offsetting the small upward translatory pull of the muscle.

FIGURE 7-41

The supraspinatus, like the other muscles of the rotator cuff, has a large Fx component that compresses the head of the humerus but, unlike the other muscles of the rotator cuff, has a *superiorly* (rather than inferiorly) directed Fx component. The Fy component of the supraspinatus lies far enough from the joint axis that it can independently abduct the humerus.



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Expanded Concepts 7–5

The Supraspinatus as an Independent Abductor

The supraspinatus can, at least theoretically, independently produce abduction of the arm through most or all of its range, whereas the deltoid cannot. The resultant of the force vectors of a supraspinatus contraction and gravity is essentially a vector identical to the resultant vector that we saw in Figure 7–35. The line of action of the supraspinatus is the same as that of the rotator interval capsule with which it blends (and to which it contributes passive tension even when the muscle is at rest). With a concentric contraction of the supraspinatus, the proportionally small superiorly directed translatory force is offset by the inferiorly directed force of gravity, which results in translatory equilibrium but effective rotation. The resultant of the gravitational and supraspinatus force vectors contributes to an inferior gliding of the humeral head articular surface during abduction of the arm, allowing full articulation of the joint surfaces and preventing abnormal superior displacement. The supraspinatus can also contribute small amounts of either medial or lateral rotation

torque, depending on the position of the arm, although its moment arm for long axis rotation is very small.[84]

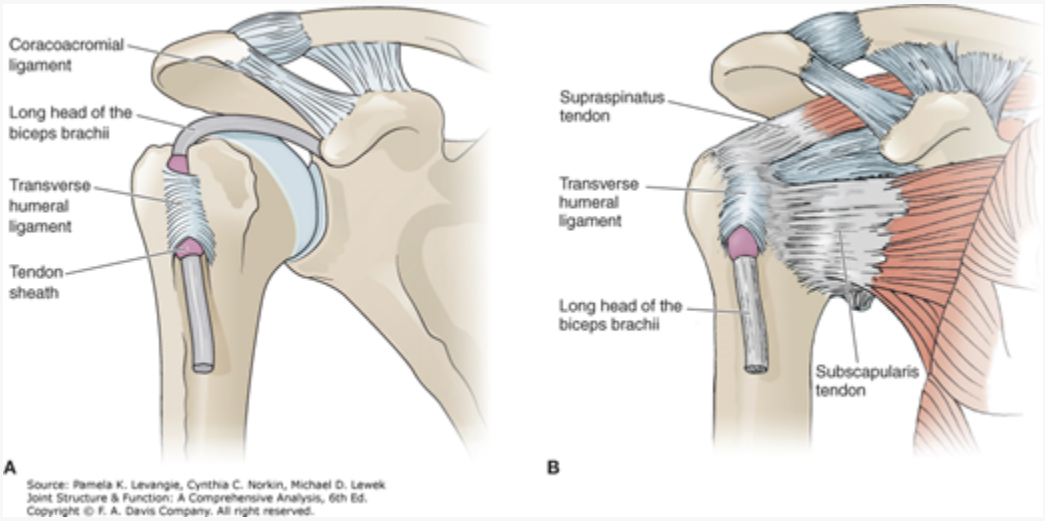
The Long Head of the Biceps Brachii and Glenohumeral Stabilization

The long head of the biceps brachii runs superiorly from the anterior shaft of the humerus through the bicipital groove between the greater and lesser tubercles to attach to the supraglenoid tubercle and superior labrum (Fig. 7–42). Within the bicipital groove, the biceps tendon is enveloped by a tendon sheath and tethered there by the **transverse humeral ligament** that runs between the greater and lesser tubercles (Fig. 7–42A). The long head tendon enters the glenohumeral joint capsule through an opening between the supraspinatus and subscapularis tendons (Fig. 7–42B) where it penetrates the capsule but not the synovium. The long head of the biceps brachii, because of its position at the superior capsule and its connections to structures of the rotator interval capsule, is sometimes considered part of the reinforcing cuff of the glenohumeral joint.[40] The biceps muscle is capable of contributing to the force of flexion and can, if the humerus is laterally rotated, contribute to the force of abduction.[54] Although elbow and shoulder position may influence its function, the long head appears to contribute to glenohumeral stabilization by centering the head in the fossa and by reducing vertical (superior and inferior) and anterior translations.[85–88] Pagnani and colleagues hypothesized that the long head may produce its effect by tightening the relatively loose superior labrum and transmitting increased tension to the superior and middle glenohumeral ligaments.[88] This idea follows from their observation that lesions of the anterosuperior labrum did not affect stability of the glenohumeral joint unless the attachment of the long head of the biceps brachii was also disrupted.[89] The overall contribution of the long head to glenohumeral

stabilization is supported by the observation that the tendon sometimes hypertrophies with rotator cuff tears.[88] However, others contend the stabilization role of the biceps long head is not critical, and it is sometimes surgically cut to reduce shoulder pain.

FIGURE 7-42

The long head of the biceps brachii. **A.** The tendon passes through a fibro-osseous tunnel formed by the bicipital groove and the transverse humeral ligament, protected by a tendon sheath. **B.** The tendon enters the glenohumeral joint capsule (but not its synovial lining) through an opening between the supraspinatus and subscapularis tendons.



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Foundational Concepts 7–2

Dynamic Stabilization

Given what we know about the glenohumeral joint thus far, we can summarize that dynamic stabilization at any point in the glenohumeral range is a function of (1) the force of the prime mover or movers; (2) the force of gravity; (3) the force of the

muscular stabilizers; (4) articular surface geometry; and (5) passive capsuloligamentous forces. Inman and colleagues also add (6) the force of friction and (7) the joint reaction force, because any shear force within the glenohumeral joint creates some friction across its joint surfaces and because all forces that compress the head into the glenoid fossa must be opposed by an equal force from the glenoid fossa in the opposite direction (joint reaction force).[54] Due to muscular compression forces, joint reaction forces can reach magnitudes of 9 to 10 times the weight of the upper extremity as the arm is elevated.[26,54] When the medially directed resultant muscular forces have slightly superior or inferior components, shear forces will occur. The greatest shear forces during humeral elevation typically occur between 30° and 60° of elevation.[26]

Costs of Dynamic Stabilization of the Glenohumeral Joint

When all stabilization forces and factors are intact and properly functioning, the head of the humerus rotates into flexion or abduction around a relatively stable axis with minimal translation. Over time, however, even normal stresses resulting from the complex dynamic stabilization process may lead to degenerative changes or dysfunction at the glenohumeral joint. Any disruption in the synergistic action of the dynamic stabilization factors may accelerate degenerative changes in or around the joint.

The supraspinatus muscle is a particularly key structure in dynamic stabilization. The supraspinatus is either passively stretched or actively contracting when the arm is at the side (depending on load); it also participates in humeral elevation throughout the ROM. Consequently, the tendon is under tension most of a person's waking hours and is vulnerable to tensile overload and chronic overuse. Mechanical compression and impingement of the stressed supraspinatus tendon may occur on either the superior or inferior surface for a variety of reasons. Compression may be increased when the subacromial space is reduced by osteoligamentous factors, when there is increased

superior or anterior translation of the humeral head center with less favorable glenohumeral mechanics, when the scapula does not posteriorly tilt or upwardly rotate adequately during humeral elevation, or when occupational factors require heavy lifting or sustained overhead arm postures. Mechanical "impingement" of the undersurface of the supraspinatus can also occur relative to the glenoid at higher angles of humeral elevation.[90] The supraspinatus tendon is the most vulnerable of the rotator cuff muscles. However, the overuse and potential impingement issues also apply to the other cuff muscles. Symptomatic and asymptomatic partial or full thickness rotator cuff tears are seen in many people over the age of 60, with the supraspinatus likely to show lesions before the other tendons of the cuff.[91] Rotator cuff tendinopathy or tears typically produce pain between 60° and 120° of humeral elevation in relation to the trunk. This range constitutes what has been described as the **painful arc.** It is within this ROM that the tendons of the rotator cuff were previously described as passing beneath the coracoacromial arch in a cadaveric investigation.[43] However, as noted earlier, more recent investigation of active motion in human subjects has revealed that beyond 60° of scapulohumeral abduction, the supraspinatus tendon has rotated past the overlying acromion in most cases.[44] Subsequently, the source of the pain in a painful arc of motion may be related to other factors than supraspinatus compression.



Patient Application 7–3

Glenohumeral Joint Instability

Megan Schultz is a 17-year-old who reports symptoms of pain, weakness, and a feeling of "giving out" during some reaching activities. She is unable to lie on her shoulder when sleeping because of this same "giving out" sensation. The position of 90° abduction and 90° external rotation is uncomfortable for her. These symptoms are consistent with loss of static stabilization of the GH joint, which is often related to

hypermobility or laxity of the anterior capsule and glenohumeral ligaments. In such cases, the dynamic stabilizers—primarily the rotator cuff—must be relied on to support the joint and maintain the humeral head position on the glenoid. These patients often note increased symptoms at the end of the day or after repetitive use of the upper extremity, presumably from fatigue of the dynamic stabilizers.

Integrated Function of the Shoulder Complex

Integrated Joint Function

The shoulder complex acts in a coordinated manner to provide the smoothest and greatest ROM possible to the upper limb. Motion available to the glenohumeral joint alone would not account for the full range of elevation (abduction or flexion) available to the humerus. The remainder of the range is contributed by the scapula on the thorax through the sternoclavicular and acromioclavicular joints. Combined **scapulohumeral motion** (1) distributes the motion between the joints, permitting a large ROM with less compromise of stability than would occur if the same range occurred at one joint; (2) maintains the glenoid fossa in an optimal position in relation to the head of the humerus, increasing joint congruency while decreasing shear forces; and (3) permits muscles acting on the humerus to maintain a good length-tension relationship while minimizing or preventing active insufficiency of the glenohumeral muscles.

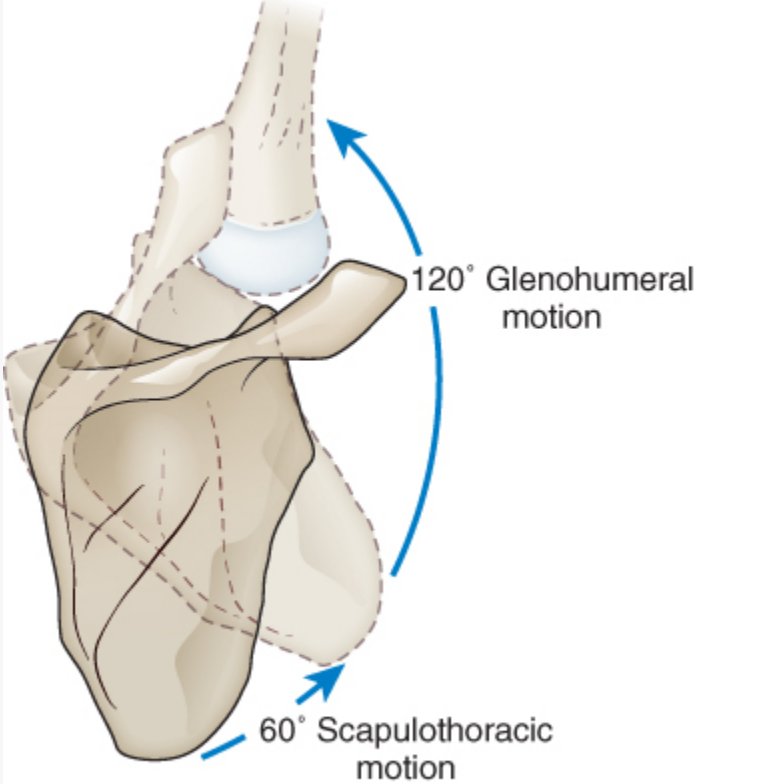
Scapulothoracic and Glenohumeral Function

The scapula on the thorax contributes to elevation (flexion and abduction) of the humerus by upwardly rotating the glenoid fossa 50° to 60° from its resting position.[16,55] If the humerus were immobile at the glenohumeral joint, the scapula alone would theoretically result in up to 60° of elevation of the humerus relative to the thorax. The humerus, of course, is not immobile but can move independently on the glenoid fossa.

The glenohumeral joint contributes 100° to 120° of flexion and 90° to 120° of abduction. The combination of scapular and humeral movement results in a maximum range of elevation of 150° to 180° (**Fig. 7–43**).[65,92] Some of the variability in ranges reported by investigators is due to individual structural variations (especially for the glenohumeral joint). Another factor in variability may be the extent to which trunk contributions were isolated from humeral motions during the measurement. If trunk motions are not included in the measurements, the overall range of elevation for most people is closer to 150° than 180°.[55,65] An *overall* ratio of 2° of glenohumeral to 1° of scapulothoracic motion during arm elevation is commonly described, and this combination of concomitant glenohumeral and scapulothoracic motion is most commonly referred to as **scapulohumeral rhythm.** According to the 2-to-1 ratio framework, flexion or abduction of 90° in relation to the thorax would be accomplished through approximately 60° of glenohumeral and 30° of scapulothoracic motion. It must also be recognized, however, that elevation of the arm is accompanied not only by elevation of the humerus but also by lateral rotation of the humerus in relation to the scapula. During flexion, an average of 51° of lateral rotation has been reported.[16] In addition, elevation of the humerus may occur in a slightly different plane than that of the scapula.[16] When asked to actively elevate in a scapular plane (40° anterior to the frontal plane), subjects on average positioned the humerus 5° anterior to the scapular plane.[16] During flexion, the glenohumeral plane of elevation increased up to 30° anterior to the scapula plane, and in abduction, the glenohumeral plane of elevation was up to 20° posterior to the scapular plane.[16]

FIGURE 7-43

When it is assumed that the total range of elevation is 180°, it is common to attribute 120° of the ROM to the humerus at the glenohumeral joint and 60° to the scapula on the thorax—with the two segments moving *concomitantly* rather than sequentially.



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The 2-to-1 ratio of glenohumeral to scapulothoracic contribution to elevation of the arm is acknowledged to be an oversimplification, with substantial variability in scapular and humeral contributions at different points in the ROM and among individuals. However, the general statement is still a useful conceptualization of a typical motion pattern. The distinction must also be made between elevation of the arm from vertical and elevation

of the arm relative to the trunk. The trunk may laterally flex or extend to gain additional range for the arm. However, we will consistently refer to elevation of the arm *in relation to the trunk* unless otherwise stated. When the trunk does *not* participate in the motion, the degree of elevation of the arm from vertical and elevation of the arm relative to the trunk will be the same.



Expanded Concepts 7–6

Variations in Scapulohumeral "Rhythm"

Inman and coworkers reported that during the initial 60° of flexion or the initial 30° of abduction of the humerus, an inconsistent amount and type of scapular motion occurred in relation to glenohumeral motion.[54] The scapula has been described as seeking a position of stability in relation to the humerus during this period (setting phase).[54,78,93] In this early phase, motion occurs primarily at the glenohumeral joint, although stressing the arm may increase the scapular contribution.[69] A number of studies have investigated the scapulohumeral "rhythm," with ratios reported varying between 1.25:1 and 2.69:1.[21,68,69,82,93,94] Ratios are often described as nonlinear, indicating changing ratios during different portions of the ROM for elevation of the arm. The rhythm varies among individuals and may vary with external constraints.[94] Synchronous upward rotation of the scapula with glenohumeral elevation is certainly an important concept. However, the utility of the term *rhythm* seems limited because there does not appear to be a definitive scapulohumeral "rhythm."

The scapula contributes to elevation of the arm not only by upwardly rotating on the thorax but also through other scapulothoracic motions. The exact magnitudes of different scapular motions vary across studies. The general patterns of scapular motions, however, are relatively consistent. As the arm is elevated into flexion, scapular plane abduction (scaption) or abduction in the frontal plane, the scapula posteriorly tilts on the thorax.[16-18,55,63] As the arm moves from the side to 150° of elevation, the magnitude of posterior tilting is about 20° to 30°.[16,55] This posterior tilting allows the

inferior angle of the scapula to move anteriorly and stay in contact with the thorax as it slides upward and around the rib cage. Posterior tilting of the scapula also has the effect of bringing the anterior acromion up and back. This may serve to minimize reduction in the subacromial space as the humerus elevates.

During elevation, internal/external rotation of the scapula is more variable within and between subjects.[16,17,55] In general, the scapula is externally rotating on the thorax at the end ranges of elevation.[16,17,55,63] However, slight internal rotation of the scapula on the thorax occurs early in the ROM for flexion. Prevention of excess internal rotation of the scapula is important for keeping the scapula, particularly the medial border, in contact with the thorax as the scapula upwardly rotates during arm elevation. In flexion, the scapula initially protracts and internally rotates to orient the glenoid fossa anteriorly (in the sagittal plane).[16,55] Structural limitations or the inability of muscles to appropriately stabilize the scapula may result in anterior tilting, internal rotation, and downward rotation of the scapula with attempted flexion of the arm, through reverse action of the deltoid (**Fig. 7–44**).

FIGURE 7-44

Structural limitations or the inability of muscles to appropriately stabilize the scapula may result in anterior tilting, internal rotation, and downward rotation of the scapula during elevation of the arm, lifting the inferior and medial angles of the scapula off the thorax.



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Sternoclavicular and Acromioclavicular Function

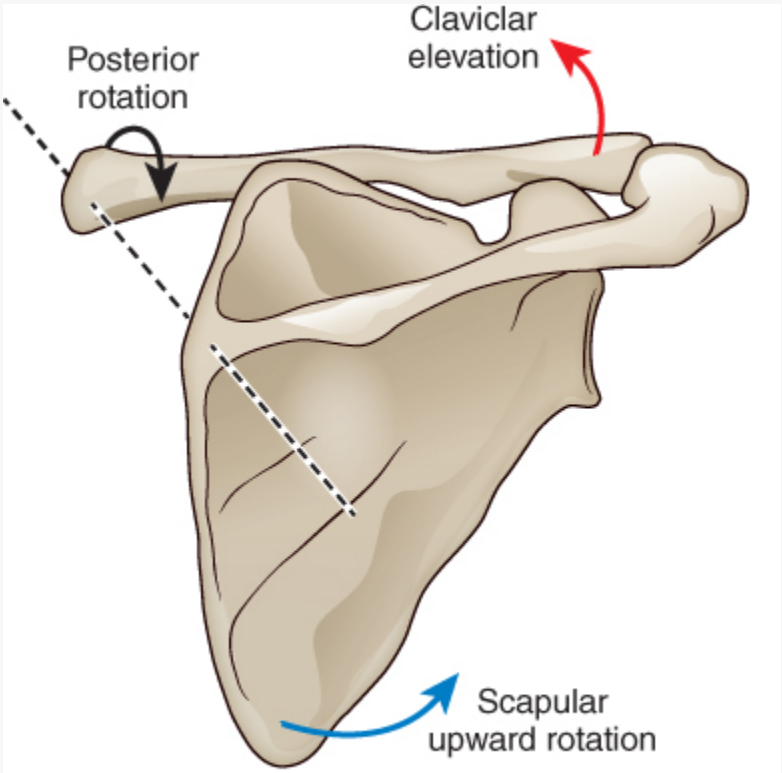
Elevation of the arm in any plane involves motion of the sternoclavicular and acromioclavicular joints to produce the necessary scapulothoracic motion. Given the complexity of the linkages, there is limited description in the literature on the relative contributions of the sternoclavicular and acromioclavicular joints to the potential 60°

arc of upward rotation of the scapula on the thorax as the scapula moves through its full ROM. However, recent investigations report consistent average values for sternoclavicular and acromioclavicular joint motions during active elevation despite varying measurement methods.[16,55,58]

The initiation of scapulothoracic upward rotation as the arm is flexed or abducted appears to couple with clavicular posterior rotation and elevation at the sternoclavicular joint, based on the non-parallel claviculoscapular axis relationships previously described (see Fig. 7–29).[78] This scapular upward rotation occurs around an oblique AP axis, passing through the costoclavicular ligament (sternoclavicular joint motion), and projecting backward through the root of the scapular spine (scapulothoracic motion) (**Fig. 7–45**). As elevation of the arm progresses, the scapulothoracic axis of rotation gradually shifts laterally, reaching the acromioclavicular joint in the final range of scapular upward rotation (**Fig. 7–46**).[78] This major shift in the axis of rotation happens because the scapulothoracic motion can occur only through a combination of motions at the sternoclavicular and acromioclavicular joints. When the axis of scapular upward rotation is near the root of the scapular spine, scapulothoracic motion is primarily a function of sternoclavicular joint motion. When the axis of scapular upward rotation is at the acromioclavicular joint, acromioclavicular joint motions predominate; and when the axis of scapular upward rotation is in an intermediate position, both the sternoclavicular and acromioclavicular joints are contributing to scapulothoracic motion.

FIGURE 7-45

With elevation of the arm, the scapulothoracic upward rotation contribution begins as posterior rotation and elevation at the sternoclavicular joint around an axis that appears to pass posteriorly from the costoclavicular ligament to the root of the spine of the scapula.

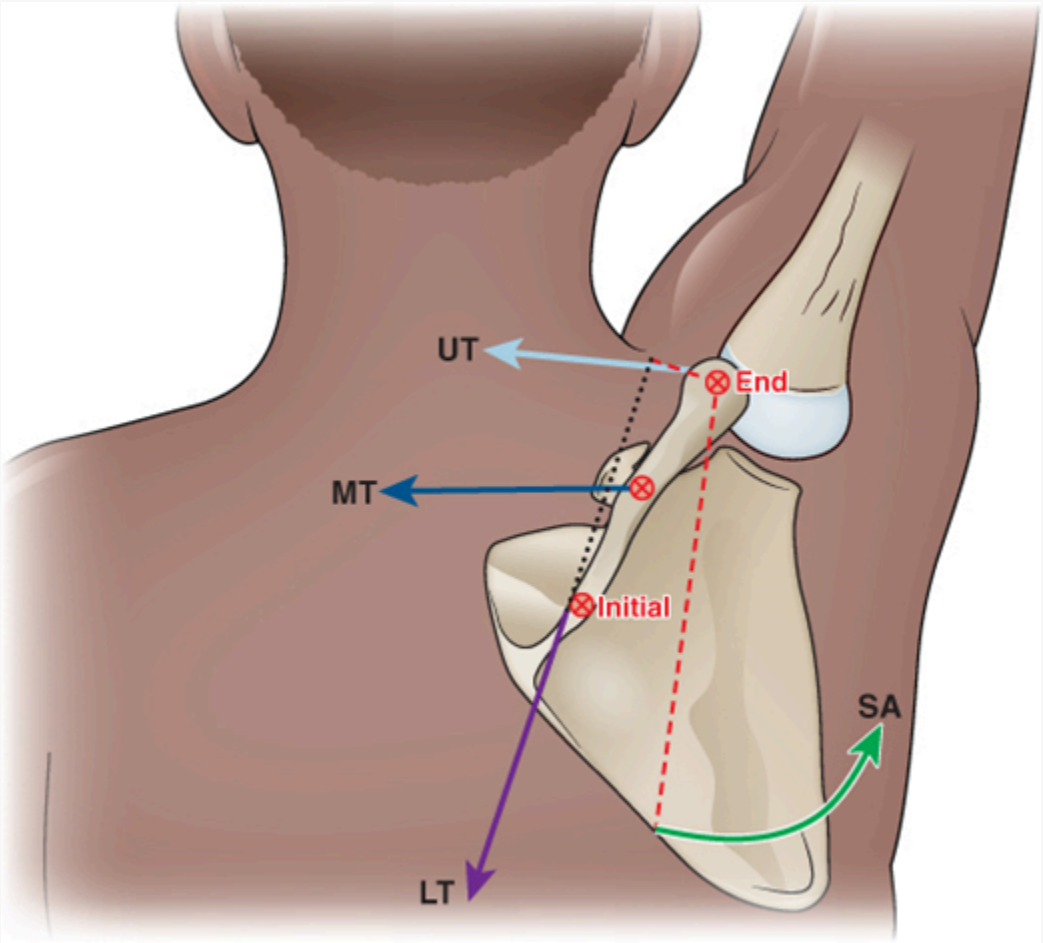


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FIGURE 7-46

The axis of scapular upward rotation (red circle) progresses from its initial position near the root of the scapular spine, laterally to its final location near the acromioclavicular joint at the end of the motion.

With the exception of the upper trapezius (UT) attaching to the clavicle, the action lines of the middle trapezius (MT), lower trapezius (LT), and serratus anterior (SA) muscles combine to produce upward rotation of the scapula on the thorax. The red dashed lines show the moment arms of the lower trapezius (its vector extended by the black dotted line) and serratus anterior with respect to the axis of rotation at the end of the motion, with the moment arm of the serratus being much longer.



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Inman and coworkers in 1944 described the relative contributions of the sternoclavicular and acromioclavicular joints to scapulothoracic upward rotation during arm elevation to be about 50% from sternoclavicular elevation and 50% from acromioclavicular upward rotation (20°–30° each to obtain 50°–60° upward rotation).[54] However, current 3D descriptions of clavicular motion show only about 10° or less of clavicular elevation during arm elevation.[16,58] In addition, as described previously (see Fig. 7–29), only about one-third of this elevation (about 3°) will couple with scapular upward rotation.[62] For upward rotation of the scapula to occur at the acromioclavicular joint (which has been described in 3D studies as 15°–20° of scapular upward rotation at the AC joint), limitation to acromioclavicular motion imposed by the coracoclavicular ligament must be overcome.[16,58] Tension in the coracoclavicular ligament (especially the conoid portion) is produced as the coracoid process of the scapula gets pulled downward with muscle forces attempting to upwardly rotate the scapula at the acromioclavicular joint. The tightened conoid ligament pulls its posteroinferior clavicular attachment forward and down as the coracoid process drops, causing the clavicle to posteriorly rotate. Posterior rotation of the clavicle around its longitudinal axis will result in additional scapulothoracic upward rotation (see Figs. 7–22 and 7–28). The scapulothoracic upward rotation occurs both as the lateral end of the S-shaped clavicle flips up, coupling about two-thirds of this posterior sternoclavicular joint rotation to scapulothoracic upward rotation, and as the tension in the coracoclavicular ligament is reduced, now permitting upward rotation to occur at the acromioclavicular joint.[62] The magnitude of posterior rotation of the clavicle may be anywhere from 30° to 50°, contributing 20° or more to scapulothoracic upward rotation.[16,54] Subsequently, sternoclavicular and acromioclavicular joint rotations work cumulatively to contribute

to overall scapular upward rotation on the thorax during arm elevation.

Throughout elevation of the arm (flexion, scapular plane abduction, or frontal plane abduction), the clavicle also retracts at the sternoclavicular joint, typically 20° to 30°, which couples with external rotation of the scapula on the thorax.[16,55,58] Because of the approximately vertical orientation of the sternoclavicular and scapulothoracic axes for protraction, sternoclavicular retraction and protraction will directly result or couple with scapulothoracic external and internal rotation, respectively. However, in functional arm elevation movements, at the same time the sternoclavicular joint is retracting, the acromioclavicular joint is internally rotating 10° to 15°.[16,55] The magnitude of this internal rotation is slightly greater in flexion and less in frontal plane abduction.[16] Acromioclavicular internal/external rotation occurs about an approximately vertical axis as well, parallel to the scapulothoracic internal/external rotation axis. As a result, the acromioclavicular joint internal rotation offsets a large portion of the scapulothoracic external rotation that would otherwise occur from sternoclavicular joint retraction during arm elevation.

The magnitudes of acromioclavicular joint internal rotation with elevation can be expected to differ with variations in scapular resting position, rib cage configuration, and muscle dynamics. Although the ranges vary somewhat, the component motions of the sternoclavicular (retraction) and acromioclavicular (internal rotation) joints are similar regardless of whether the arm elevation motion is performed in the sagittal, scapular, or frontal planes. In other words, with arm elevation in each of these planes, it is expected that the sternoclavicular joint will always retract, and the acromioclavicular joint will always internally rotate in a healthy shoulder motion.[16]

Besides differences in the magnitudes of the component motions of the sternoclavicular and acromioclavicular joints, the other difference between the performances of sagittal plane and frontal plane elevation is based on the joints' initial positions. The clavicle and scapula begin flexion in less retraction and more internal rotation, respectively, to bring the glenoid fossa forward, keeping the fossa in line with the elevating humeral head.[16] The clavicle and scapula begin abduction in more retraction and less internal rotation, respectively, to bring the glenoid fossa more lateral, again obtaining improved alignment with the humeral head during the motion.

The final component motion of the scapula on the thorax occurring during arm elevation in all planes is posterior tilting. Although about one-third of sternoclavicular posterior rotation of the clavicle will couple with scapulothoracic posterior tilting, two-thirds of sternoclavicular elevation (about 6°) will couple with scapulothoracic anterior tilting during the same amount of arm elevation.[62] As a reminder, this is because the acromioclavicular joint internal rotation angle is about two-thirds of the way to a 90° alignment between the clavicular long axis and the scapular plane. In a 90° alignment, clavicular elevation at the acromioclavicular joint would produce scapulothoracic anterior tilting. As a result, the rotations of the sternoclavicular joint offset one another with regard to scapulothoracic tilting, and minimal scapulothoracic posterior tilting is produced at the sternoclavicular joint during normal arm elevation. The predominance of scapulothoracic posterior tilting during elevation of the arm occurs with acromioclavicular joint posterior tilting, which averages 20° or more.[16,58]

 Expanded Concepts 7–7
Thoracic Spine Motion

In addition to the motions that occur at the shoulder complex joints during arm elevation, there is also concurrent motion of the thoracic spine. With unilateral arm elevation, the lower thoracic spine side-bends contralaterally, whereas the upper thoracic spine undergoes contralateral axial rotation.[95] During bilateral arm elevation there is extension of both the lower and upper regions.[95] Mobility of the thoracic spine region should also be considered when assessing shoulder complex function, as decreased or increased thoracic motion has the potential to influence shoulder kinematics.

Scapula Motion and Muscle Function

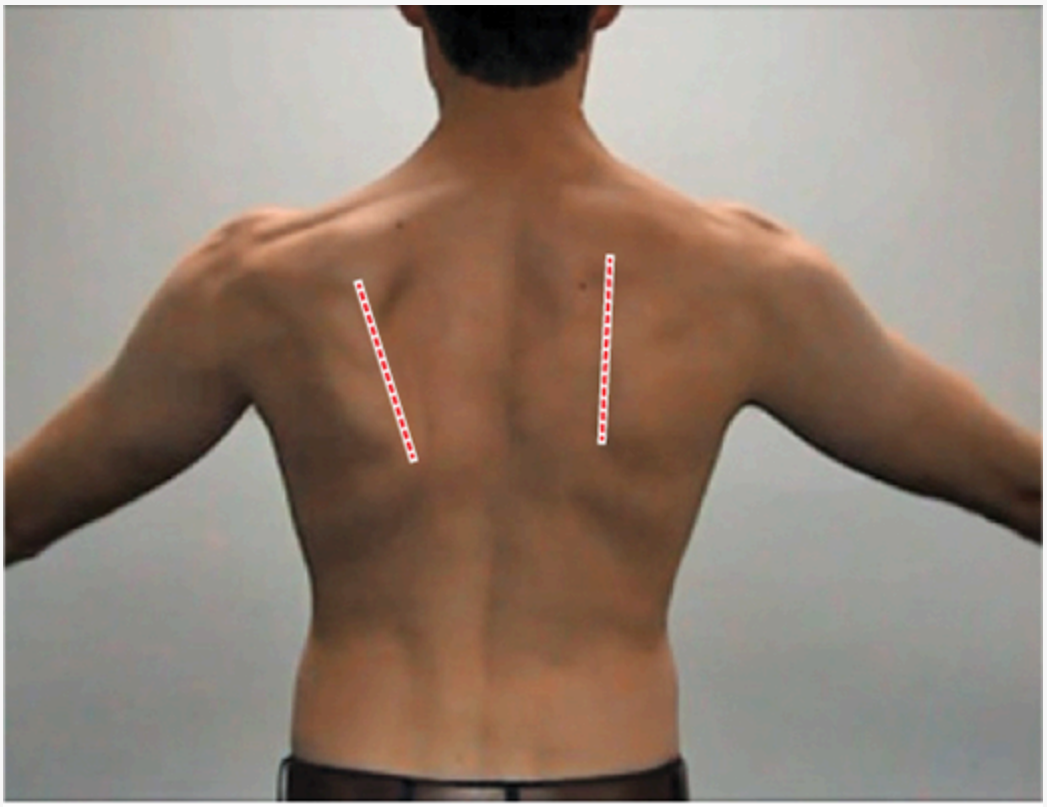
There is agreement that the motions of the scapula are primarily produced by a balance of the forces between the trapezius and serratus anterior muscles through their attachments on the clavicle and the scapula. The upper portion of the trapezius muscle is attached to the clavicle and is positioned to contribute directly to the initial elevation of the clavicle as well as to the sternoclavicular joint retraction that occurs during normal arm elevation.[96] The serratus anterior muscle makes its contribution to the combined clavicular and scapular motion through its action on the scapula. When the location of the axis for scapulothoracic upward rotation is at or approaching its final position near the acromioclavicular joint, the lower trapezius may contribute to upward rotation, although the lower serratus anterior muscle has a much greater moment arm (see Fig. 7–46).

There are substantial implications for muscle function during scapulothoracic upward rotation due to the large shift in the scapulothoracic (combined sternoclavicular and acromioclavicular) axis of rotation. The torque capabilities of the lower trapezius change across the range of elevation as the axis of rotation shifts.[96] The serratus anterior muscle maintains a large moment arm for scapulothoracic upward rotation throughout the entire range of elevation.

Despite lines of action and moment arms that are not as effective for producing scapulothoracic upward rotation, the trapezius is clearly active during arm elevation and plays a role in the balance of forces that move and control the scapula on the thorax.[54] If the serratus anterior muscle acted in isolation, the lateral line of action would result in substantial lateral translation and less effective upward rotation of the scapula as is seen with accessory nerve damage affecting the trapezius (**Fig. 7–47**). The medial translatory action of the trapezius is necessary to offset the lateral translatory action of the serratus and results in more effective upward rotation by the serratus.

FIGURE 7-47

Individual with left spinal accessory nerve palsy, with partial paralysis of trapezius, particularly middle and lower trapezius. Note visible atrophy of all aspects of trapezius as well as reduced scapular upward rotation and increased lateral translation of the root of the scapular spine on the involved side during abduction. The unopposed action of the serratus anterior results in excessive lateral translation and, consequently, less effective scapular upward rotation.



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Foundational Concepts 7–3

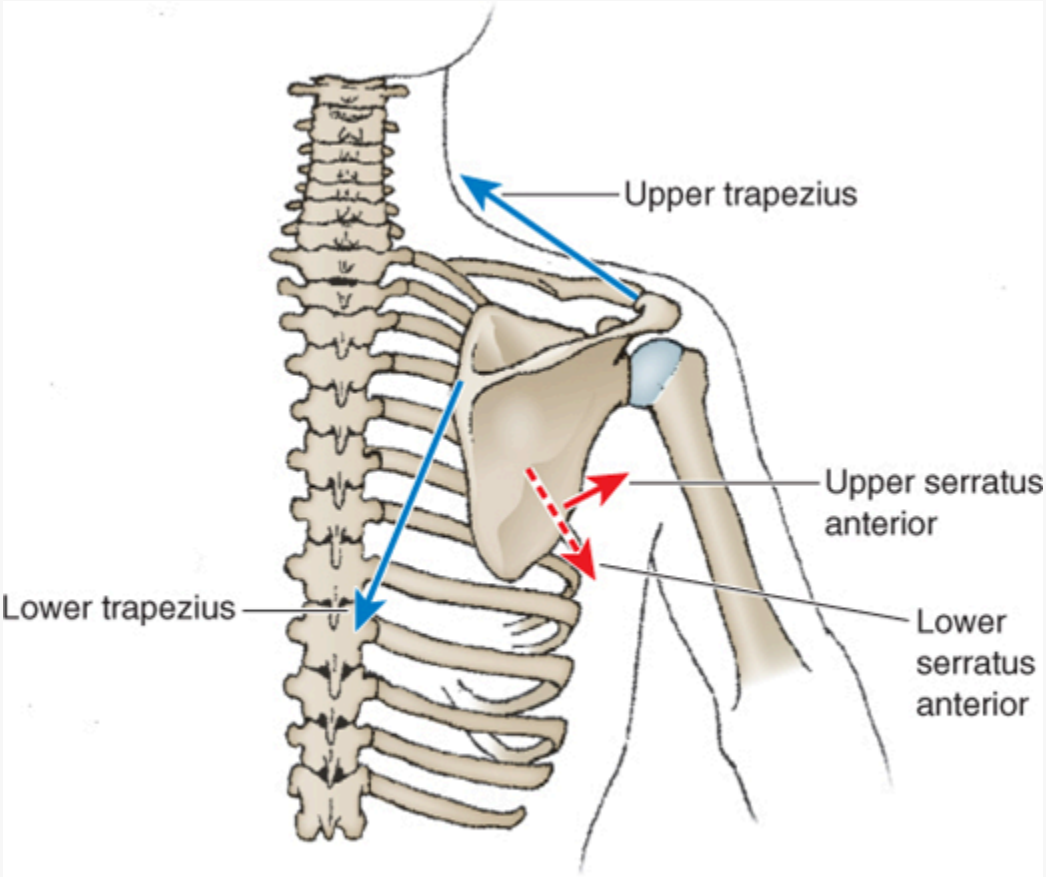
Trapezius Function Reconsidered

Johnson and colleagues identified how the lines of action for the trapezius muscle were based on actual muscle fiber orientation that differed from the traditional descriptions and depictions of muscle line of action in older textbooks.[96] Historically, the upper trapezius has been shown with a superior and medial line of action on the scapula and the lower trapezius with an inferior medial line of action (**Fig. 7–48**). Johnson and colleagues suggested, on the basis of cadaveric dissection, that the upper trapezius fibers act minimally on the scapula and more through elevation and retraction of the clavicle, thus only minimally contributing directly to upward rotation of the scapula on the thorax.[96] Johnson and colleagues also suggested, on the basis of actual fiber orientation and cross-sectional area, that the lower trapezius action line should be directed more medially and less inferiorly than traditionally

depicted.[96] Consequently, this muscle (see Fig. 7–46) would have a lesser moment arm to contribute to scapular upward rotation on the thorax than has been classically described. These descriptions are consistent with Dvir and Berme's model of shoulder function, which identifies the lower serratus anterior muscle as the prime mover of the scapula and the trapezius as the prime stabilizer.[78]

FIGURE 7-48

Classically described action lines for the upward rotators of the scapula.



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The contribution of scapulothoracic muscles to producing and controlling other scapulothoracic motions (anterior/posterior tilting and internal/external rotation) is not

well described in the literature. The middle and lower serratus anterior muscles, with their insertions into the inferior angle and medial border of the scapula, play a primary role in stabilizing the scapula against the thorax. Although the function of the serratus anterior muscle is traditionally considered to be the production of scapular protraction, its line of action indicates the capability of producing acromioclavicular joint external rotation as it pulls the scapula laterally on the thorax.[97] Protraction produced by the serratus anterior might help to produce sternoclavicular joint protraction as the scapula is pulled around the thorax with the medial border stabilized on the thorax. In fact, paralysis of the serratus anterior muscle classically presents with scapular "winging" (see Fig. 7–32). Scapular winging includes internal rotation and anterior tilting of the scapula, produced by the remaining muscles without the stabilizing external rotation and posterior tilting influence of the middle and lower serratus. The lower serratus anterior muscle also has a large moment arm to produce posterior tilting of the scapula. The middle and lower trapezius can also contribute to external rotation torques of the scapula at the acromioclavicular joint, and the upper trapezius to clavicular retraction torque at the sternoclavicular joint.[96,97]

Integrated Shoulder Muscle Function: Elevation

Elevation and depression have been described as the two primary patterns of shoulder complex function. Elevation activities are those that require muscles to overcome or control the weight of the limb and its load. The completion of normal elevation depends on not only the freedom of movement and integrity of the sternoclavicular, acromioclavicular, and glenohumeral joints, but also the appropriate strength and function of the muscles producing and controlling movement. A closer look at the activity of these muscles should enhance an understanding of normal function, as well

as contribute to an understanding of the deficits seen in pathologic situations.

Deltoid

The deltoid is at resting length (optimal length-tension) when the arm is at the side. When at resting length, the deltoid's angle of pull results in a predominance of superior translatory pull on the humerus with an active contraction (see Fig. 7–38). With an appropriate synergistic inferior pull from the infraspinatus, teres minor, and subscapularis muscles, the rotary component of the deltoid muscle is an effective primary mover for elevation. The anterior deltoid is the prime mover for flexion but also assists with abduction.[80,98] During abduction in the plane of the scapula, the anterior and middle deltoid segments are optimally aligned to produce elevation of the humerus.[25] The line of action of the posterior deltoid has too small a moment arm (and too small a rotary component) to contribute effectively to frontal plane abduction. As such, minimal EMG activity is noted in the posterior deltoid during abduction, and it serves primarily as a joint compressor and in motions such as horizontal abduction and extension.[22,77]

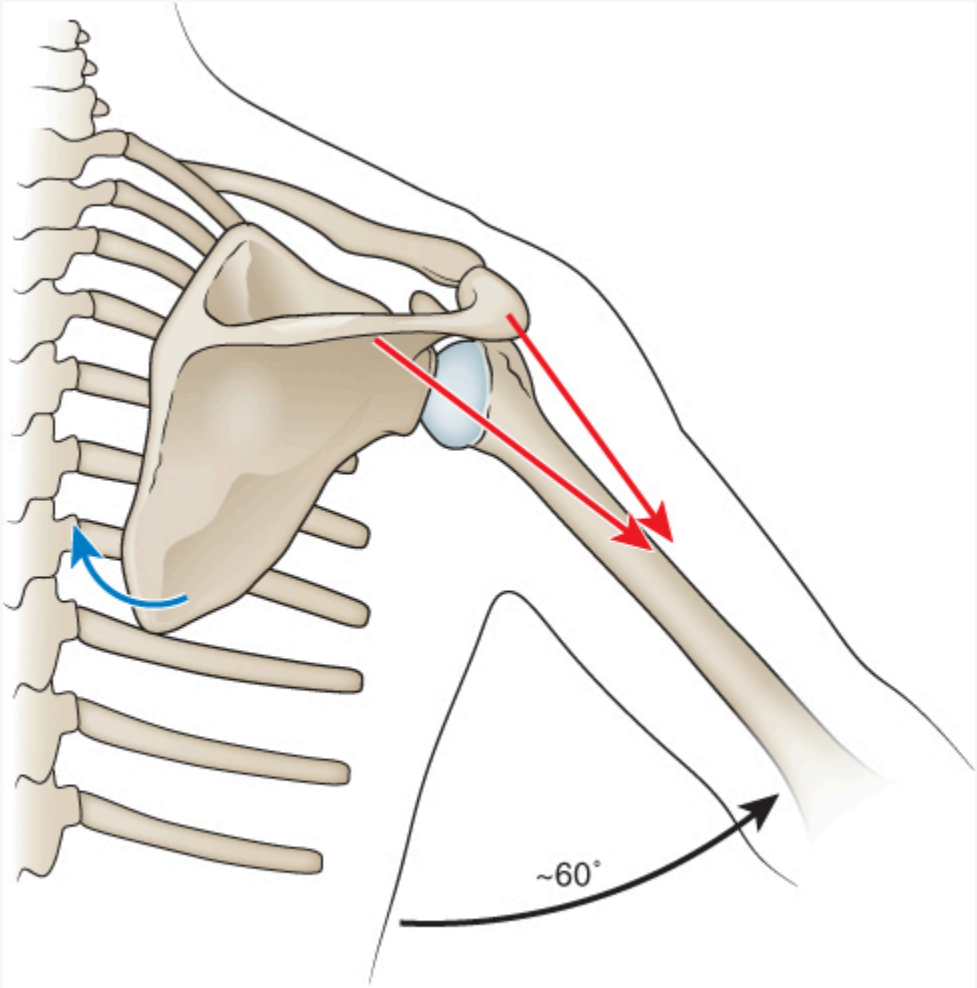
As the humerus elevates, the translatory component of the deltoid diminishes its superior dislocating influence as its resultant force vector shifts increasingly toward the glenoid fossa. At the same time, the rotary component of the deltoid must counteract the increasing torque of gravity as the arm moves toward horizontal. Analysis of EMG shows gradually increasing activity in the deltoid, peaking between 90° to 120° of humeral abduction with a plateau or drop off for the remainder of the motion.[82,98] The shortening deltoid is not able to produce as much active tension, and passive tension is diminished. As a result, a greater number of motor units must be recruited to maintain

force output. The multipennate structure and considerable cross-sectional area of the deltoid help compensate for its relatively small moment arm, low mechanical advantage, and less than optimal length-tension relationship as elevation progresses.

Maintenance of an appropriate length-tension relationship of the deltoid is strongly dependent on simultaneous scapular movement or scapular stabilization. When the scapula is restricted and cannot upwardly rotate, the loss of tension in the deltoid with increased shortening has been reported to result in a reduced range of active glenohumeral abduction (whether the supraspinatus is available for assistance or not).[54] If the scapular upward rotators (serratus anterior and trapezius muscles) are absent, the middle and posterior fibers of the active deltoid (originating on the acromion and spine of the scapula) will not move the heavier arm but will, instead, move the lighter scapula; that is, without the stabilizing force of the upward rotators, the middle and posterior deltoid will downwardly rotate the scapula (**Fig. 7–49**), which is typically referred to as reverse action. Although the deltoid can still achieve the glenohumeral motion attributed to it when the scapular motion is restricted or limited, the glenohumeral motion occurs on a downwardly rotated scapula. Depending on the plane of elevation and amount of loss of scapulothoracic muscle force production, deltoid reverse action can also produce undesired scapular anterior tilt, and increased scapular internal rotation. The net effect of attempted abduction by the deltoid in the presence of trapezius and serratus anterior muscle paralyses is that the arm will rise from the side only about 60° to 75° (Fig. 7–49).[99]

FIGURE 7-49

Without the upward rotatory effect of the trapezius and serratus anterior, activity of the middle and posterior deltoid acting on the scapula during attempted abduction will downwardly rotate the lighter scapula while also attempting to abduct the heavier arm. The net effect of abduction of the GH joint on the downwardly rotated scapula will be to achieve only 45° to 60° of active abduction of the *arm from the side.*



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As we discussed earlier, effective deltoid activity also depends on intact rotator cuff muscles. With complete derangement of the cuff, a contraction of the deltoid results in a shrug of the shoulder (clavicular elevation at the sternoclavicular joint and upward translation of the humerus) rather than in abduction of the humerus from the side (Fig. 7–50). Isolated electrical stimulation of the axillary nerve (innervating the deltoid and teres minor muscles alone) produces approximately 40° of abduction.[100] Partial tears in or partial paralysis of the cuff will weaken the effective elevation produced by the deltoid, even though deltoid activation may increase.[101]

FIGURE 7-50

Patient attempting to elevate both arms. On the left, there is excess clavicle elevation (shoulder shrugging) through increased upper trapezius activation and deltoid activation. These muscle force imbalances often contribute to excess superior humeral translation, and inadequate scapular upward rotation as noted in this patient. This movement pattern in varying degrees may be a result of a massive rotator cuff tear, extreme shoulder stiffness with adhesive capsulitis, severely reduced glenohumeral mobility with osteoarthritis, or force production deficits of the inferior force producing components of the rotator cuff or the serratus anterior.



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Supraspinatus

The supraspinatus muscle is considered an abductor of the humerus. Like the deltoid muscle, it functions in all planes of elevation of the humerus. Its role, according to MacConaill and Basmajian,[30] is quantitative rather than specialized. The pattern of activity of the supraspinatus is essentially the same as that found in the deltoid.[54] The moment arm of the supraspinatus is fairly constant throughout the ROM and is larger than that of the deltoid for the first 60° of shoulder abduction.[22] When the deltoid is paralyzed, the supraspinatus alone can bring the arm through most, if not all, of the glenohumeral range, but the motion will be weaker. With a suprascapular nerve block paralyzing the supraspinatus and the infraspinatus but leaving the deltoid intact, the strength of elevation in the plane of the scapula is reduced by 35% at 0° and by 60% to

80% at 150°.[102]

The secondary functions of the supraspinatus are to compress the glenohumeral joint, to act as a “steerer” for the humeral head, and to assist in maintaining the stability of the dependent arm. With isolated and complete paralysis of the supraspinatus muscle, or an isolated supraspinatus tear, some loss of abduction force is evident, but most of its functions can be performed by remaining musculature. Isolated paralysis of the supraspinatus is unusual, however, because its innervation is the same as the infraspinatus and related to that of the teres minor muscle. Often, tears of the rotator cuff muscles do not remain isolated to the supraspinatus but extend to the infraspinatus or subscapularis, producing a more extensive deficit than is seen with paralysis of the supraspinatus alone.

Infraspinatus, Teres Minor, and Subscapularis

When Inman and coworkers assessed the combined actions of the infraspinatus, teres minor, and subscapularis muscles, EMG activity indicated a nearly linear rise in action potentials from 0° to 115° elevation.[54] Activity dropped slightly between 115° and 180°. These patterns are generally consistent with that reported by a more recent study in abduction.[98] Total activity in flexion was slightly greater than that in abduction.[54] As noted earlier, the medial rotary function of the subscapularis muscle diminishes with abduction, serving instead to steer the head of the humerus horizontally while continuing to work with the other cuff muscles to compress and stabilize the joint.[80,82]

Trapezius and Serratus Anterior

The upper trapezius, along with the levator scapula muscle, supports the shoulder girdle against the downward pull of gravity. Although support of the scapula in the

pendant limb is nearly passive in many individuals, loading the limb will produce activity in these muscles.[30,54] As previously discussed, the trapezius and lower serratus anterior muscles work synergistically to produce upward rotation of the scapula on the thorax. When activity of the upper and lower trapezius and serratus anterior muscles was monitored by EMG during humeral elevation, the curves were similar and complementary. Activity in the trapezius peaks above 90° in abduction, with less activity in flexion. The serratus anterior muscle shows an increase in action potentials above 90° in flexion, with less activity in abduction.[54,98] The middle trapezius muscle is also active during elevation (especially abduction) and may contribute to upward rotation of the scapula early in the range; however, it is likely to be more important in contributing to medial stabilization against the lateral pull of the serratus anterior.



Expanded Concepts 7–8

EMG and Muscle Function

EMG activity must be interpreted as only an indirect representation of muscle force production. If the length of the muscle is not held constant, an increase in EMG activity is not necessarily indicative of increased force production but, rather, may be compensating for a muscle's loss of tension with active shortening, as is proposed to be the rationale for increased end-range activity of many muscles. Higher activity levels are commonly seen in muscles at the end, despite the necessity for peak torque production near the midrange of motion when gravitational resistance torques are often greatest. In addition, for increased EMG activity to relate to increased muscle force, the velocity of contraction and type of contraction (concentric, eccentric, isometric) must not change across comparison conditions. Finally, moment arms may change during movement, which will modify the force output requirements of muscle and result in an altered EMG signal.

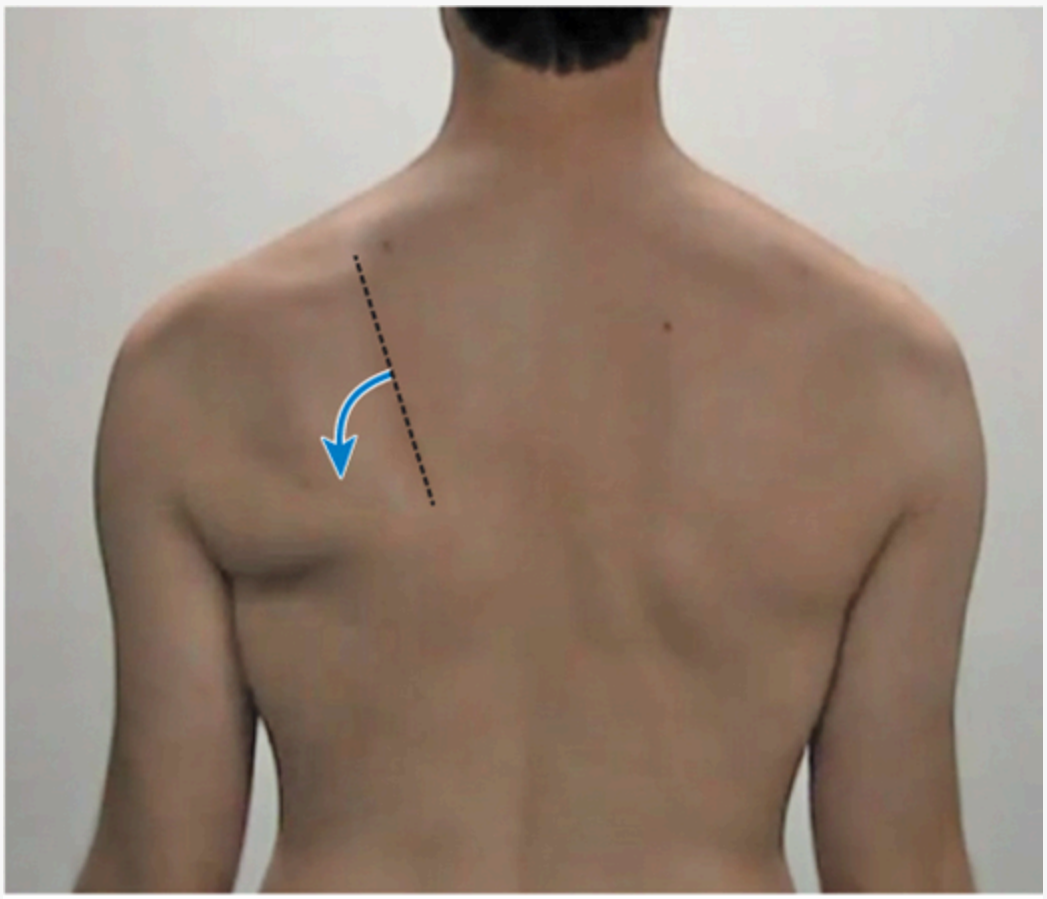
The EMG activity of a muscle during a specific motion does not define the function of the muscle. Often, muscles are active to offset unwanted translatory components of another muscle force, rather than to produce a primary rotational torque. For

example, if a muscle is active during scapular upward rotation, it cannot be presumed that this muscle has a moment arm to produce scapular upward rotation; rather, it may be active to stabilize against excessive upward rotation. This was discussed previously with trapezius function to offset lateral scapular motion from the pull of the serratus anterior, and will also be discussed as it occurs with rhomboid function during arm elevation.

In active abduction of the arm, the force and alignment of the trapezius seems more critical to scapulothoracic motion than it is in active flexion. When the trapezius is intact and the serratus anterior muscle is paralyzed, full active abduction of the arm can occur, although it is weakened, whereas flexion is more difficult. With EMG analysis, the trapezius has been found to be more active in abduction than in flexion.[97] This may be related to the increased scapulothoracic and acromioclavicular joint internal rotation noted in flexion as compared with abduction.[16] Without the trapezius (with or without the serratus anterior muscle), the scapula rests in a downwardly rotated position as a result of the unopposed effect of gravity on the scapula (**Fig. 7–51**). In addition, with full paralysis of the trapezius, full abduction may not be possible even with a fully intact serratus anterior.

FIGURE 7-51

Weakness or paralysis of the left trapezius. With trapezius weakness or paralysis, the scapula rests in a downwardly rotated position on the thorax because the pull of gravity on the shoulder girdle is largely unopposed.



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Patient Application 7–4

Upper Trapezius Overuse

Excess activation of the upper trapezius has been identified in some patients with shoulder impingement symptoms.[17] This can result in an imbalance of forces between the trapezius and serratus. The result can be a shoulder shrug type of motion (excess clavicle elevation) when trying to elevate the arm and less efficient upward rotation of the scapula on the thorax, although typically less pronounced than that seen with a massive rotator cuff tear or other severe conditions (see Fig. 7–50). Because of the coupling of clavicle elevation to scapular anterior tilt, excess upper trapezius activation may also result in reduction of the normal scapular posterior tilt that should occur during arm elevation. Prolonged overuse of the upper trapezius can result in muscle fatigue and pain in this muscle. Patients who report

pain over the upper trapezius region should be evaluated for an upper trapezius overuse pattern with arm elevation.

Although the trapezius is more active in abduction, the serratus anterior muscle seems to be better aligned and the more critical of the two muscles in producing scapular upward rotation during flexion of the arm. If the serratus anterior muscle is intact, trapezius muscle paralysis results in a loss of force of shoulder flexion but a fairly normal range of flexion. If the serratus anterior muscle is paralyzed (even in the presence of a functioning trapezius), flexion will be diminished in strength and limited in range. When the scapular retraction component of the trapezius is not opposed by the absent serratus anterior muscle, the trapezius is unable to effectively upwardly rotate the scapula.

The role of the serratus anterior muscle in normal shoulder function appears to be essential in many aspects. This includes it being the only muscle capable of producing simultaneous scapular upward rotation, posterior tilting, and external rotation, the three component motions of the scapula on the thorax that have been identified as occurring during elevation of the arm. The serratus also has the largest moment arms of any of the scapulothoracic muscles, regardless of the changing scapulothoracic axis of rotation (see Fig. 7–46). The serratus is the primary stabilizer of the inferior angle and medial border of the scapula to the thorax. Finally, reduced serratus anterior muscle activity has been identified in patients with shoulder impingement, which further suggests the importance of this muscle to normal shoulder function.[17]

The serratus anterior is the prime mover for upward rotation of the scapula. The serratus and trapezius are also synergists for the deltoid during abduction at the

glenohumeral joint. The trapezius and serratus anterior muscles stabilize the scapula and prevent the undesired downward rotary movement of the scapula by the middle and posterior deltoid segments that are attached to the scapula (see Fig. 7–49). The trapezius and serratus anterior muscles maintain an optimal length-tension relationship with the deltoid and permit the deltoid to carry its heavier distal lever through its full ROM. Thus, the role of the scapular forces of the trapezius and the serratus anterior muscles is both agonistic to scapular movement and synergistic with glenohumeral movement.

Rhomboids

The **rhomboid major** and **minor** muscles are active in elevation of the arm, especially in abduction.[98] These muscles serve a critical function as stabilizing synergists to the muscles that upwardly rotate the scapula. If the rhomboids, which are downward rotators of the scapula, are active during upward rotation of the scapula, these muscles must be working eccentrically to control the change in position of the scapula produced by the trapezius and the serratus anterior muscles.[103] Paralysis of these muscles causes disruption of the normal scapulohumeral rhythm and may result in diminished ROM. Like the middle and lower trapezius, the rhomboid muscles act primarily to offset the lateral translation component of the serratus anterior muscle and help prevent excess internal rotation of the scapula at the acromioclavicular joint by stabilizing the medial or vertebral border of the scapula to the thorax.

Integrated Shoulder Muscle Function: Depression

Shoulder **depression** is the second of the two primary patterns of shoulder complex function. Depression involves the *forceful* downward movement of the arm against

resistance. When the arm is weightbearing or holding on to an object (e.g., a chinning bar), shoulder depression moves the trunk upward relative to the arm. During shoulder depression, the scapula tends to rotate downward, tilt anteriorly and adduct, but a consistent pattern or ratio of scapula to humeral movement has not been described.

Latissimus Dorsi and Pectoralis

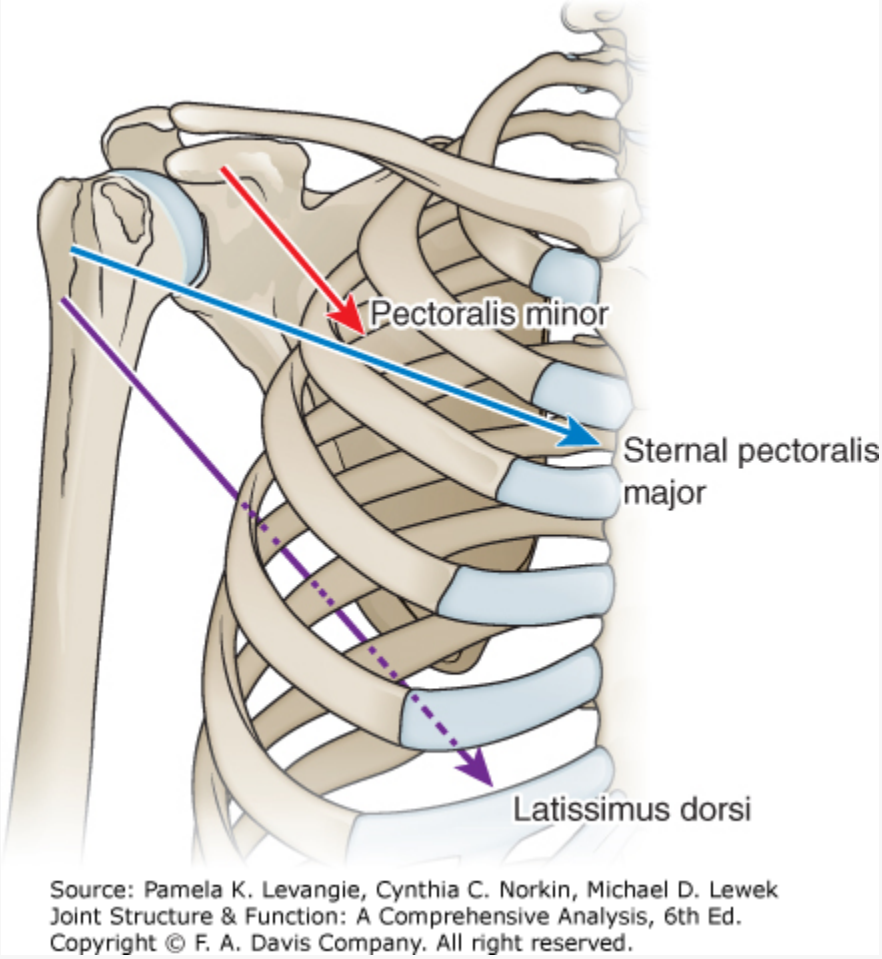
Through its attachment to both the scapula and humerus, **latissimus dorsi** activation will adduct and depress the scapula and raise the trunk relative to the humerus when the arm is weightbearing or fixed. The latissimus dorsi is a critical muscle when a cane, crutches, or walker is needed to assist with gait. When the hands are bearing weight through one of these assistive devices, latissimus dorsi activation lifts the pelvis and lower extremity beneath the fixed scapula to allow forward progression of the legs.

The **sternal portion** of the **pectoralis major** is also an important shoulder complex depressor. The action of the pectoralis major parallels that of the latissimus dorsi muscle, with the pectoralis major located anterior to the glenohumeral joint rather than posterior (Fig. 7–52). During upper-extremity weightbearing, the pectoralis major and the latissimus dorsi muscles combine to depress the shoulder complex while synergistically offsetting anterior/posterior translation of the humerus and protraction/retraction of the shoulder. The **pectoralis minor** muscle assists the latissimus dorsi and pectoralis major (Fig. 7–52) by directly depressing the scapula through its attachment on the coracoid process. The pectoralis minor is oriented to internally rotate, downwardly rotate, and anteriorly tilt the scapula so the length of this muscle may influence full scapula movement during arm elevation.[104] The pectoralis minor is considerably active during weightbearing activities that involve scapula internal

rotation.[105]

FIGURE 7-52

Muscles of depression. The anteriorly located sternal portion of the pectoralis major and pectoralis minor works with the large posteriorly located latissimus dorsi to forcefully depress the shoulder girdle.



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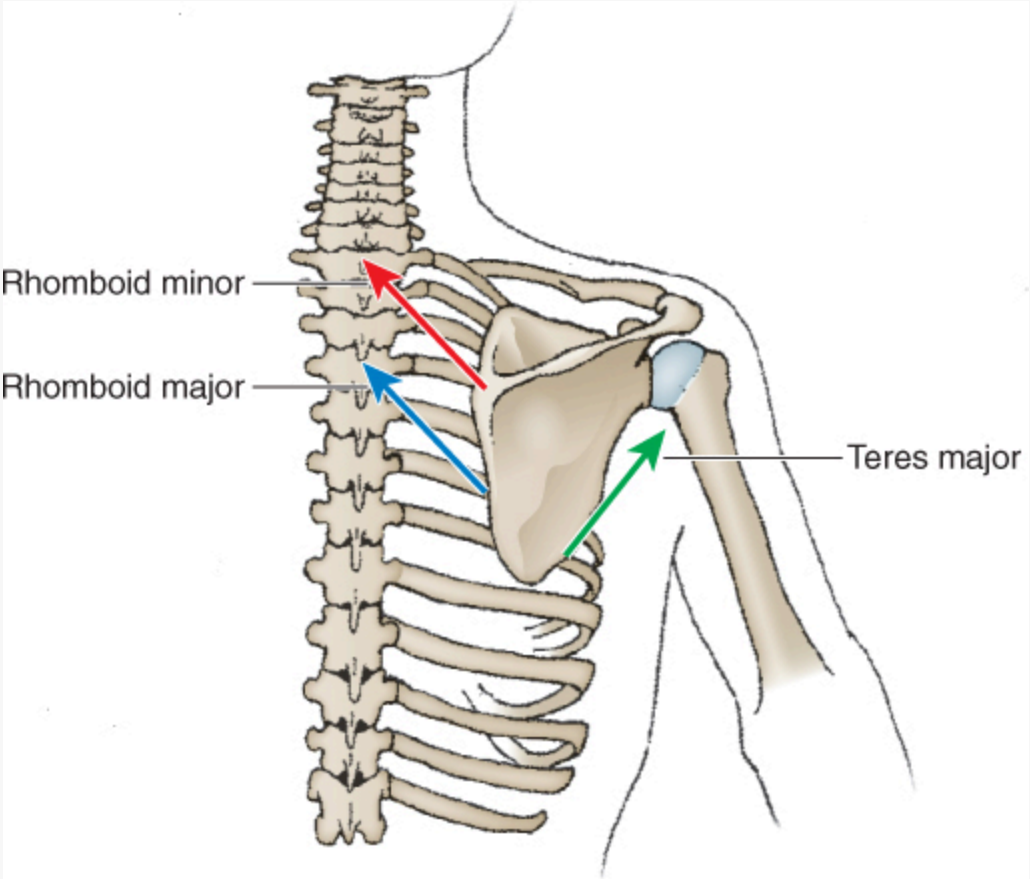
Teres Major and Rhomboids

The **teres major** and rhomboid muscles are strongly synergistic during adduction and depression of the humerus. Teres major activation is high during resisted humeral adduction and extension, but these functions depend on stability of the muscles'

proximal attachment on the scapula. Without this stability, teres minor activation would move the lighter scapula toward the heavier humerus in another example of reverse action. The rhomboids, through their attachment on the scapula medial border and their oblique orientation, provide this proximal segment stability (Fig. 7–53). The downward rotation torque of the rhomboid muscles offsets the undesired upward rotary torque of the teres major muscle, allowing the teres major muscle to move the heavier humerus. The rhomboids are assisted in this stabilization function by the anteriorly located pectoralis minor muscle.

FIGURE 7-53

For the teres major muscle to extend the heavier humerus rather than upwardly rotate the lighter scapula, the synergy of the rhomboid muscles is necessary to stabilize the scapula.



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Patient Application 7–5

Postural Faults

Louis Haddad is a 17-year-old who has recently taken up free weights in an attempt to correct his bad posture but is now having shoulder pain when he reaches over shoulder level. He presents with a forward shoulder posture that may be contributing to his symptoms. This posture is often related to medial rotation of the humerus and, indirectly, clavicular protraction due to pectoralis major tightness, and to scapular downward rotation, internal rotation, and anterior tilting from pectoralis minor tightness.[104] These motions are all antagonistic to the motions of the scapula and humerus that must occur during normal arm elevation. Because of the attachment of the pectoralis minor muscle to the coracoid process and rib cage (see Fig. 7–52), decreased extensibility or hypertonicity during arm elevation could cause the muscle

to limit scapular upward rotation, posterior tilting, and external rotation.[104] Decreased extensibility in the pectoralis major could limit the ability to laterally rotate the humerus and retract the clavicle during arm elevation. A subtle but potentially important effect may also be to increase the risk of mechanical impingement of the rotator cuff tendons and long head of the biceps brachii in elevation of the arm. The risk may be increased as a result of a decreased subacromial space because of the inability of the scapula to posteriorly tilt and externally rotate to clear the acromion during elevation. A reduction in scapular upward rotation may also result in hypermobility of the glenohumeral joint, which increases the likelihood that the humerus will impinge on the coracoacromial arch. Furthermore, a restriction in lateral rotation of the humerus from pectoral major tightness may not allow the greater tubercle to adequately clear the acromion.

Life Span and Clinical Considerations

Life Span Considerations

As might be expected, aging and the cumulative effects of joint loading may have negative effects on the shoulder complex. However, the relatively minimal weightbearing function of the upper extremity results in less severe degenerative changes than those recognized in the large lower extremity joints. Degenerative changes to the shoulder complex joints and structures usually begin around the fourth decade and occur most often at the acromioclavicular joint and lateral clavicle. These changes include osteophytes, sclerosis, and joint narrowing or degeneration.[106,107] Acromial osteophytes in particular are more frequently seen with aging, whereas glenohumeral degenerative changes become more frequent after age 60.[108,10] The more frequent degenerative changes at the acromioclavicular joint suggest that this articulation is subject to high stresses over the life span. Bone mineral density of the proximal humerus also declines with age.[110]

Gross shoulder motion gradually declines with age, with decreasing shoulder flexion and abduction after the fifth decade, and small decreases in lateral rotation ROM that are correlated with age.[111–113] Kinematic studies report less glenohumeral joint lateral rotation and scapulothoracic internal rotation during functional tasks in older individuals, whereas children have demonstrated decreased scapulothoracic posterior tilt during arm elevation compared with healthy adults.[114,115] Age-related declines in isometric strength are seen in both men and women, with greater rotational strength decreases noted in men only after age 60.[113,116] Despite age-related strength and motion declines, corresponding decreases in function were not present when measured using both functional tasks and standardized outcome measures.[113]

Degenerative changes in the acromioclavicular joint may result in pain in the same area of the shoulder as pain from supraspinatus or rotator cuff lesions. Pain due to acromioclavicular degeneration is reported to be found more typically when the arm is raised to end range or when the arm is adducted across the body, compressing the acromioclavicular joint surfaces.[117] The long head of the biceps brachii similarly can produce pain in the anterosuperior shoulder. Because the long head of the biceps tendon also passes directly beneath the impinging structures of the coracoacromial arch, it is subject to some of the same degenerative changes and the same trauma seen in the tendons of the rotator cuff. Whether the biceps is actively contributing to elevation of the arm, to joint stabilization, or is passive, the tendon of the biceps must slide within the bicipital groove and under the transverse humeral ligament as the humerus moves. If the bicipital tendon sheath is worn or inflamed, or if the tendon is hypertrophied (as often seen with rotator cuff tears), the gliding mechanism may be interrupted and pain produced. A tear in the transverse humeral ligament may result in

the tendon of the long head popping in and out of the bicipital groove with rotation of the humerus, a potentially wearing and painful microtrauma.

Patient Application 7–6

AC Joint Degeneration

Mary Czerny is a 71-year-old who presents with shoulder pain located at the acromioclavicular joint, which may be related to AC joint degeneration. The AC joint frequently shows evidence of degeneration after the sixth decade, which may be a source of pain, crepitus, and movement faults. When pain is noted at the end of arm elevation, it: may be indicative of acromioclavicular joint pathology or degeneration.

Structural Dysfunction

Completion of the range of elevation of the arm depends on the ability of the glenohumeral, sternoclavicular, and acromioclavicular joints to each make the needed contribution. Disruption of movement in any of the participating joints can result in a loss of ROM. Once restrictions to function are introduced, the concept of scapulohumeral rhythm is altered; that is, a reduction in glenohumeral joint range may *not* result in a proportional decrease in scapulothoracic range. The ratio of movement is no longer consistent because the body will likely recruit remaining motion at other joints.[118] Although it is not necessarily predictable, the restriction of motion at any one joint in the shoulder complex may result in the development of some hypermobility (and reduced stability) in remaining articulations.

Foundational Concepts 7–4

Glenohumeral Joint Hypomobility

If motion at the glenohumeral joint is restricted by pain or disease, the total ROM available to the humerus will be reduced. Whatever portion of the motion remains at

the glenohumeral joint will still be accompanied by scapulothoracic motion. A restriction of the humerus, limiting glenohumeral joint abduction to approximately 60°, theoretically can combine with up to 60° of scapulothoracic motion to provide a total available range of 120° as the arm is raised from the side. Recent research has also provided evidence of increased scapulothoracic upward rotation, or scapular "substitution" in cases where glenohumeral mobility is reduced, such as glenohumeral osteoarthritis or adhesive capsulitis.[118]

 Expanded Concepts 7–9

Acromioclavicular Joint Hypomobility

Hypomobility of the acromioclavicular joint is far less common than hypermobility and instability. Fusion of the joint generally occurs only through surgical fixation. With limited or no acromioclavicular joint mobility, motion throughout the closed chain formed by the acromioclavicular, sternoclavicular, and scapulothoracic joints could be limited. If the clavicle attempted to protract with a fused acromioclavicular joint (and a fixed scapuloclavicular relationship), the clavicular protraction would snug the scapula into the thorax, and further motion would be restricted or absent. Clavicular protraction (and scapular internal rotation at the initiation of flexion of the arm) is also dependent on the ability of the acromioclavicular joint to allow the scapula to internally rotate to orient the glenoid fossa anteriorly and to accommodate to the curvature of the thorax. Given the strong forces acting on the scapula and clavicle, surgical fixation of the acromioclavicular joint often fails and is now an uncommon procedure.

Mechanical deviations in glenohumeral stabilization factors may result in injury to other structures of the joint besides the rotator cuff (e.g., the glenoid labrum) and to subluxation of the glenohumeral joint. The glenohumeral joint is the most frequently dislocated of all the joints in the body. Capsuloligamentous and muscle reinforcement to the glenohumeral joint are weakest inferiorly, but it is most common for the glenohumeral joint to dislocate anteriorly, or anteriorly and inferiorly, because of the type of forces to which it is exposed. Although the subscapularis and the glenohumeral

ligaments reinforce the capsule anteriorly, a force applied to an abducted, laterally rotated arm can force the humeral head beyond the limits of the anterior glenoid. A predisposition to glenohumeral subluxation or dislocation is multifactorial. Saha suggested that people are most susceptible when their individual structural variations are in the direction of (1) anterior tilt of the glenoid fossa in relation to the scapular plane, resulting in less of a mechanical block of the glenoid fossa to anterior translation; (2) excessive retroversion of the humeral head; or (3) weakened rotator cuff muscles.[82] Alternatively, Weiser suggested that scapular internal rotation results in less anterior humeral head translation and greater tension in the anterior capsule.[119]

The most common diagnosis for shoulder pain and dysfunction is shoulder "impingement." This is typically divided into entrapment of the rotator cuff tendons beneath the coracoacromial arch (referred to as *subacromial impingement*) or entrapment between the humeral head and glenoid (referred to as *internal impingement*).[90] However, it is becoming increasingly recognized that the impingement diagnosis is overly broad and may include a wide variety of tissue pathologies and movement presentations.[120]

Two other common clinical presentations are shoulder instabilities and stiffness. Instabilities or hypermobilities of the shoulder joint are not surprising, as noted earlier, given the large ROM necessary for normal function of the shoulder joint. Extremes of shoulder stiffness or hypomobility are most commonly seen with adhesive capsulitis or "frozen shoulder," where the large redundancy of the glenohumeral joint capsule can become adhered to itself or to the humerus, substantially limiting ROM. Glenohumeral hypomobility can also occur with osteoarthritis or rheumatoid arthritis. Continued

enhancement of our understanding of joint structure and function will be integral to continuing to advance the most useful diagnostic classifications for shoulder pain and dysfunction, including diagnoses based on the movement system. Diagnostic classification provides a necessary foundation for targeted and effective preventive and rehabilitative strategies.

Summary

- In this chapter, we laid the foundation for understanding more distal upper extremity joint function by exploring the intricate dynamic stabilization of the shoulder complex. The more distal joints of the upper extremity depend on the dual mobility/stability roles of the shoulder complex.
- Whereas function in the hand, for instance, can continue on a limited basis with loss of shoulder mobility, loss of shoulder stability can render the remaining function in the hand unusable, with the hand unable to reach a necessary positional workspace. In the next chapter, we will explore the elbow as the intermediary between the shoulder and the hand.

Study Questions

1. Describe the intra-articular motions of the sternoclavicular joint for elevation/depression, protraction/retraction, and axial rotation.
2. Describe the roles of the costoclavicular and interclavicular ligaments at the sternoclavicular joint.

3. Discuss the relevance of the sternoclavicular disc to sternoclavicular joint congruency, joint motion, and joint function.

4. Describe the scapular movements that take place at the acromioclavicular joint.

5. Discuss the relevance of the coracoclavicular ligament to acromioclavicular joint function.

6. Discuss the anatomical configuration of the humerus and the glenoid fossa as they relate to glenohumeral joint stability. What role do the glenoid labrum and joint capsule play in joint stability?

7. What is the most frequent direction of glenohumeral dislocation? Why?

8. Compare the relative stability and tendency toward degenerative changes in the glenohumeral, acromioclavicular, and sternoclavicular joints.

9. What are the advantages of the coracoacromial arch? What are the disadvantages?

10. What intra-articular motions must occur at the glenohumeral joint for full abduction to occur? What is the normal ROM?

11. What muscle is the prime mover in shoulder glenohumeral flexion and abduction? What synergy is necessary for normal function of this muscle? Why?

12. Why is the supraspinatus able to abduct the shoulder without additional muscular synergy?

13. What accounts for the static stabilization of the glenohumeral joint when the arm is at the side? What happens if you excessively load the hanging (dependent) limb?

14. Identify five factors that contribute to dynamic stabilization of the glenohumeral joint in either flexion or abduction.

15. What is the total ROM available to the humerus in elevation? How is this full range achieved?

16. How does the shape of the clavicle contribute to elevation of the arm?

17. What muscles are necessary to produce the normal scapular and humeral movements in elevation of the arm?

18. If the scapulothoracic joint were fused in neutral position, what active range of elevation would still be available to the upper extremity?

19. What is the most common traumatic problem at the acromioclavicular joint? What deficits is a person with this disability likely to encounter?

20. What are the consequences of a rupture of the coracoclavicular ligaments?

21. If the glenohumeral joint were immobilized by osteoarthritis, what range of elevation would be available to the upper extremity?

22. If isolated paralysis of the supraspinatus were to occur, what would be the likely functional deficit?

23. If the muscles of the rotator cuff are paralyzed, what is the effect when abduction of the arm is attempted?

24. When there is paralysis of the trapezius and the serratus anterior muscles, what is the functional deficit when abduction of the arm is attempted?

25. If the deltoid alone is paralyzed, what happens with attempted abduction of the arm? With attempted flexion of the arm?

26. What is the role of the rhomboids in elevation of the arm?

27. What differences do you see in attempted abduction if the trapezius alone is paralyzed, compared with the differences you see if the serratus anterior muscles are paralyzed?

28. What muscular synergy does the teres major muscle require to perform its function?

29. Describe why electromyographic activity of the deltoid in normal abduction typically shows a rise in activity beyond the ROM (90°) where the greatest gravitational torque resists its function?

30. Which of the joints of the shoulder complex is most likely to undergo degenerative changes over time? Which is least likely?

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Chapter 8: The Elbow Complex

Amee Seitz; Jeff Hartman

Anatomy Overview

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MUSCLE ATTACHMENTS OF THE ELBOW JOINT		
TABLE KEY: Primary movers Secondary movers		
Sagittal Plane (Humeroulnar and humeroradial joints)	Flexion	Extension
	Biceps brachii Brachialis Brachioradialis Pronator teres	Triceps brachii Anconeus
Transverse Plane (Radioulnar joints)	Supination	Pronation
	Biceps brachii Supinator	Pronator teres Pronator quadratus

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ELBOW MUSCLE ATTACHMENTS

Muscles	Proximal Attachment	Distal Attachment
Biceps brachii	Short head: Tip of coracoid process of scapula Long head: Supraglenoid tubercle of scapula	Tuberosity of radius and fascia of forearm via bicipital aponeurosis
Brachialis	Distal half of anterior surface of humerus	Coronoid process and tuberosity ulna
Brachioradialis	Proximal two thirds of lateral supracondylar ridge of humerus	Lateral surface of distal end of radial styloid process
Pronator teres	Ulnar Head: Coronoid process humeral head: medial epicondyle of humerus	Middle convexity of lateral surface of radius
Triceps brachii	Long head: Infraglenoid tubercle of scapula Lateral head: Posterior surface of humerus, superior to radial groove Medial head: Posterior surface of humerus, inferior to radial groove	Proximal end of olecranon of ulna
Anconeus	Lateral epicondyle of humerus	Lateral surface of olecranon and superior part of posterior surface of ulna

Introduction

The joints and muscles of the elbow complex are designed to serve the hand. They provide mobility for the hand in space by shortening and lengthening the upper extremity, which allows the hand to be brought close to the face for eating and grooming or to be placed at a distance from the body equal to the length of the entire upper extremity. Rotation at the elbow complex provides additional mobility for the hand. In conjunction with providing mobility for the hand, the elbow complex structures also provide stability for skilled or forceful movements when performing manual activities with tools or implements. Many of the 15 muscles that cross the elbow complex also act at either the wrist or shoulder, thereby linking the elbow with function of the joints proximal and distal to the elbow.[1]

The elbow complex includes the elbow joint (**humeroulnar** and **humeroradial joints**) and the **proximal** and **distal radioulnar joints.** The elbow joint is considered to be a compound joint that functions as a modified or loose hinge joint. The elbow joint has one degree of freedom, permitting the motions of flexion and extension in the sagittal plane around a coronal axis. A small amount of axial rotation and side-to-side motion (abduction/adduction) of the ulna occurs during flexion and extension, which is why the elbow is considered to be a modified or loose hinge joint rather than a pure hinge joint.[2] Two major ligaments and five muscles are directly associated with the elbow joint. Three of the muscles are flexors that cross the anterior aspect of the joint. The other two muscles are extensors that cross the posterior aspect of the joint.

The proximal and distal radioulnar joints are linked and function as one joint. The two joints acting together produce rotation of the forearm and have one degree of freedom of motion. The radioulnar joints are uniaxial diarthrodial joints of the pivot (trochoid)

type and permit rotation (supination and pronation), which occurs in the transverse plane around a longitudinal axis. Six ligaments and four muscles are associated with these joints. Two muscles produce supination and two produce pronation. The elbow joint and the proximal radioulnar joint are enclosed in a single joint capsule, but constitute distinct articulations.

The remaining content of this chapter will expand on this introduction and will explore in more depth the structure and function of the elbow complex to provide a better understanding of and appreciation for the interaction of the joint structures that provide stability and mobility in multi-planes. We will also develop an understanding of how the elbow is a vital link in the entire upper extremity chain, rendering it vulnerable to traumatic and repetitive injury.

Structure and Function of the Humeroulnar and Humeroradial Articulations

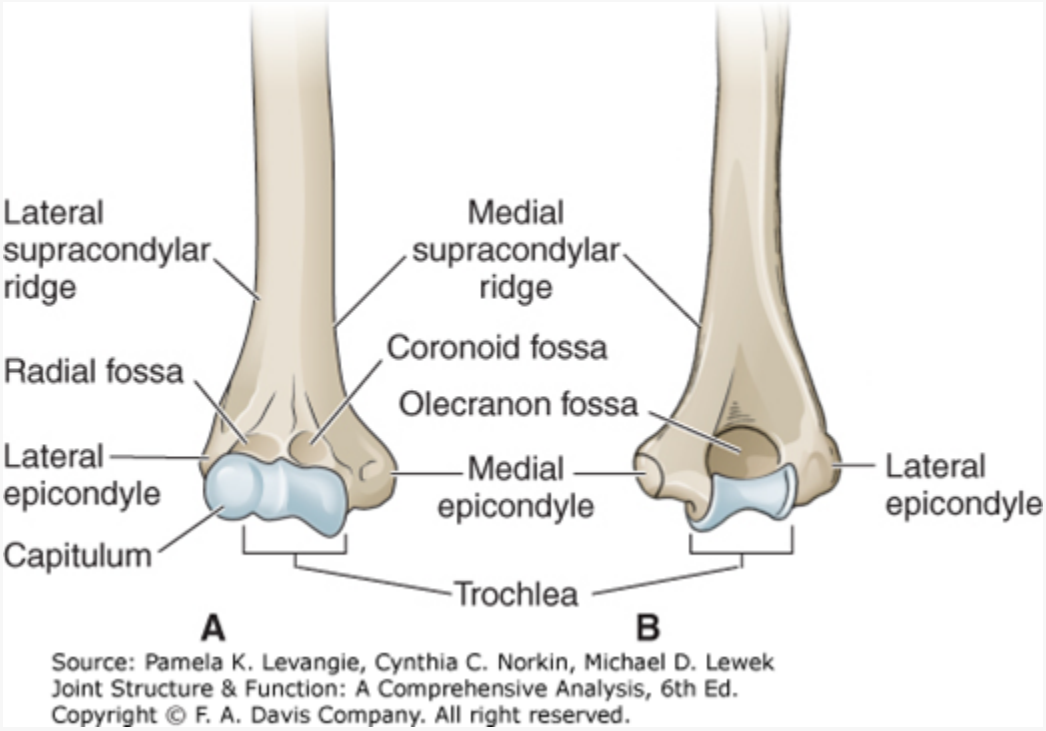
Structure

Proximal Joint Surface

The articulating surfaces on the anterior aspect of the distal humerus are the hourglass-shaped trochlea and the spherical capitulum (Fig. 8–1). These structures are situated between the medial and lateral epicondyles of the humerus. The **trochlea** forms the proximal portion of the medially located humeroulnar articulation and is set at an angle on the medial aspect of the distal humerus, lying slightly anterior to the humeral shaft. The **trochlear groove** spirals obliquely around the trochlea and divides the trochlea into medial and lateral portions.

FIGURE 8-1

Proximal articular surfaces of the right elbow joint. **A.** Articulating surfaces on the anterior aspect of the right distal humerus. **B.** The olecranon fossa on the posterior aspect of the right distal humerus.



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
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The indentation in the humerus called the **coronoid fossa** is designed to receive the coronoid process of the ulna at the end of the elbow flexion range of motion (ROM). The **capitulum** is the proximal portion of the humeroradial articulation and is located on the lateral distal humerus. The capitulum, like the trochlea, lies anterior to the shaft of the humerus. The **capitulotrochlear groove** separates the capitulum from the trochlea (see Fig. 8–1A). The **radial fossa** just above the capitulum is designed to receive the head of the radius in full elbow flexion. Posteriorly, the distal humerus is indented by the **olecranon fossa,** designed to receive the olecranon process of the ulna at the end of the

elbow extension ROM (Fig. 8–1B).

Distal Joint Surfaces

The articulating surfaces of the ulna and radius correspond to the proximal articulating surfaces on the humerus (Fig. 8–2). The ulnar articulating surface of the humeroulnar joint is a deep, semicircular concavity called the **trochlear notch** (Fig. 8–3).[3] The proximal portion of the notch is divided into two unequal parts by the **trochlear ridge** that corresponds to the trochlear groove on the humerus. The **coronoid process** of the ulna forms the distal end of the notch, whereas the **olecranon process** forms the proximal end of the notch. There is a facet on the ulna lateral to the coronoid process, the **radial notch,** where the head of the radius articulates. The distal articulating surface of the humeroradial joint is composed of the **head of the radius** (Fig. 8–4). The radial head has a slightly cup-shaped concave surface called the **fovea** that is surrounded by a rim. The radial head's convex rim fits into the **capitulotrochlear groove** (see Figure 8–1A).

FIGURE 8-2

Right elbow articulations. **A.** An anterior view of the right elbow showing the humeroulnar and humeroradial joints. **B.** A posterior view of the right elbow showing the humeroulnar and humeroradial joints.

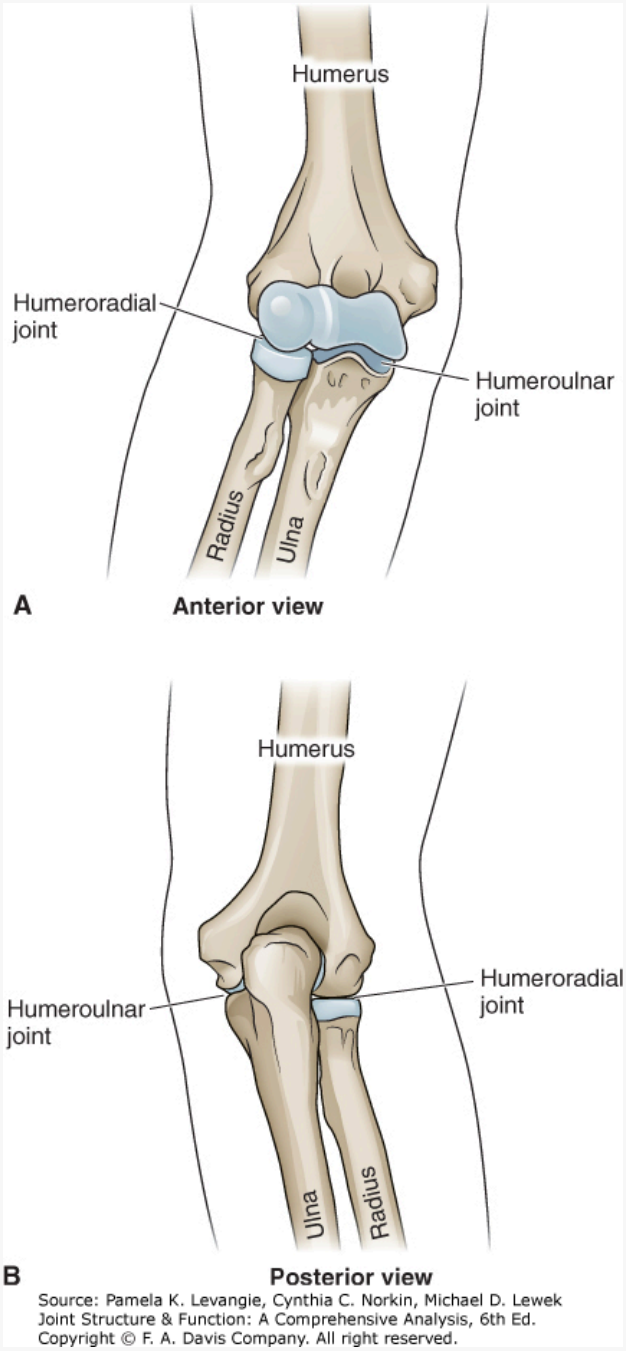
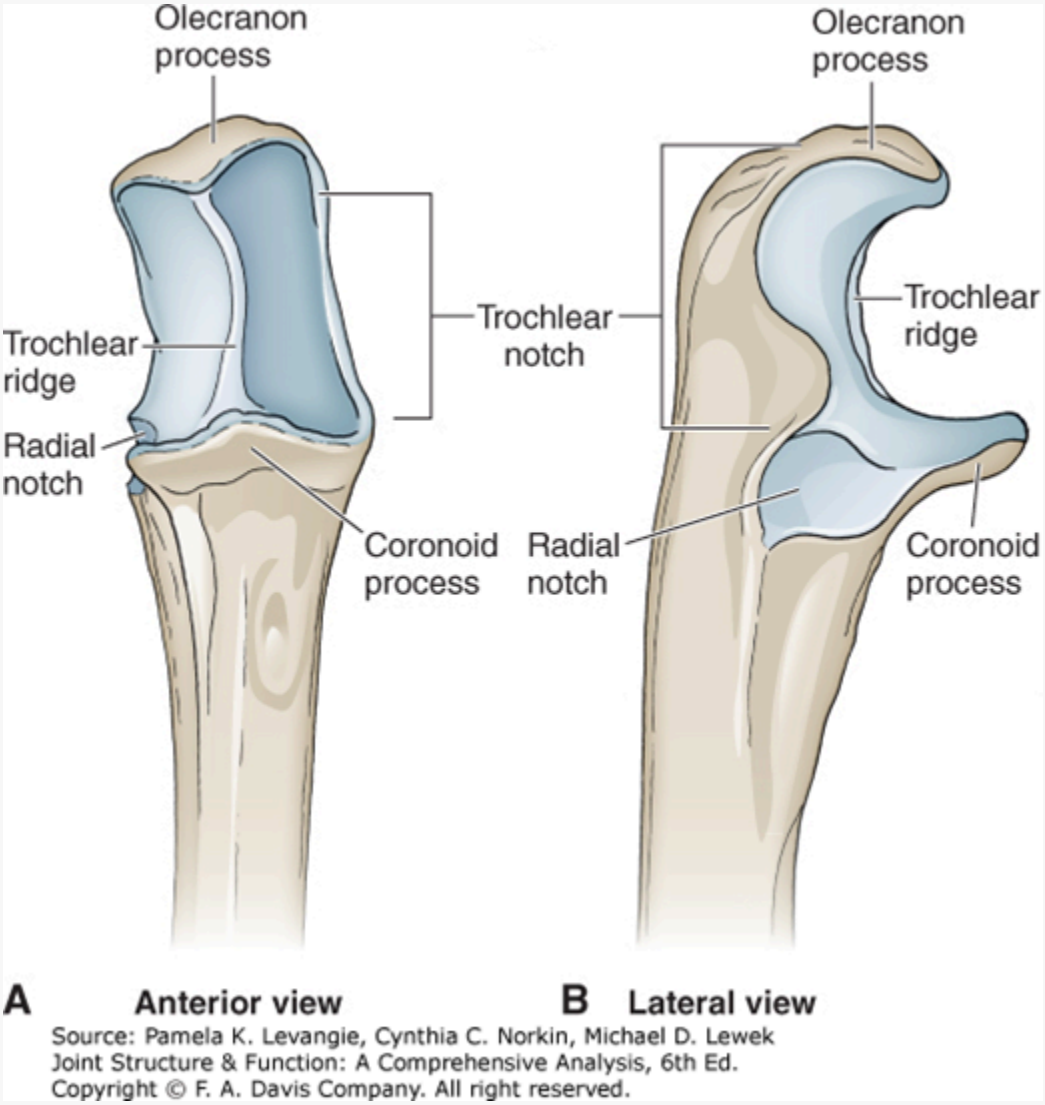


FIGURE 8-3

A. Anterior view of the right ulna's trochlear notch and ridge. B. Lateral view of the right trochlear notch and ridge. The coronoid process

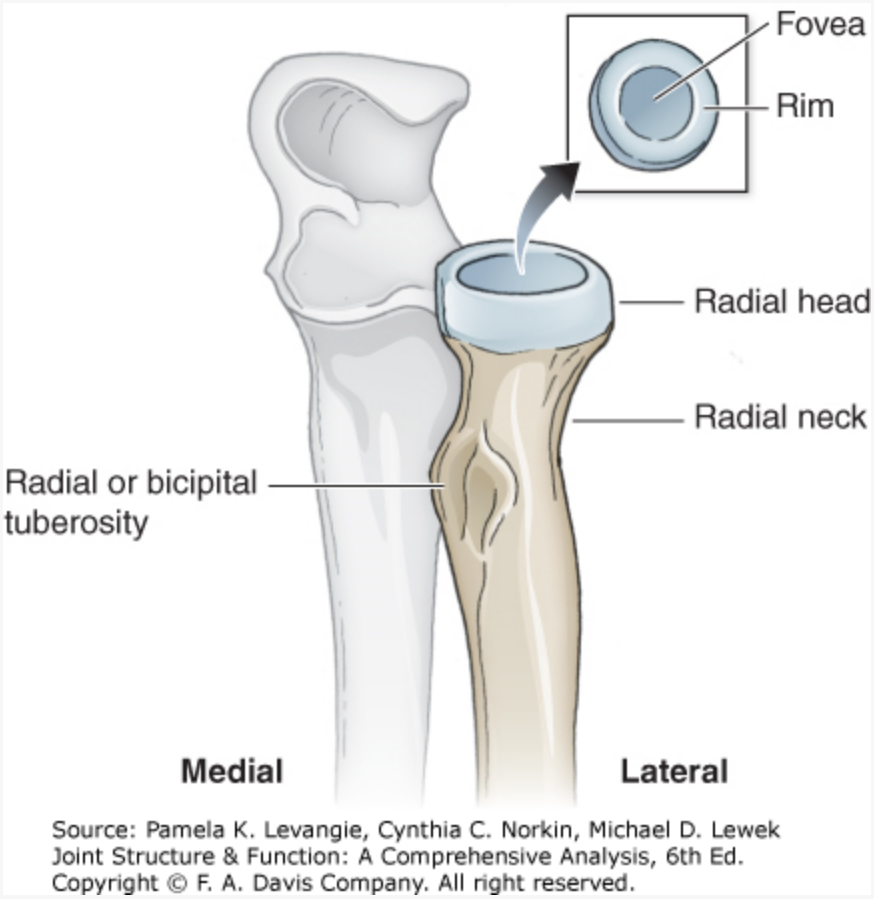
forms the distal end of the trochlear notch and the olecranon process projects over the proximal end.



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FIGURE 8-4

Fovea and rim of the radial head.



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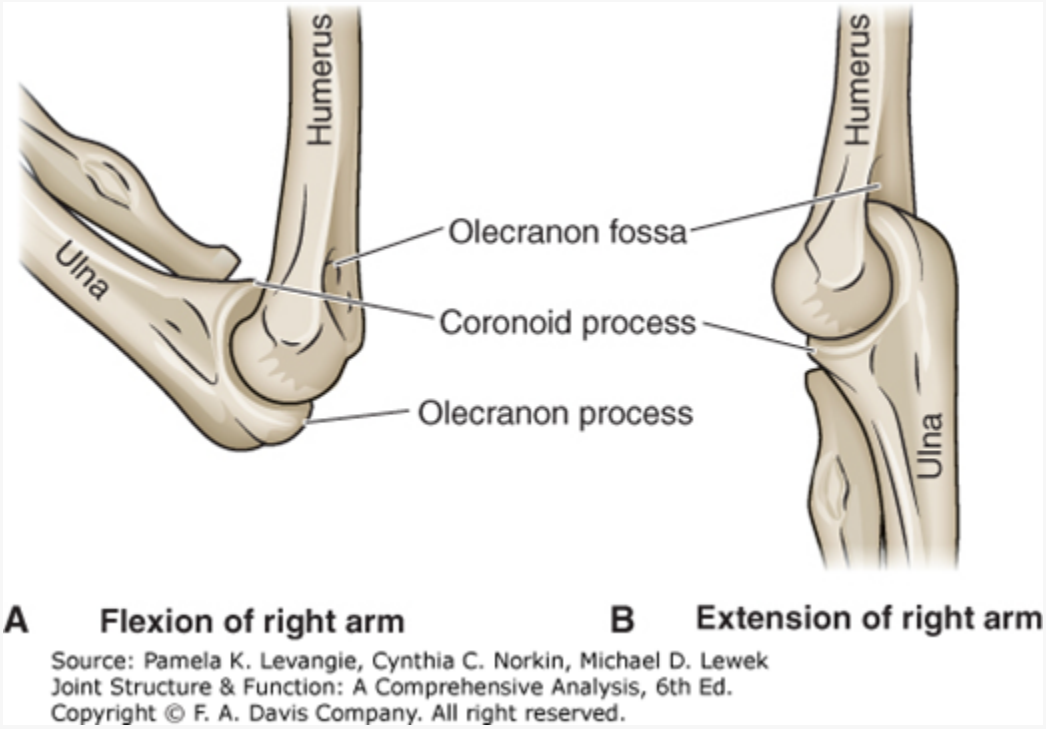
Joint Articulations

Motion at the humeroulnar joint occurs primarily as a sliding motion of the trochlear notch of the ulna on the trochlea of the humerus (Fig. 8–5). In flexion, the trochlear notch of the ulna slides along the trochlea until the coronoid process of the ulna enters the coronoid fossa in full flexion (Fig. 8–5A).[3] In extension, sliding continues until the olecranon process enters the olecranon fossa (Fig. 8–5B).

FIGURE 8-5

A. In flexion, the coronoid process reaches the coronoid fossa. B. In

extension, the olecranon process enters the olecranon fossa.

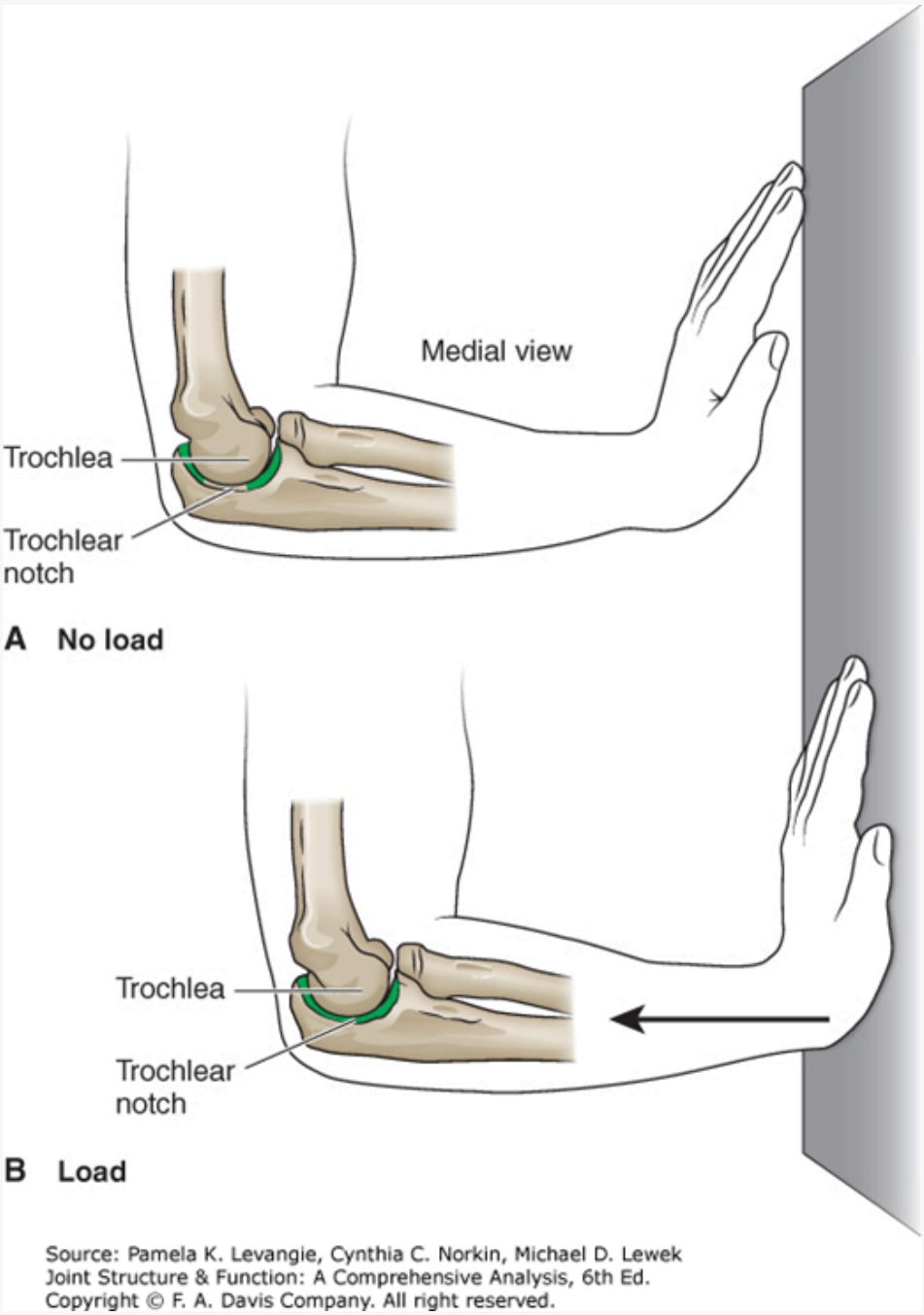


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Although the opposing articulating surfaces of the trochlea and trochlear notch appear to be completely congruent, experiments with cadaveric specimens have shown that the articulating surface of the trochlea does not contact the deepest portion of the notch unless the joint is heavily loaded (Fig. 8–6).[4–7] For example, Eckstein and colleagues determined that at loads of 25 N (simulating resisted elbow extension) with the elbow positioned at 30° increments up to 120° of flexion, no surface contact occurred between the trochlea and the depths of the trochlear notch in any of the six elbow specimens studied (Fig. 8–6A).[4] At a load of 500 N (approximately 112 lb), the articulating surface contact areas expanded from the sides toward the depths of the notch (Fig. 8–6B).

FIGURE 8-6

Left elbow joint. **A.** No contact occurs between the trochlea and deepest portion of the trochlear notch from 30° to 120° of flexion. Under no-load conditions, contact is primarily on the sides of the notch. **B.** Contact areas expand from the sides toward the depth of the trochlear notch when a load is applied.



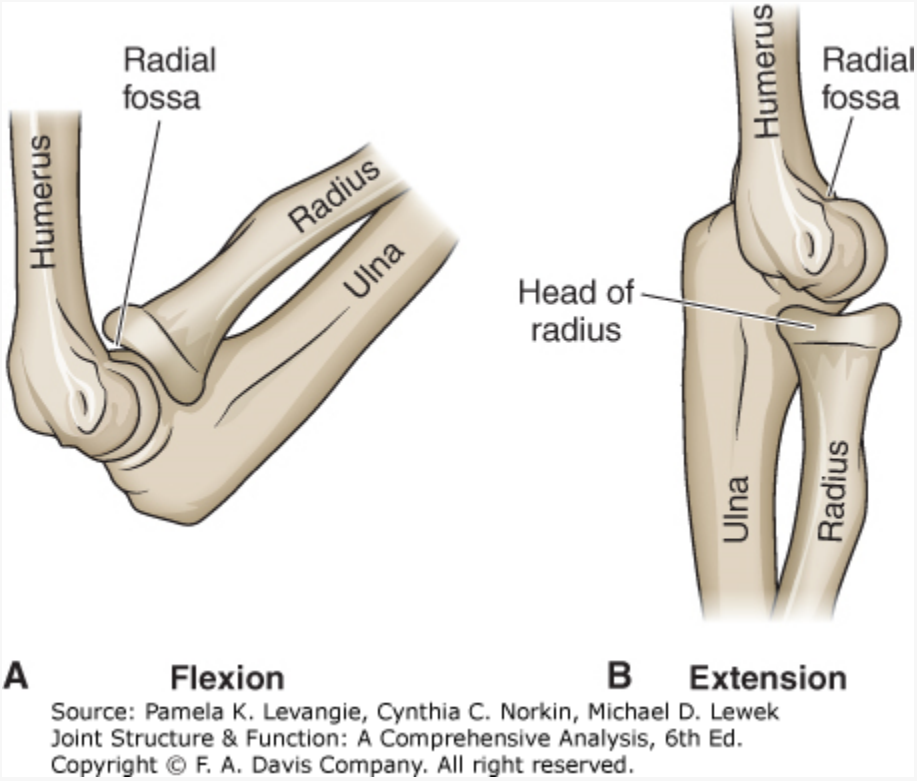
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Articulation between the radial head and the capitulum at the humeroradial joint involves sliding the shallow concave radial head over the convex surface of the capitulum (Fig. 8–7). The humeral capitulum is slightly smaller than the corresponding radial fovea, so the joint surfaces are slightly incongruent.[8] In full flexion, the rim of the

radial head slides into the radial fossa as the end of the flexion range is reached (Fig. 8–7A).[9] In full extension, no contact occurs between the articulating radial head and capitulum (Fig. 8–7B).

FIGURE 8-7

A. During elbow flexion, the rim of the radius slides in the radial fossa on the humerus. **B.** In full elbow extension, there is no contact between the capitulum and the radial head.



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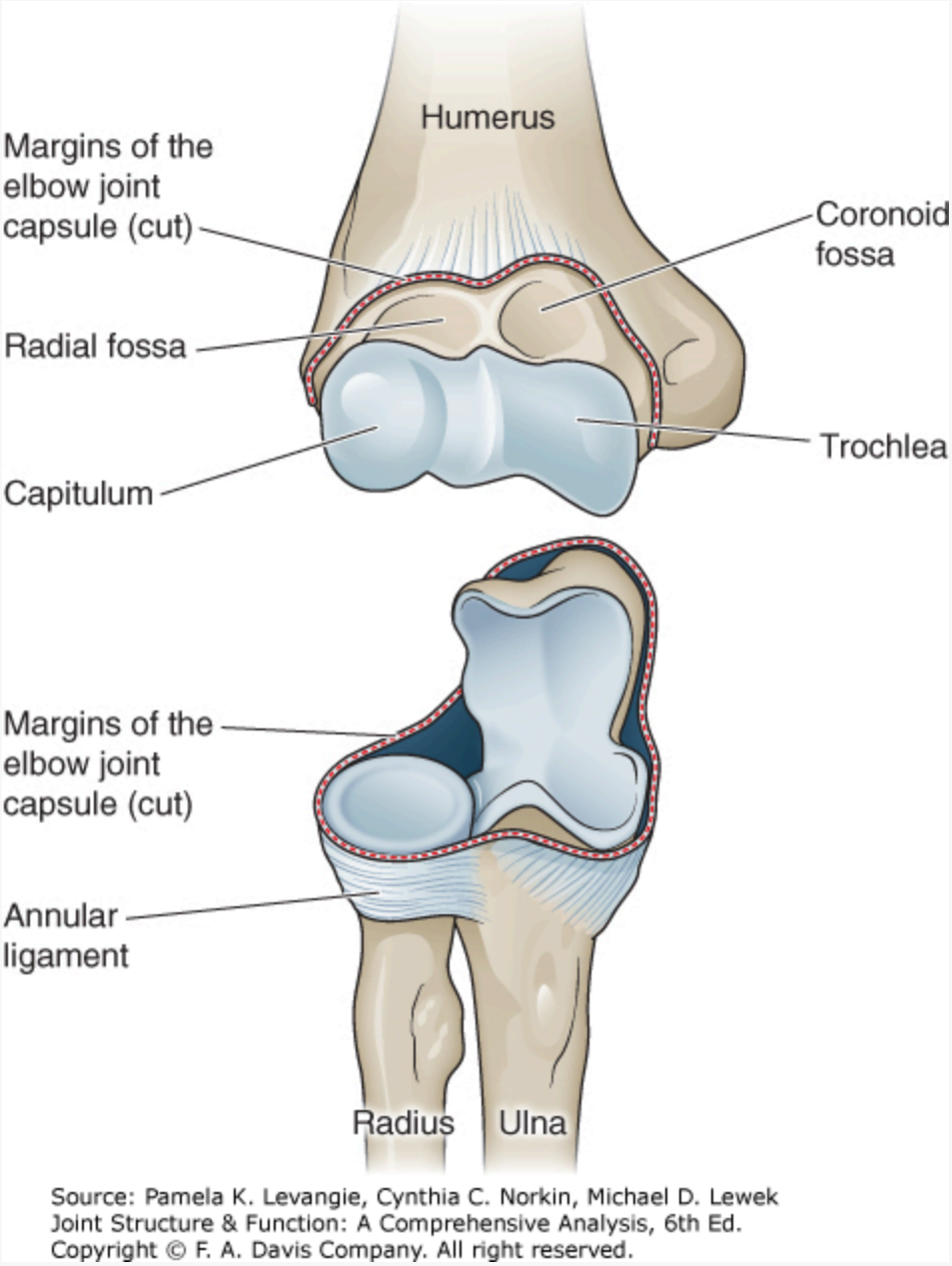
Joint Capsule

The humeroulnar, humeroradial, and superior radioulnar joints are enclosed in a single joint capsule. As shown in Figure 8–8, the proximal humeral attachment of the capsule is just above the coronoid and radial fossae, whereas the capsule attaches distally to

the ulna along the margin of the coronoid process and blends with the proximal border of the annular ligament. Medially and laterally, the capsule is continuous with the collateral ligaments.[3] Posteriorly, the capsule is attached to the humerus along the upper edge of the olecranon fossa and to the back of the medial epcondyle.[3] The capsule passes just below the annular ligament to attach to the posterior and inferior margins of the neck of the radius.[3]

FIGURE 8-8

The margins of the excised elbow joint capsule shown in dashed red encompass both the humeroradial and the humeroulnar articulations.



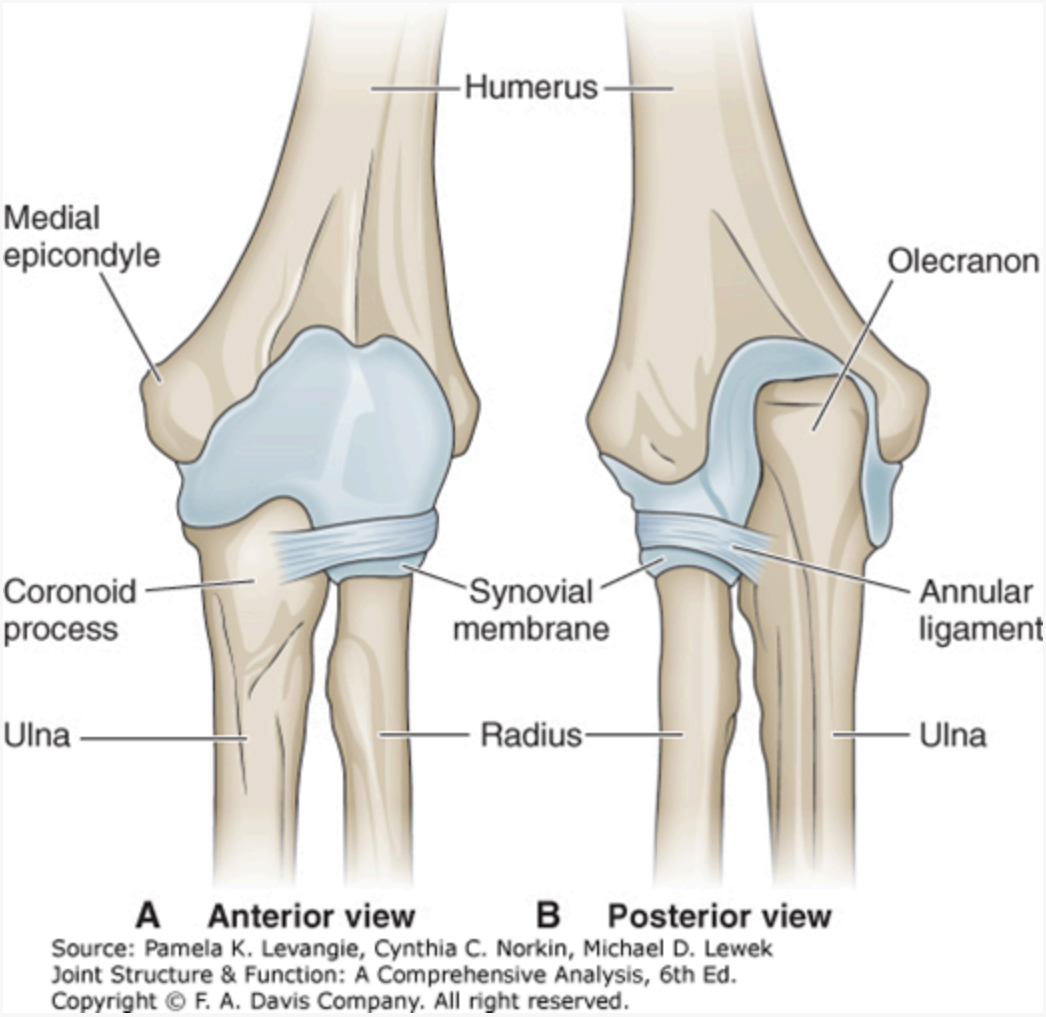
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The capsule is fairly large, loose, and weak anteriorly and posteriorly, and contains synovial folds that are able to expand to allow for a full range of elbow motion. Fat pads are located between the capsule and the synovial membrane adjacent to the olecranon, coronoid, and radial fossae.[3] Taterally and medially, the capsule is reinforced by the collateral ligaments.

The capsule's synovial membrane (Fig. 8–9) lines the coronoid, radial, and olecranon fossae. It also lines the flat medial trochlear surface and the lower part of the annular ligament. A triangular synovial fold inserted between the proximal radius and ulna partially divides the elbow joint into two joints.[3] Duparc and colleagues found that in the majority of joints examined in 50 adult cadavers, the triangular synovial fold was located at the proximal radioulnar joint near the junction of the annular ligament and the joint capsule.[10] The synovial folds varied from 1 to 4 mm in thickness and from 9 to 51 mm in length, and contained fat pads and nerve fibers.[10]

FIGURE 8-9

Synovium of the left elbow joint in an **A.** anterior view and **B.** a posterior view,



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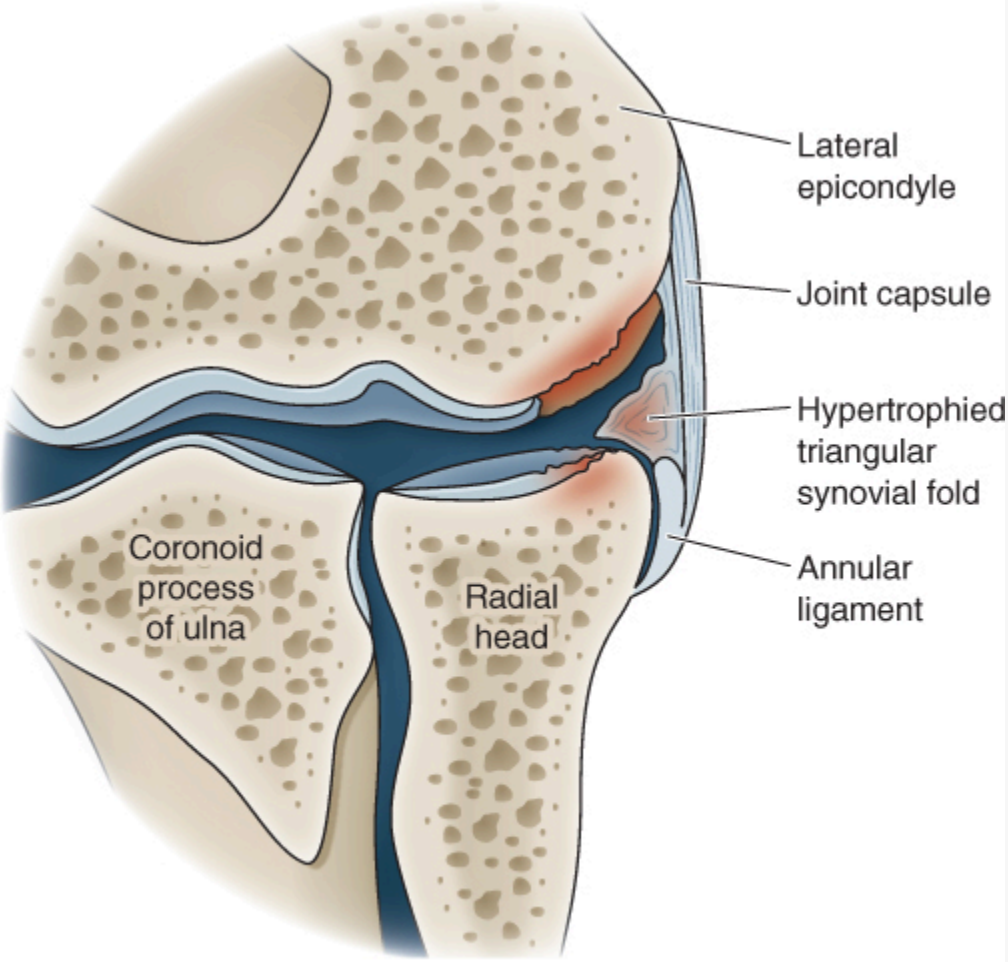
Expanded Concepts 8–1

Hypertrophied Synovial Folds

Hypertrophied triangular synovial folds have been identified as a source of pain in cases of lateral epicondylalgia (pain in the region of the lateral epicondyle).[10,11] Lateral elbow pain may arise from an irritation of the nerve fibers in a hypertrophied triangular synovial fold located between the radial head and ulna (inside the annular ligament) or at the junction of the annular ligament and joint capsule (Fig. 8–10).

FIGURE 8-10

In this cross-section of the elbow articulation, hypertrophy of the radial portion of the synovial fold can lead to entrapment in the humeroradial joint and potentially be a cause of lateral elbow pain and inflammatory or degenerative changes.



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Patient Application 8–1

Bill Garcia, a 40-year-old male, reports to physical therapy complaining of an insidious onset of right lateral elbow pain approximately 2 months ago. Pain is

worsened with repetitive gripping, prolonged computer work, and doing push-ups. Key objective findings are as follows:

- No pain with palpation along the lateral epicondyle or extensor tendons
- Pain with palpation along the humeroradial joint line
- Pain with active and passive wrist extension, gripping, and compression of the humeroradial joint
- Significant reduction of pain with distraction of humeroradial joint

This patient's presentation is a classic case of synovial fold syndrome of the elbow. Hypertrophied triangular synovial folds have been identified as a source of pain in cases of lateral epicondylalgia (pain in the region of the lateral epicondyle).[10,11] Lateral elbow pain may arise from an irritation of the nerve fibers in a hypertrophied triangular synovial fold located between the radial head and ulna (inside the annular ligament) or at the junction of the annular ligament and joint capsule (Fig. 8–10).

The patient's compression of the humeroradial joint as a result of repetitive wrist extension from computer work, gripping, and push-ups was enough to create inflammation of the synovial tissue that extended into the joint space. If not thoroughly examined, this presentation can be easily mistaken for a tendinopathy, which would require a very different treatment approach.

Ligaments

Most hinge joints in the body have collateral ligaments, and the elbow is no exception. Collateral ligaments are located on the medial and lateral sides of hinge joints to provide medial/lateral stability and to keep joint surfaces in apposition. The two main ligaments associated with the elbow joints are the medial (ulnar) and lateral (radial) collateral ligaments.

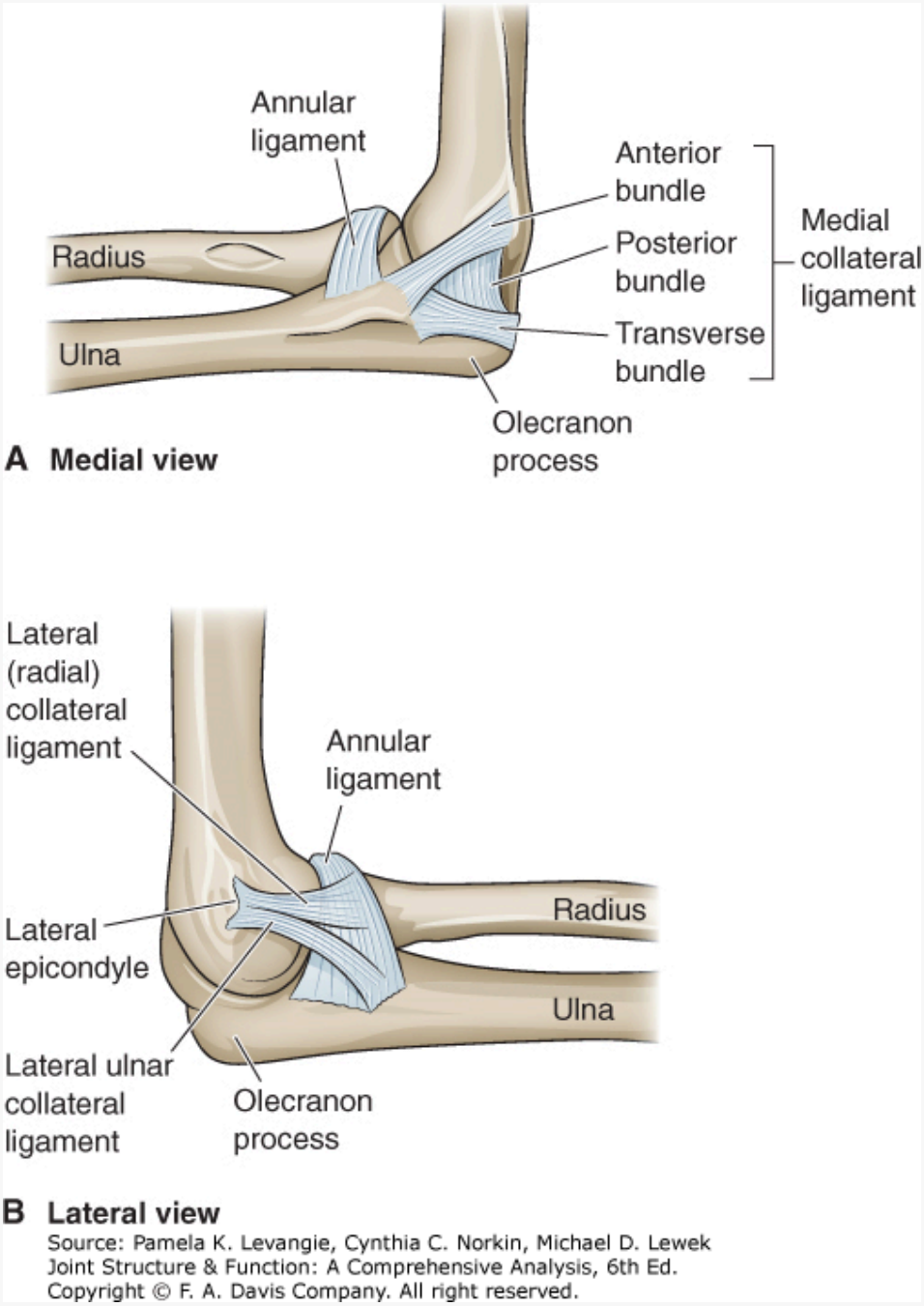
MEDIAL (ULNAR) COLLATERAL LIGAMENT

The **medial collateral ligament** consists of anterior, posterior, and transverse parts (**Fig. 8–11**).[12–17] The anterior and posterior parts may be referred to as either the **anterior** and **posterior oblique ligaments,** or simply **anterior** and **posterior bundles.** When the medial

collateral ligament is dissected free from other structures, the proximal enthesis of the ligament is described as a round area on the flat portion of the anterior and inferior aspect of the medial humeral epicondyle.[14,18] Milz and colleagues determined that the enthesis of the medial collateral ligament was essentially fused with the common flexor tendon as well as with part of the humeral articular cartilage where the ligament wraps around the articular cartilage of the edge of the trochlea.[18]

FIGURE 8-11

A. Three parts of the medial (ulnar) collateral ligament are shown on the medial aspect of the right elbow. The musculature and joint capsule have been removed to show the ligaments' attachments. **B.** The lateral collateral ligament complex includes the lateral (radial) collateral ligament, lateral ulnar collateral ligament, and annular ligament. The musculature and the joint capsule have been removed to show the ligaments' attachments.



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The distal enthesis of the medial collateral ligament on the coronoid process of the ulna is broad proximally and tapers distally, with fibers of the ligament crossing the coronoid process and inserting further distally on the proximal and medial ulna.[14] This type of spread-out insertion at an enthesis is also a mechanism for distribution of stress.[19]

Mechanoreceptors (Golgi organs, Ruffini terminals, Pacini corpuscles, and free nerve endings) are densely distributed near the ligament's humeral and ulnar attachments.[20]

The anterior bundle of the medial collateral ligament is considered to be the primary ligamentous restraint to valgus stress from 20° to 120° of elbow flexion.[21–23] Callaway and colleagues described the anterior bundle as consisting of anterior and posterior bands that tighten in a reciprocal manner as the elbow flexes and extends.[13] Along with the anterior bundle, the anterior capsule and radial head also make contributions to medial joint stability, but these structures fail to provide stability against valgus stress when the anterior bundle is cut.[24] The posterior bundle of the medial collateral ligament is not as distinct as the anterior bundle, and its fibers may blend with the fibers from the medial portion of the joint capsule. The posterior bundle extends from the posterior aspect of the medial epicondyle of the humerus to attach to the coronoid and olecranon processes of the ulna. The posterior bundle limits elbow extension but plays a less significant role than the anterior bundle in providing valgus stability for the elbow.[21–23] In Figure 8–11A, one can see that the oblique (transverse) fibers of the medial collateral ligament extend between the olecranon and ulnar coronoid processes. This portion of the ligament, also known as Cooper's ligament, appears to contribute little to valgus stability, but may help to keep the joint surfaces in approximation.



Foundational Concepts 8–1

Functional Summary for Medial (Ulnar) Collateral Ligament

1. Stabilizes against valgus torques at the medial elbow[13,21-26]
2. Limits extension at the end of the elbow extension ROM[15,17]

3. Guides joint motion throughout flexion ROM[15]
4. Provides some resistance to longitudinal distraction of joint surfaces.

LATERAL COLLATERAL LIGAMENTOUS COMPLEX

The **lateral collateral ligament complex** includes the **lateral (radial) collateral ligament,** the **lateral ulnar collateral ligament,**[15,27-29] and the **annular ligament** (Fig. 8–11B).[29] The lateral radial collateral ligament is a fan-shaped structure that extends from the inferior aspect of the lateral epicondyle of the humerus to merge with the annular ligament that encircles the head of the radius. Ligamentous tissue extending from the lateral humeral epicondyle to the lateral aspect of the ulna and the annular ligament is referred to as the lateral ulnar collateral ligament.[17] This ligament adheres closely to the supinator, extensor, and anconeus muscles that lie just posterior to the lateral collateral ligament.[30]



Expanded Concepts 8–2

Effect of Forearm and Elbow Position on Posterolateral Elbow Stability

Milz found that the entheses of the lateral collateral ligament complex and the common extensor tendon were fused at the lateral epicondyle.[18] In a study of cadaveric specimens, Wavreille determined that the positions of the forearm and the elbow affect the length of the lateral collateral ligaments and, therefore, may affect the posterolateral stability of the elbow.[31] For example, when the forearm was in neutral pronation/supination, the lateral collateral ligament was longest between 0° and 30° and at 90° of flexion. When the forearm was in full pronation, the results differed. In contrast to the effects of forearm rotation on the lateral collateral ligament, forearm rotation had little effect on the length of the lateral ulnar collateral ligament.[31]

Certain functions are attributed to individual ligaments in the lateral collateral ligament

complex, whereas other functions are thought to be attributable to the entire complex. The lateral radial collateral ligament provides reinforcement for the humeroradial articulation, offers some protection against varus stress in some positions of the elbow, and assists in providing resistance to longitudinal distraction of the joint surfaces.[32] Some fibers of the lateral radial collateral ligament remain taut throughout the flexion ROM when either a varus or a valgus moment is applied.[17] O'Driscoll described the lateral radial collateral ligament as a key structure that is always disrupted in elbow dislocations.[33]

Olsen and colleagues concluded that the lateral radial collateral ligament is the primary soft tissue restraint, whereas the lateral ulnar collateral ligament and the annular ligament are secondary restraints to combined forced varus and supination stresses and forced valgus stress.[34] Others found that the lateral ulnar collateral ligament has the potential for assisting the lateral radial collateral ligament in resisting varus stress and in providing lateral support to the elbow joint.[30,34] Kim and associates suggested that the lateral ulnar collateral ligament is not a static stabilizer, but acts as a dynamic stabilizer together with related muscles.[35]



Foundational Concepts 8–2

Functional Summary for Lateral Collateral Ligament Complex

1. Stabilizes elbow against varus torque[28,29,32,36,37]
2. Stabilizes against combined varus and supination torques[28,29,36,37]
3. Reinforces humeroradial joint and helps provide some resistance to longitudinal distraction of the articulating surfaces[32]
4. Stabilizes radial head, thus providing a stable base for rotation[37]

5. Maintains posterolateral rotatory stability[29,30,34,38-40]
6. Prevents subluxation of humeroulnar joint by securing ulna to humerus
7. Prevents the forearm from rotating off of the humerus in valgus and supination during flexion from the fully extended position.



Patient Application 8–2

Joshua Rodes, a 27-year-old man, reports to physical therapy complaining of pain and weakness in his right upper extremity and apprehension with using it during activities such as pulling weeds, pushing a stroller, and lifting weights. He feels that his elbow is "unstable." In fact, he has felt his elbow "shift" a couple times during these activities. While giving his history, the patient explained how he fell on an outstretched hand while playing soccer 4 months ago, an injury termed a FOOSH (fall on outstretched hand). This fall resulted in immediate pain and his elbow "looking funny." The patient attempted to straighten his elbow, resulting in a "clunk" and his elbow returning back to a normal appearance. The patient did not seek further medical intervention and managed with rest and ice. Upon testing in physical therapy, the patient presented with positive findings of lateral ligament complex laxity and elbow instability and was referred for further confirmation with imaging and surgical consultation.

The lateral collateral ligament and the medial collateral ligament are attached to the joint capsule, with free nerve endings and mechanoreceptors distributed near the humeral and ulnar capsular attachments.[20] Isolated tears of the lateral collateral ligament are uncommon, but chronic insufficiency may lead to symptomatic posterolateral joint subluxation.[40] Complete disruption of the lateral collateral ligament complex is most often seen in fracture dislocations and fractures involving the coronoid or radial head.[41] A patient presenting with painful clicking or locking of the elbow could have posterolateral instability, which would require further examination and stress radiographs.[33]

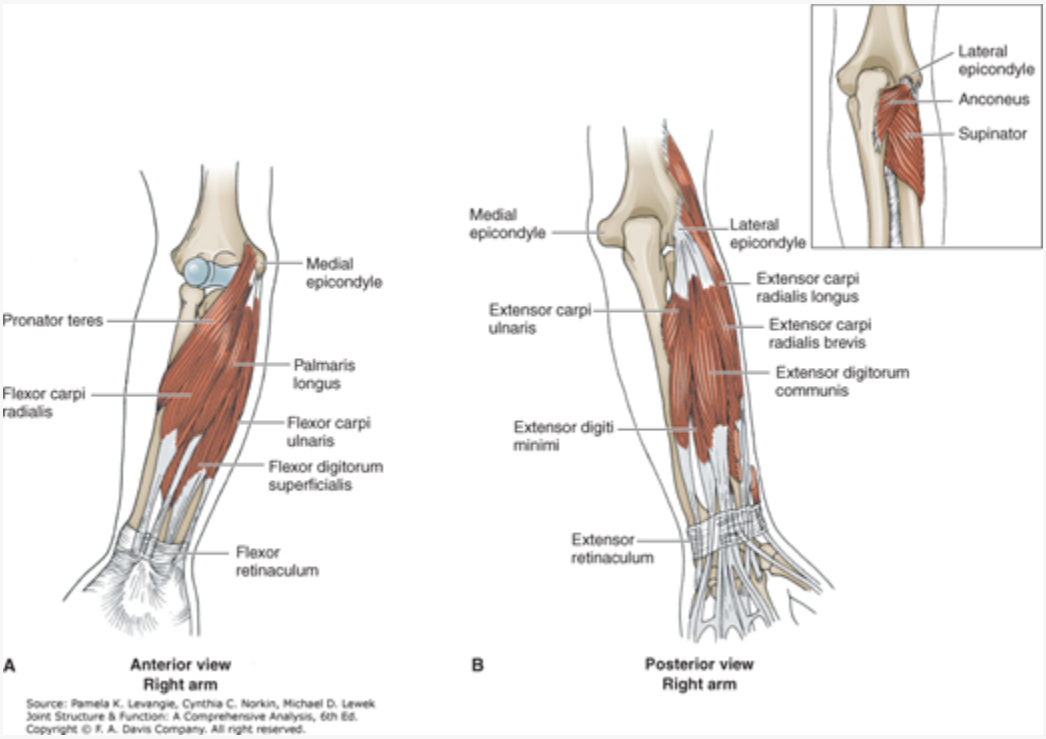
Muscles

Nine muscles cross the anterior aspect of the elbow joint, but only three of these muscles (the brachialis, biceps brachii, and brachioradialis) have primary functions at

the elbow joint. The **supinator** and **pronator teres** have major functions, instead, at the radioulnar joints. The remaining four muscles (**flexor carpi radialis, flexor carpi ulnaris, flexor digitorum superficialis,** and **palmaris longus**) have primary functions at the wrist, hand, and fingers, and are considered to be weak flexors of the elbow (Fig. 8–12A).

FIGURE 8-12

Right arm. **A.** Insertion of flexor muscles on the medial epicondyle of the humerus. **B.** Insertion of extensor muscles on the lateral epicondyle of the humerus.



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The major flexors of the elbow are the **brachialis,** the **biceps brachii,** and the **brachioradialis.** The brachialis muscle arises from the anterior surface of the lower portion of the humeral shaft and attaches by a thick, broad tendon to the ulnar tuberosity and coronoid process. The biceps brachii arises from two heads. The short

head arises as a thick, flat tendon from the coracoid process of the scapula, and the long head arises as a long, narrow tendon from the scapula's supraglenoid tubercle. The muscle fibers unite in the middle of the upper arm to attach by way of the strong flattened tendon on the tuberosity of the radius. Other fibers of the biceps brachii insert into the bicipital aponeurosis that extends medially to blend with the fascia that lies over the forearm flexors.[3] The brachioradialis muscle arises from the lateral supracondylar ridge of the humerus and attaches distally just proximal to the styloid process of the radius near the wrist.

Passing posterior to the elbow joint, the two extensors of the elbow are the triceps brachii and the anconeus. The **triceps brachii** has long, medial, and lateral heads. The long head arises from the infraglenoid tubercle of the scapula by a flattened tendon that blends with the glenohumeral joint capsule, thus crossing both the glenohumeral and the elbow joints. The medial head arises from the entire posterior surface of the humerus, whereas the lateral head arises from only a narrow ridge on the posterior humeral surface. Unlike the long head, neither crosses the glenohumeral joint. The three heads insert via a common tendon into the olecranon process. The **anconeus** is a small triangular muscle that arises from the posterior surface of the lateral epicondyle of the humerus and extends medially to attach to the lateral aspect of the olecranon process and the adjacent proximal ulna.[3]

A number of muscles with primary actions at the wrist and fingers insert into the lateral humeral epicondyle, along with the anconeus, by way of a common extensor tendon. Because these muscles cross the elbow, they can potentially act on the elbow, but their effect is predominantly compressive (stabilization). These muscles include the **extensor**

carpi radialis longus, extensor carpi radialis brevis, extensor digitorum communis, extensor carpi ulnaris, and extensor digiti minimi (see Fig. 8–12B).



Foundational Concepts 8–3

Muscles and Tendons of the Elbow Complex

The extensor carpi radialis longus and brevis and the extensor carpi ulnaris are active during gripping, hammering, and sawing tasks. The repetitive pull of these muscles during these activities could injure the actual muscle belly or common extensor tendon, either in its substance or at the enthesis. Pathology at the enthesis (enthesopathies) are commonly seen in tennis elbow and jumper's knee.[42] If a patient reports numerous episodes of a consistent presentation of pain over a number of years, one may suspect a chronic condition that results from degenerative changes affecting the tendon.

The types of changes that might be seen in either a patient's tendon or muscle are summarized in Table 8–1. A shift in understanding of the disease process involved in tennis elbow has led to the diagnosis of epicondylitis being abandoned by some and replaced with a diagnosis of epicondylalgia or insertional tendinopathy.[43–45] The basis for the shift in terminology is that the observed changes appear to be more suggestive of a degenerative process than of a simple inflammatory process. According to Benjamin, fibrocartilaginous tendon insertional problems rarely show evidence of inflammation; instead, tendons show thinning, disruption of collagen fibers, increased vascularity and cellularity, granulation tissue, and increased proteoglycan content in the extracellular matrix.[19] Faro and Wolf suggested that a failed reparative process might be the cause of the tissue changes, but indicated that our understanding of the exact disease process involved is still incomplete.[44]

Imaging studies such as ultrasound and magnetic resonance imaging (MRI) have been used to identify signs of edema, thickening, and tears in common extensor tendons and peritenon edema in patients with lateral elbow pain.[46] Although ultrasound imaging of patients with lateral epicondylalgia shows evidence of degenerative changes such as tendon thickening, neovascularization, and disruption of fibrils and intrasubstance tears, similar findings are present in approximately 50% of healthy asymptomatic age-matched individuals.[47]

Table 8-1

Tennis Elbow: Changes in Tendons and Muscles

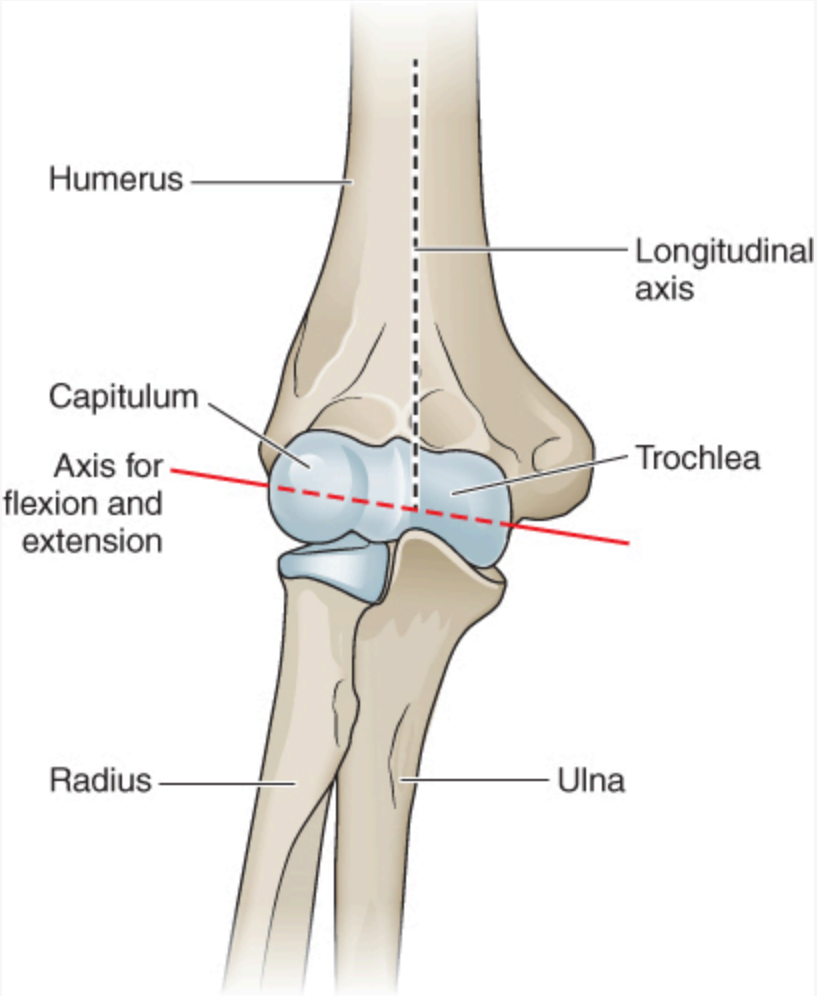
Function

Axis of Motion

Traditionally, the axis for flexion and extension has been described as a relatively fixed line that passes horizontally through the center of the trochlea and capitulum and bisects the longitudinal axis of the shaft of the humerus (Fig. 8–13).[48,49] However, some studies have found that the axis is not as fixed as previously thought.[50–52]

FIGURE 8-13

Right arm. The axis of motion for elbow flexion/extension is centered in the middle of the trochlea on a line that intersects the longitudinal (anatomic) axis of the humerus.



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An exact determination of the axis of motion at the elbow is important because of the need to position elbow prostheses in such a way that they correctly mimic elbow joint motion. In the past, elbow prostheses that were modeled as pure hinge devices often became loose during motion. Variations found in the inclination of the instantaneous axis support the hypothesis that activity of the various muscles may influence the pattern of motion during active flexion and that differences in contours of the joint surfaces may explain inter-individual differences. Ericson and coworkers found that the

orientation of the instantaneous axes varied from 1° to more than 14° between subjects, more in the frontal plane than in the transverse plane.[50] However, the inclination of the average axis across subjects in the transverse plane differed little from a line through the centers of the trochlea and capitulum. Bottlang and colleagues similarly found in their research that all instantaneous axes of rotation nearly intersected on the medial facet of the trochlea.[52]

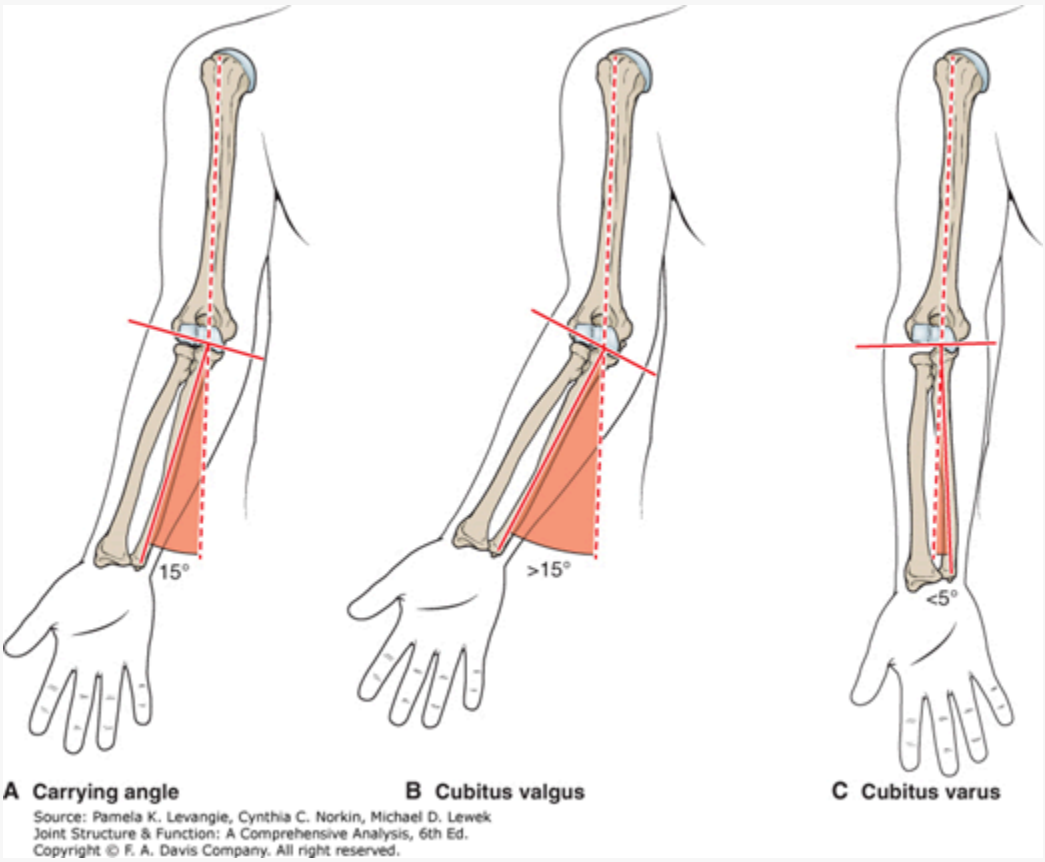
LONG AXES OF THE HUMERUS AND FOREARM (CARRYING ANGLE OF THE ELBOW)

When the upper extremity is in the anatomical position (shoulder in external rotation, elbow in extension, and forearm fully supinated), the long axes of the humerus and forearm form an acute angle laterally at the elbow (Fig. 8–14). The angulation occurs because the medial aspect of the trochlea extends more distally than does the lateral aspect, with the capitulum slightly above the trochlea (see Fig. 8–13). This angulation is called the **carrying angle** and is typically around 15° (Fig 8–14A), but may vary from about 8° to 15°. When this carrying angle is atypical and exceeds 15°, the condition is referred to as **cubitus valgus** (Fig. 8–14B), whereas a reduced (< 5°) or reversed carrying angle is referred to as **cubitus varus** (Fig. 8–14C). If the angulation is a result of a postinjury complication (i.e., supracondylar fracture), it can be referred to as a "gunstock deformity."[53]

FIGURE 8-14

Angulation at the elbow. **A.** The forearm typically lies slightly lateral to the humerus when the elbow is fully extended in the anatomic position; this angulation is referred to as the carrying angle of the

elbow. **B.** When the carrying angle is excessive (>15°), the angulation is referred to as cubitus valgus. **C.** When the carrying angle at the elbow is reduced (<5°) or even reversed, it is referred to as cubitus varus.



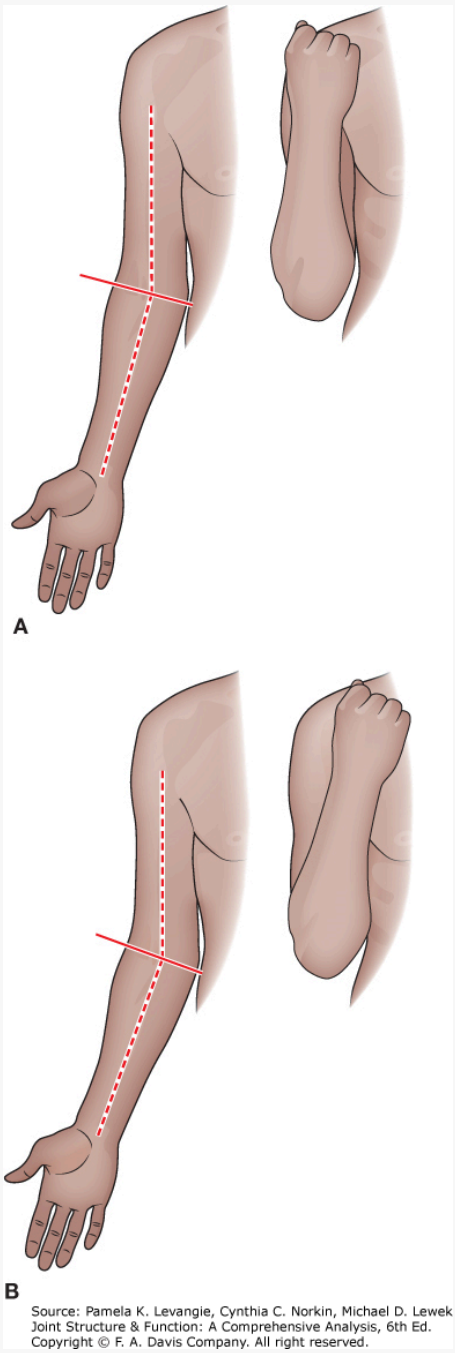
Khare and colleagues found that in the first year of life there is no difference in the carrying angle between males and females.[54] However, the mean carrying angles have been found to be significantly larger in female children than in male children in the same age group from age 1 year on.[54–57]

Although there is a normal angulation between the humerus and the forearm in elbow extension, the obliquity of the elbow axis results in a change in the relationship of the forearm and humerus with elbow flexion (Fig. 8–15). With a typical inclination of the

elbow joint axis, the ulna is guided progressively medially from extension to flexion so that in full flexion, the forearm comes to rest in the same plane as the humerus (Fig. 8–15A).[9] With an increase in the elbow joint axis (cubitus valgus), the forearm will come to rest medial to the humerus in full flexion (Fig. 8–15B), or may come to rest lateral to the humerus with more extreme degrees of cubitus varus.[9,48]

FIGURE 8-15

Position of the right forearm in passive flexion. **A.** In the most common inclination of the elbow joint axis (15°), the ulna is guided progressively medially from extension to flexion so that in full flexion the forearm comes to rest in the same plane as the humerus. **B.** When the inclination of the elbow joint axis is increased (e.g., 20°), the forearm comes to rest slightly medially to the humerus in passive flexion.



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Mobility and Stability

A number of factors determine the amount of motion that is available at the elbow joint. These factors include the type of motion (active or passive), the position of the forearm (pronation/supination), body mass index (BMI), and the position of the

shoulder. The range of active flexion at the elbow is usually less than the range of passive motion because the bulk of the contracting flexors on the anterior surface of the humerus may interfere with the approximation of the forearm with the humerus. The active ROM for elbow flexion with the forearm supinated is typically considered to be from about 135° to 145°, whereas the range for passive flexion is between 150° and 160°.[9] The position of the forearm also affects the flexion ROM. When the forearm is either in pronation or in neutral supination/pronation, the ROM is less than it is when the forearm is supinated. A BMI that is high (indicating overweight) may also be a factor limiting elbow ROM.

The position of the shoulder may affect the ROM available to the elbow joint because of the influence of the two- or multi-joint biceps brachii and the triceps brachii. If a full ROM is attempted at the shoulder and the elbow simultaneously, these muscles may become actively or passively insufficient.



Foundational Concepts 8–4

Effects of Two-Joint Muscles on Elbow ROM and Function

Two- or multi-joint muscles do not have sufficient length (or ability to generate active tension) to allow (or complete) a simultaneous full ROM at all joints crossed. In Figure 8–16, the woman is attempting to complete elbow flexion as she performs a chin-up to the bar. With simultaneous shoulder and elbow flexion, the long head of the two-joint triceps is lengthened over both joints, whereas the three-joint biceps is shortened across the flexed shoulder, flexed elbow, and supinated forearm. The ability of the biceps to actively complete the elbow flexion ROM is limited by poor active and passive tension generation (active insufficiency), whereas the lengthened state of the triceps is simultaneously providing passive resistance to further elbow flexion.

FIGURE 8-16

Pull-up. In a pull-up exercise, the long head of the biceps brachii is attempting to shorten over the flexed shoulder while both heads are attempting to shorten over the flexed elbow, and supinated forearm. The muscle may not be able to shorten sufficiently or create enough tension to complete the available range of motion at all three joints. At the same time, the long head of the triceps brachii is being stretched over the shoulder and the elbow, potentially providing passive resistance that the biceps must overcome.



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Other factors that limit the ROM but help to provide stability for the elbow are the

configuration of the joint surfaces, the ligaments, and the joint capsule. The elbow has inherent articular stability at the extremes of extension and flexion.[22,23,32] In full extension, the humeroulnar joint is in a close-packed position. Bony contact of the olecranon process in the olecranon fossa limits the end of the extension range, and the configuration of the joint structures helps provide valgus and varus stability. The bony components provide 50% of the resistance to varus stress in full extension, and the lateral collateral complex and joint capsule provide the other 50% of the resistance.[32] Resistance to joint distraction in the extended position is provided entirely by soft tissue structures. The anterior portion of the joint capsule provides the majority of the resistance to anterior displacement of the distal humerus out of the trochlear notch; the medial and lateral collateral ligaments contribute only slightly.[23]

Approximation of the coronoid process with the coronoid fossa and the rim of the radial head in the radial fossa limit extremes of flexion. In 90° of flexion, the anterior part of the medial collateral ligament provides the primary resistance to both distraction and valgus stress. If the anterior portion of the medial collateral ligament becomes lax through overstretching, medial instability will result when the elbow is in flexed positions. The majority of the resistance to varus stress when the elbow is flexed to 90° is provided by the osseous structures of the joint; only a slight amount is provided by the lateral collateral ligament and the joint capsule. Active and passive tension in the muscles that cross the elbow, as is true of most muscles, contributes to stabilization of the joint through compression. Co-contractions of the flexor and extensor muscles at the elbow provide a stable base for forceful motions of the wrist and fingers because several of the muscles that cross the wrist and fingers also cross the elbow joint.

Swelling or pain may limit the range of elbow motion. McGuigan and Bookout investigated the effects of intra-articular fluid on ROM. They found that the flexion arc of motion decreased 2.1° for every millimeter of injected fluid.[58]

Muscle Action

FLEXORS

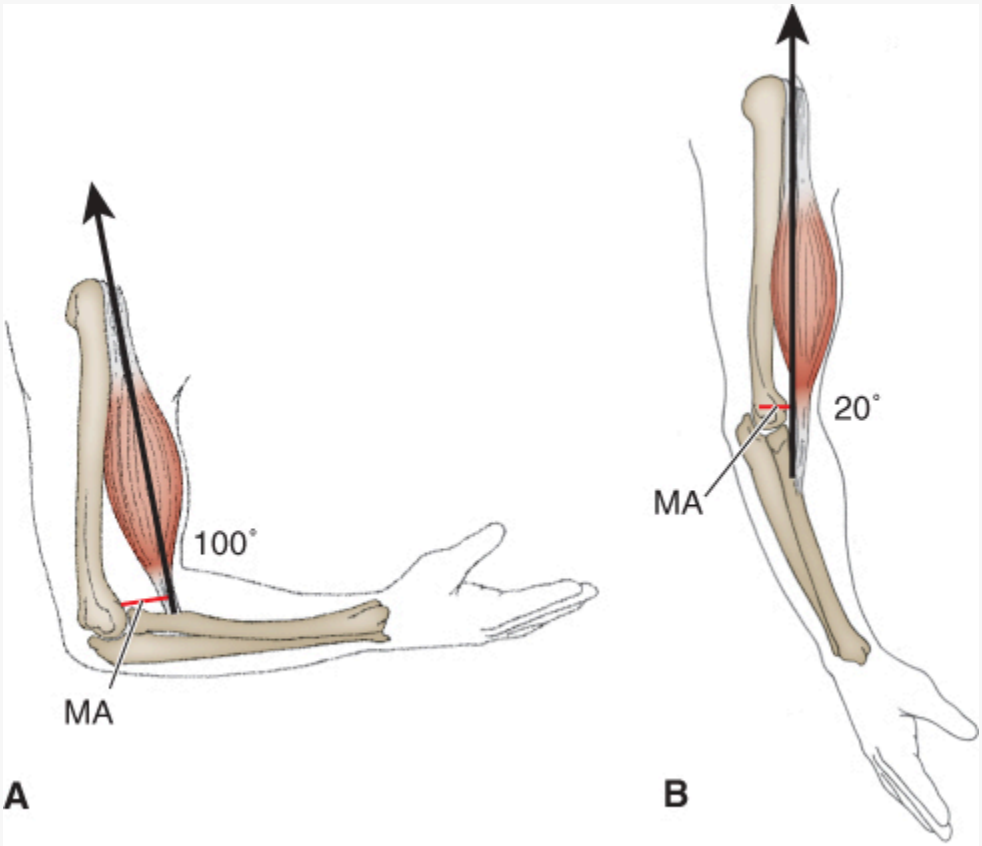
The brachialis is considered a mobility muscle because its insertion is close to the elbow joint axis, an attachment that favors production of a large arc of motion at the distal segment. The muscle has a sustantial tension generation potential in that it has a large physiological cross-sectional area and a large work capacity (volume).[1] The moment arm of the brachialis is greatest at slightly more than 100° of elbow flexion.[59] A relatively large moment arm favors torque generation, but the ability of the muscle to generate muscle force (Fms) is also affected by other factors (e.g., speed and type of contraction). Because the brachialis's attachments are on the humerus and the ulna, it is unaffected by changes in shoulder or forearm position. According to electromyography studies, the brachialis muscle works in flexion of the elbow with and without resistance and during slow and fast motions.[60]

The biceps brachii, like the brachialis, is considered to be a mobility muscle because of its insertion close to the elbow joint axis that favors a large arch of distal movement. The long head of the biceps brachii has the largest volume among the flexors, but the muscle has a relatively small physiological cross-sectional area.[1] The moment arm of the biceps is greatest between 80° and 100° of elbow flexion, giving the biceps a strong torque generation potential in this range (Fig. 8–17A).[59] The moment arm of the biceps is small as the elbow approaches full extension, with most of the muscle force applied parallel to the forearm (translation) and toward the joint (compression; Fig. 8–17B).

Therefore, the biceps is less effective as an elbow flexor when the elbow is fully extended than when the elbow is flexed to 100°. When the elbow is flexed beyond 100°, the translatory component of the muscle force is directed away from the elbow joint and therefore acts as a distracting or dislocating force.

FIGURE 8-17

A. Moment arm (MA) of the biceps brachii is maximal at 100° of elbow flexion. B. As the elbow approaches full extension, the moment arm (MA) of the biceps is relatively small, and most of the muscle force is toward joint compression.



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The biceps brachii is active during unresisted elbow flexion if the forearm is supinated or in neutral pronation/supination, but tends *not* to be active in unresisted elbow flexion when the forearm is pronated. However, when the magnitude of the resistance increases much beyond limb weight, the biceps is active in all positions of the forearm.[61]

The **brachioradialis** is inserted at a distance from the joint axis, with the largest component of muscle force going toward compression of the joint surfaces and, hence, toward stability. The brachioradialis has a relatively small mean physiological cross-sectional area (1.2 cm), but a relatively large average peak moment arm in comparison with other elbow flexors, with the peak moment arm for the brachioradialis occurring between 100° and 120° of elbow flexion.[59] The brachioradialis does not cross the shoulder and, therefore, is unaffected by the position of the shoulder. The brachioradialis shows no electrical activity during eccentric flexor activity when the motion is performed slowly with the forearm supinated, or during slow, unresisted, concentric elbow flexion.[62] When the speed of the motion is increased, the brachioradialis shows moderate activity if a load is applied and the forearm is either in a neutral forearm position or in full pronation.[60,63]

The pronator teres and other muscles that cross the elbow joint (palmaris longus, flexor digitorum superficialis, flexor carpi radialis, and flexor carpi ulnaris) are weak elbow flexors with primary actions at the radioulnar and wrist joints.[3]

EXTENSORS

The effectiveness of the triceps brachii as a whole is unaffected by changes in position of the forearm because the triceps attaches to the ulna and not the radius. As already

noted, the influence of the long head of the triceps is affected by changing shoulder joint positions because the long head crosses both the shoulder and the elbow. The long head's ability to produce force will diminish when full elbow extension is attempted with simultaneous shoulder hyperextension because the muscle is shortened over both joints at the same time, thus is approaching active insufficiency.

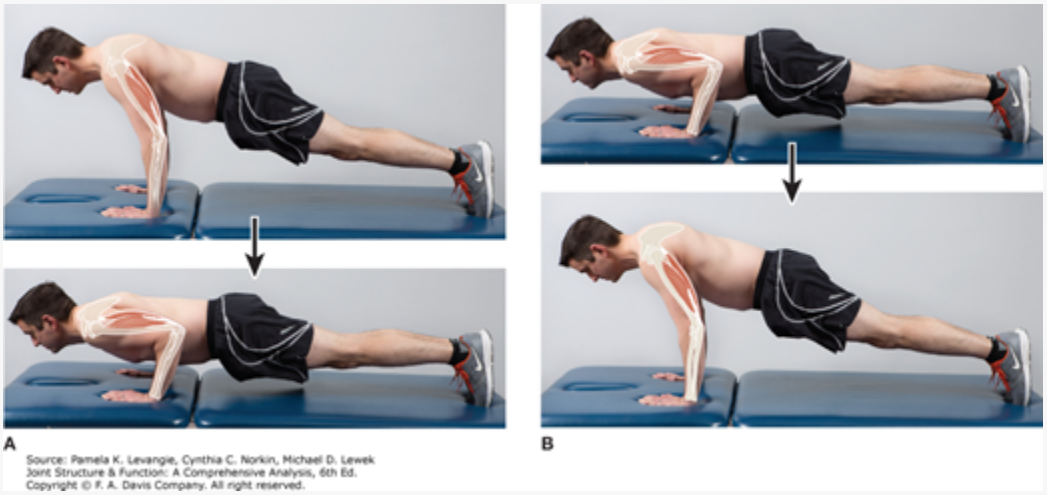
The medial and lateral heads of the triceps are one-joint muscles so are not affected by the position of the shoulder. The medial head is active in unresisted active elbow extension, but all three heads are active when heavy resistance is given to extension or when quick extension of the elbow is attempted, even in a gravity-assisted position.[60] Although maximum isometric torque has been observed at 90° of elbow flexion, the total amount of extensor torque generated at 90° varies with the position of the shoulder and the body.[64–66]

The variations in the ability of the triceps to produce effective force is most often represented by the function of the muscles during a push-up exercise (as the biceps function is during a chin-up; Fig. 8–18). The triceps are active eccentrically to control elbow flexion produced by the body weight as the body is lowered to the ground in a push-up (Fig. 8–18A). The triceps are active concentrically to extend the elbow once again against the resistance of the body weight (Fig. 8–18B). Throughout the push-up exercise, the relative ability of the long head of the triceps to generate force is maintained. When the elbow is extended (triceps shortened at the elbow), the shoulder is flexed (long head of the triceps is lengthened over the shoulder); when the elbow is flexed (triceps lengthened), the shoulder is extended (long head of the triceps shortened over the shoulder). In this way, active insufficiency of the long head is

avoided.

FIGURE 8-18

Triceps brachii activity during a push-up. **A.** The triceps work eccentrically to control the elbow flexion that occurs due to body weight during the lowering phase of a push-up. **B.** As the elbows extend against the resistance of body weight, the triceps work concentrically to return to the start position in the push-up.



Zhang and Nuber[67] found that in voluntary isometric extension, the uniarticular lateral and medial heads of the triceps provide 70% to 90% of the total elbow extension torque. The anconeus muscle contributes about 15%, whereas the biarticular long head of the triceps contributes significantly less. The authors of the study concluded that this was an example of the fact that the central nervous system selectively recruits uniarticular muscles before two-joint muscles to complete a task unless the need for substantial force is anticipated.

The triceps may be active during activities requiring stabilization of the elbow. For

example, it acts as a synergist to prevent flexion of the elbow when the biceps is acting as a supinator. The other extensor of the elbow, the anconeus, assists in elbow extension and arguably acts as a stabilizer during activity.[68] Synergistic actions of both the elbow flexor and extensor muscles have been investigated during a variety of task demands and stresses. Some flexor muscle pairs, such as the brachialis and brachioradialis, and the extensor pairs of the anconeus and medial head of the triceps brachii, are coactivated in a similar manner for all stresses. Prodoehl and colleagues demonstrated that muscle activation patterns change with the force requirements of the task and the amount of available muscle force.[69]

Structure and Function of the Proximal and Distal Radioulnar Articulations

Structure

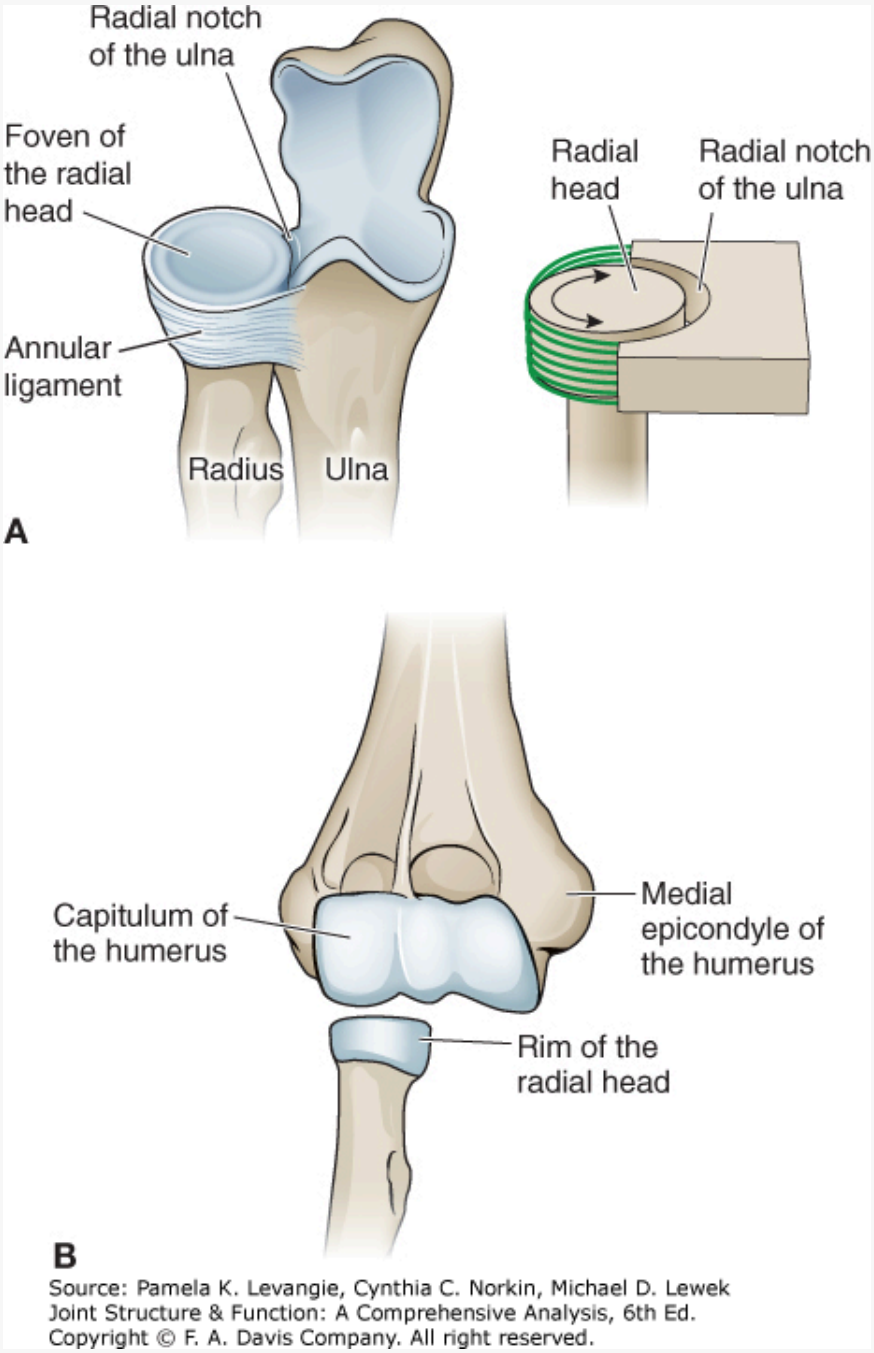
Proximal (Superior) Radioulnar Joint

The articulating surfaces of the proximal radioulnar joint include the radial notch of the ulna, the annular ligament, rim and fovea of the head of the radius, and the humeral capitulum (Fig. 8–19). The **radial notch** of the ulna is located on the lateral aspect of the proximal ulna directly below the trochlear notch. The radial notch is concave and covered with articular cartilage. The nearly circular annular ligament is attached to the anterior and posterior edges of the notch. The rim of the radial head is hyaline cartilage–covered. The inner aspect of the annular ligament is lined with articular cartilage that is continuous with the cartilage lining of the radial notch, creating a fibro-osseous ring within which the head of the radius rotates (Fig. 8–19A). The capitulum and the proximal surface of the head of the radius (the fovea) are part both of the

proximal radioulnar joint (Fig. 8–19B) and the humeroradial (elbow) articulation; the humeroradial joint was already discussed.

FIGURE 8-19

The proximal radioulnar joint. **A.** One part of the proximal radioulnar joint is formed by the fibro-osseous ring of the annular ligament and radial notch of the ulna within which the radial head rotates. **B.** The joint also includes the articulation of the fovea (see **A.**) of the radial head with the capitulum of the distal humerus.



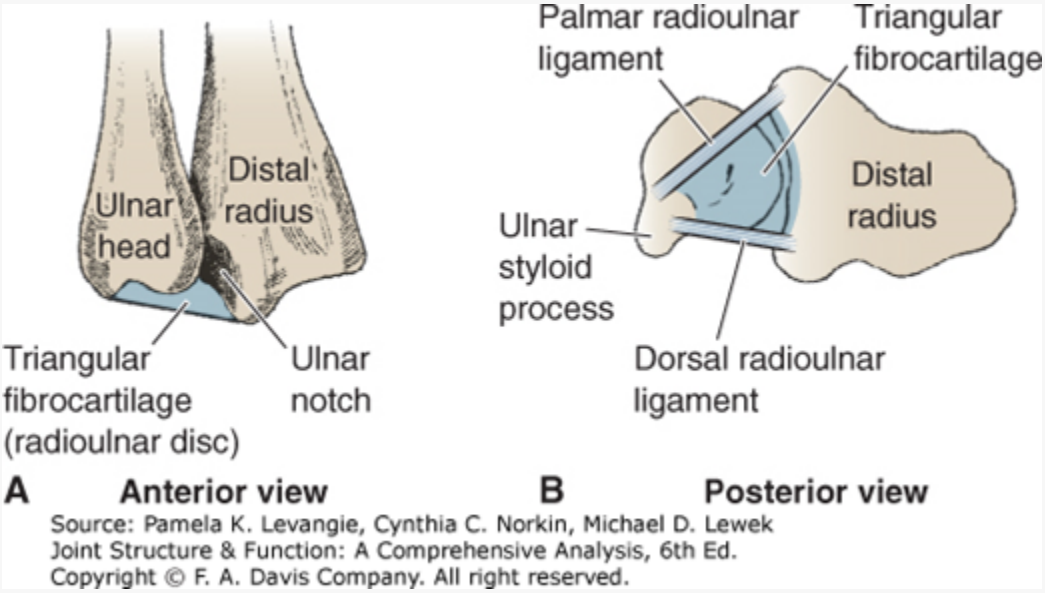
Distal (Inferior) Radioulnar Joint

The articulating surfaces of the distal radioulnar joint include the ulnar notch of the radius, the articular disc, and the head of the ulna (Fig. 8–20). The **ulnar notch** of the radius is located at the distal end of the radius. The radius of the curvature of the

concave ulnar notch is larger than that of the ulnar head. The **radioulnar disc** is commonly referred to as either the **triangular fibrocartilage (TFC)** because of its triangular shape, or as a part of the **triangular fibrocartilage complex (TFCC)** because of its extensive fibrous connections to the structures of the wrist (Fig. 8–20A).

FIGURE 8-20

Left distal radioulnar joint. **A.** An anterior view of the distal radioulnar joint shows the triangular fibrocartilage (radioulnar disc) in its normal position in a supinated left forearm. **B.** An inferior view of the distal radius and ulna shows how the triangular fibrocartilage (not actually transparent) covers the inferior aspect of the distal ulna. The palmar and dorsal radioulnar ligaments contribute to stability of the radioulnar articulation and the triangular fibrocartilage.



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The base of the triangular fibrocartilage is attached to the distal edge of the ulnar notch of the radius. The apex of the triangular fibrocartilage has one attachment to the fovea

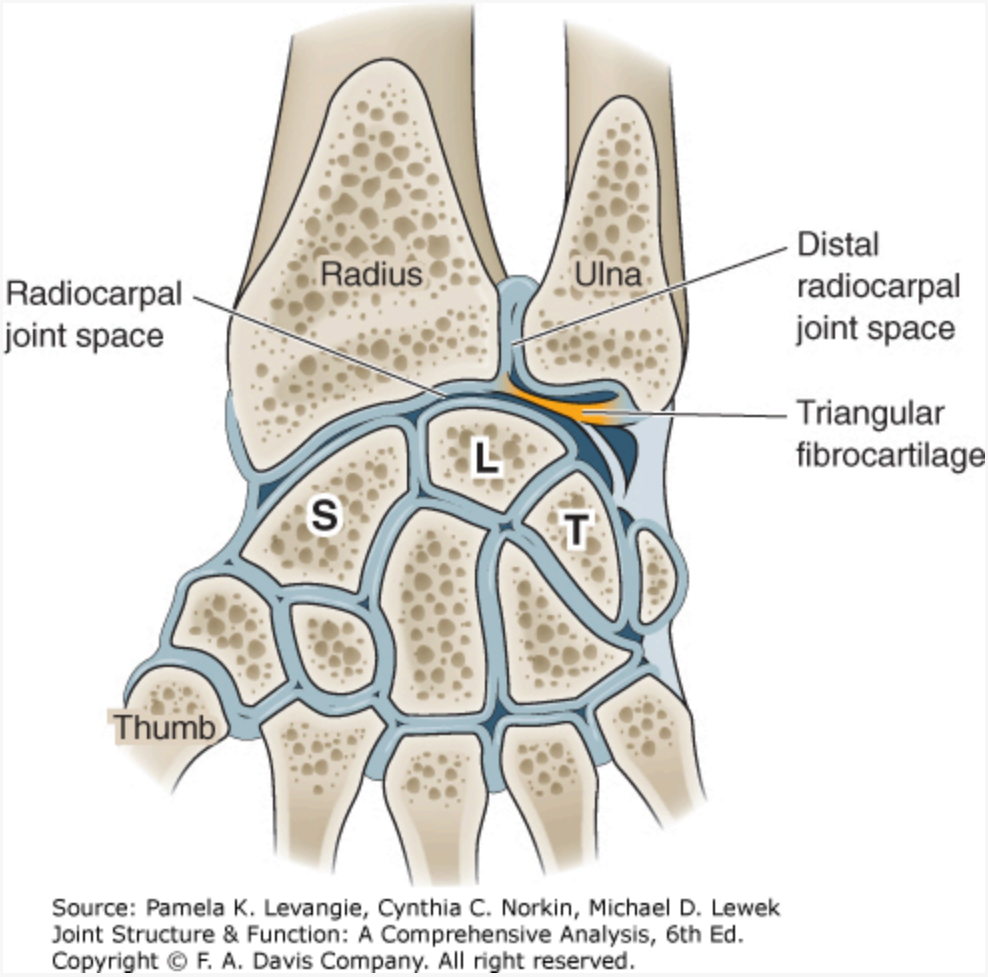
on the ulnar head and another to the base of the ulnar styloid process.[70] The triangular fibrocartilage has numerous attachments, leading to the common designation as the triangular fibrocartilage complex. It has been described as resembling a shelf whose medial border is embedded in a wedge of vascular connective tissue containing fine ligamentous bands that join the triangular fibrocartilage to the ulna and articular capsule.[71] Medially, the triangular fibrocartilage is continuous with the fibers of the ulnar collateral ligament that arises from the sides of the styloid process.[70] The margins of the triangular fibrocartilage are thickened, being thinner centrally and are either formed by or are integral parts of the distal **dorsal and palmar radioulnar ligaments** (Fig 8–20B).[72–74] The ligaments are firmly attached to the radius; the ulnar attachments are somewhat less firmly attached.[70] Chidgey and colleagues found in the majority of their cadaveric specimens that the central portion of the fibrocartilage was avascular in contrast to the peripheral area of the triangular fibrocartilage.[74] Clinically, this has led to the recommendation for repair of peripheral tears of the triangular fibrocartilage in the more commonly vascularized zone, whereas tears in the central and more commonly avascular region are simply debrided.[75] Ohmori and Azuma found free nerve endings in the ulnar side of the triangular fibrocartilage, particularly around the periphery. The authors suggested that, in view of their findings, the disc may be a source of wrist pain.[76]

The triangular fibrocartilage physically divides the distal radioulnar joint from the radiocarpal joint, each of which is anatomically separate (Fig. 8–21). Consequently, the triangular fibrocartilage actually has two articulating surfaces. The proximal (superior) surface of the triangular fibrocartilage articulates with the ulnar head at the distal radioulnar joint; its distal (inferior) surface articulates, with the carpal bones as part of

the radiocarpal joint.[70] Both the proximal and distal surfaces of the triangular fibrocartilage are concave to accommodate the convexity of the ulnar head superiorly and the convexity of the carpal bones distally.[70] The peripheral portions of both the proximal and distal triangular fibrocartilage surfaces are covered by synovium from their respective joint capsules.[73]

FIGURE 8-21

Triangular fibrocartilage. A cross-section through the right distal forearm and hand allows one to see that the triangular fibrocartilage divides and serves as a physical barrier between the distal radioulnar synovial joint and the radiocarpal synovial joint. The triangular fibrocartilage, therefore, is part of both the distal radioulnar and the radiocarpal articular surfaces. L, Lunate; S, scaphoid; T, triquetrum.



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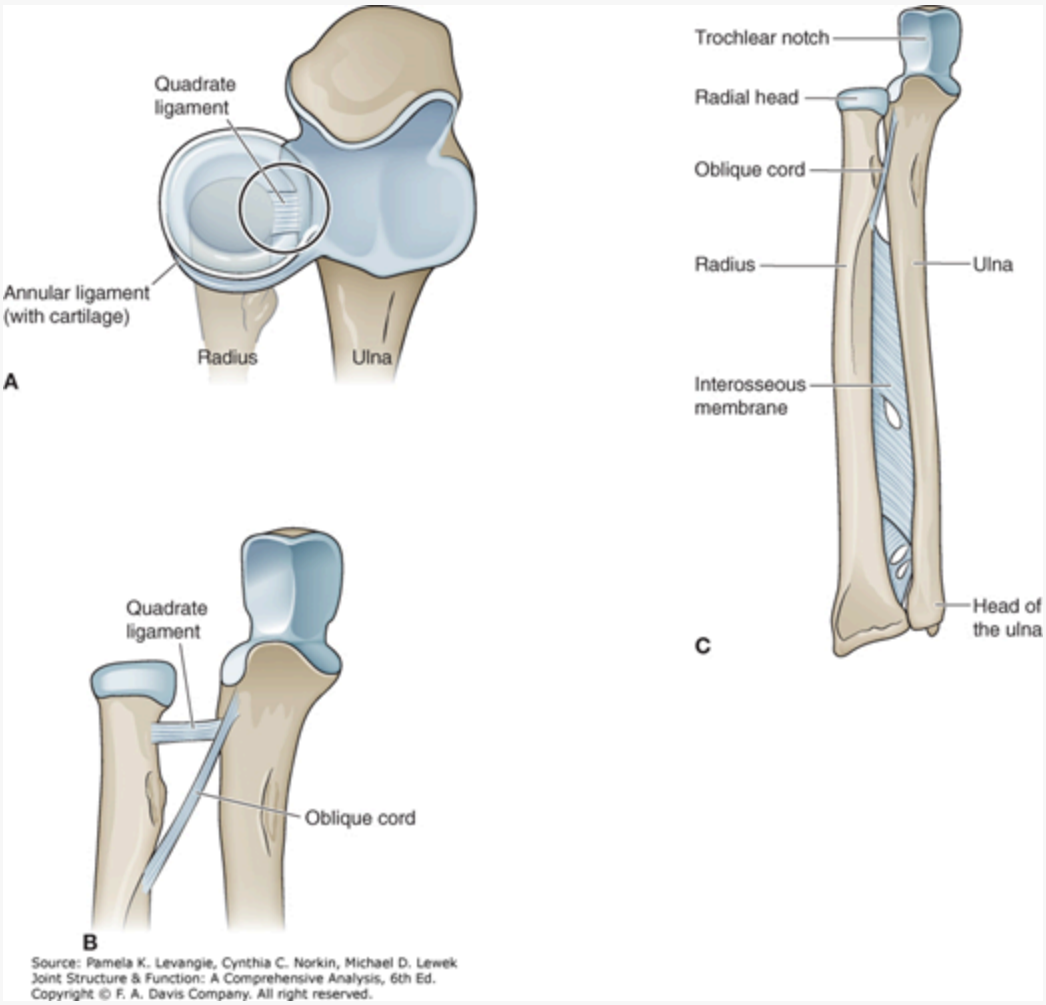
Ligaments

There are a number of ligaments that contribute to stability of the proximal and distal radioulnar joints (Fig. 8–22). Because the proximal and distal joints are mechanically linked, all radioulnar ligaments provide at least indirect support to both joints, although some are clearly associated with one joint more than the other. The three ligaments associated specifically with the proximal radioulnar joint are the annular and quadrate ligaments and the oblique cord. The annular ligament, as previously discussed, is a strong band that forms a ring that encircles the radial head and is actually part of the articular surface of the proximal radioulnar joint (see Fig. 8–19A). The proximal border

of the annular ligament blends with the joint capsule, and the lateral aspect is reinforced by fibers from the lateral collateral ligament. The **quadrate ligament** extends from the inferior edge of the ulna's radial notch to insert in the neck of the radius (Fig. 8–22A and B). The quadrate ligament reinforces the inferior aspect of the joint capsule and helps maintain the radial head in apposition to the radial notch. The quadrate ligament also limits the spin of the radial head in supination and pronation because it is attached to the medial radius just below the rim of the radial head. The proximally located **oblique cord** is a flat fascial band on the ventral forearm that extends from an attachment on the anterolateral aspect of the coronoid process on the ulna to insert just below the bicipital tuberosity on the radius (Fig. 8–22C).[77] The fibers of the oblique cord are at right angles to the fibers of the interosseous membrane. The functional significance of the oblique cord is not clear, but it may assist in preventing separation of the radius and ulna.

FIGURE 8-22

Selected ligaments of the right radioulnar joint **A.** Looking down on and "through" the semitransparent radial head, the quadrate ligament is shown running from the inferior radial notch on the ulna to the medial radius below its rim. **B.** With the annular ligament removed and the radius and ulnar distracted, the attachments of the quadrate ligament are easier to see. **C.** An anterior view of the radius and ulna in supination shows the oblique cord and the interosseous membrane.



The **dorsal** and **palmar radioulnar ligaments** provide direct support to the distal radioulnar joint. The dorsal and palmar ligaments are formed by longitudinally oriented collagen fiber bundles originating from the dorsal and palmar aspects of the ulnar notch of the radius. As shown previously, the two ligaments extend along the margins of the triangular fibrocartilage to insert on the ulnar fovea and base of the ulnar styloid process (see Fig. 8–20B).[72]

The **interosseous membrane** lies between the radius and ulna, providing stabilization at both the proximal and distal radioulnar joints (Fig. 8–22C).[77,78] It is a complex structure

consisting of a central band, a thin membranous portion, and a dorsal oblique cord. The central band is described as being strong and thick, consisting of bundles of fibers that run obliquely from the radius to the ulna.[77,79,80] When the tensile strength of the central band was compared with that of the patellar tendon, investigators found that the ultimate tensile strength of the central band was 84% of the strength of the patellar tendon.[81] Nakamura and associates found that the central band remained taut throughout forearm rotation, apparently to keep the radius and ulna from splaying apart.[82] In contrast to the central band, the membranous portion is described as a soft and thin structure that lies adjacent proximally and distally to the central band.[77] The dorsal oblique cord is considered part of the interosseous membrane and should not be confused with the oblique cord located on the ventral aspect of the forearm, which is not considered part of the interosseous membrane. The dorsal oblique cord extends from the proximal quarter of the ulna to the middle region of the radius.[77] Its fibers run counter to the central band.

The triangular fibrocartilage and distal radioulnar joint capsule also provides stability for the distal radioulnar joint by binding the distal radius and ulna together. The distal radioulnar joint capsule can be a source of limitation of motion when it is invaded by scar tissue after wrist injuries.[83]

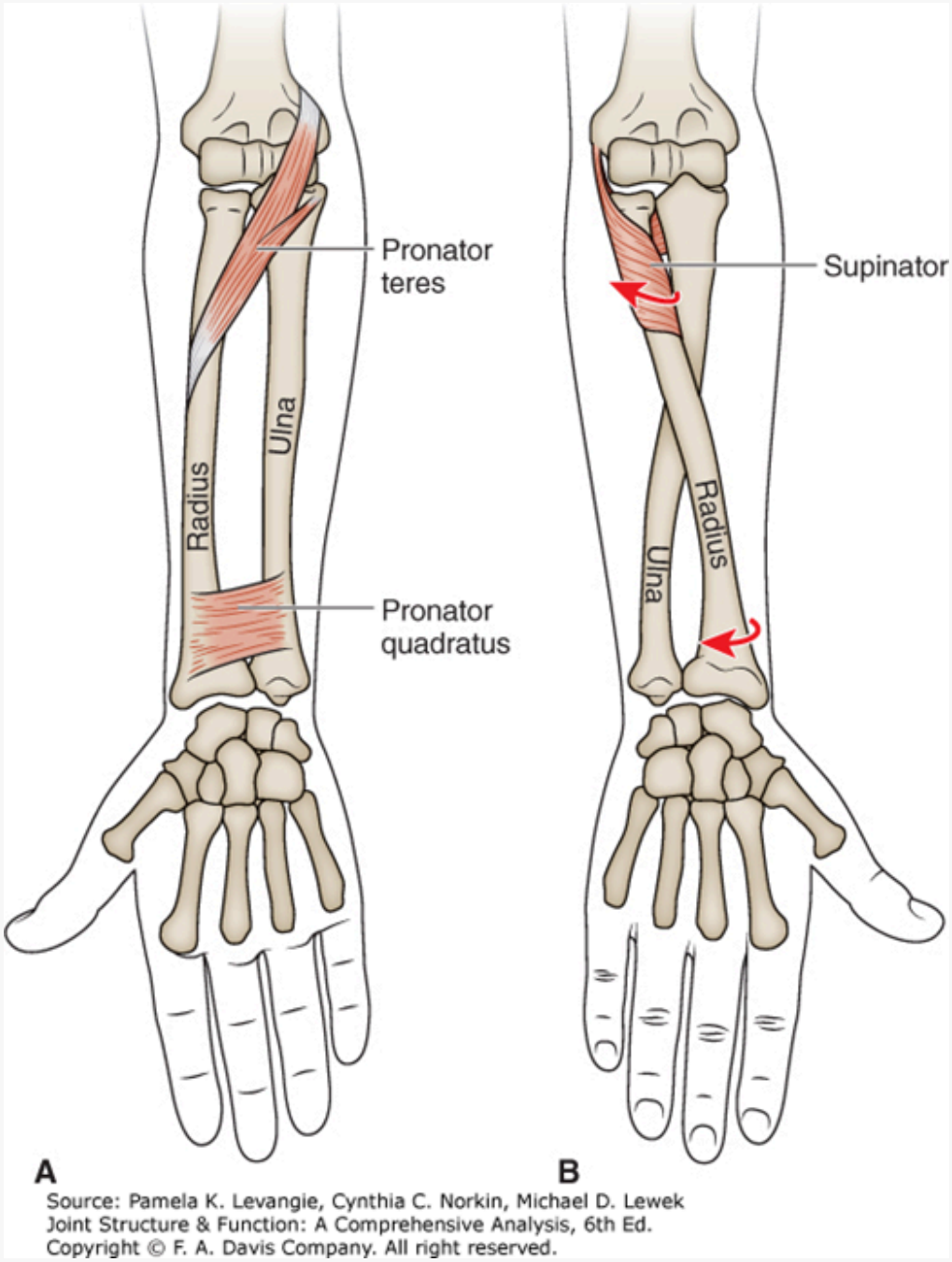
Muscles

The primary muscles associated with the radioulnar joints are the pronator teres, pronator quadratus, biceps brachii, and supinator. The two pronators are the **pronator teres** and the **pronator quadratus** (Fig. 8–23A). The pronator teres has two heads: a humeral head and an ulnar head. The humeral head comes from the common flexor

tendon on the medial epicondyle of the humerus. The smaller ulnar head arises from the medial aspect of the coronoid process of the ulna. Both heads attach distally to the surface of the lateral side of the radius at its greatest convexity. The **pronator quadratus,** located at the distal end of the forearm, also has two heads (superficial and deep). Both of these heads arise from the ulna and cross the interosseous membrane anteriorly to insert on the radius. The fibers of the superficial head pass transversely across the interosseous membrane, whereas the fibers of the deep head extend obliquely across the interosseous membrane to insert on the radius.[84]

FIGURE 8-23

Primary pronators and supinators of the right radioulnar joints. **A.** In this anterior view, the pronator teres and pronator quadratus are shown on the supinated forearm. When these muscles shorten, the radius will cross over the ulna into pronation (see position B). **B.** The supinator is shown coiled around the radius when the forearm is in pronation. When the supinator shortens, it will uncoil and pull the radius back to its position parallel to the ulna in supination (see position A).



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The biceps brachii, as was noted in the discussion of muscles acting on the elbow joint, is a radioulnar supinator as well as an elbow flexor. The one-joint supinator is the **supinator** muscle (Fig. 8–23B). The supinator is a short, broad muscle that arises from the lateral epicondyle of the humerus, radial collateral ligament, annular ligament, and

lateral aspect of the ulna to insert into the radius just inferior to the bicipital tuberosity. In pronation, the supinator wraps around the radius, uncoiling and spinning the radius when the supinator actively shortens.

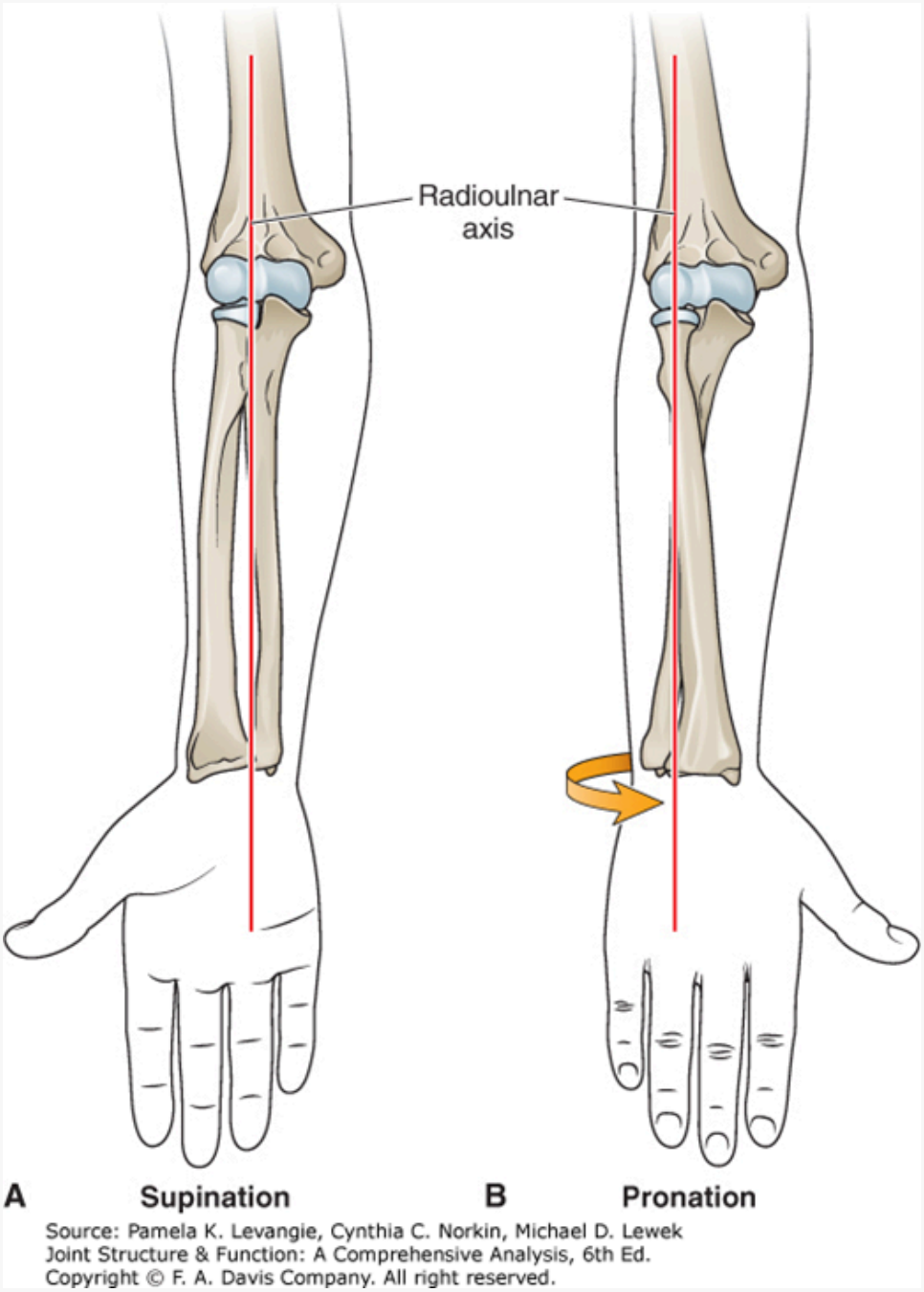
Function

Radioulnar Axis and Radioulnar Motions

The proximal and distal radioulnar joints are mechanically linked by a single longitudinal axis that extends from the center of the radial head proximally to the center of the ulnar head distally (Fig. 8–24).[70] Because the linked proximal and distal radioulnar joints have a common axis, motion must occur simultaneously at the proximal and distal joints.

FIGURE 8-24

Mechanical linkage of the proximal and distal radioulnar joints **A.** The radius and ulna are parallel to each other in the supinated position of the forearm, with the single longitudinal axis passing through the proximal and distal radioulnar joints. **B.** During pronation, the radius rotates within the annular ring proximally and pivots around the triangular fibrocartilage distally, with the moving radius crossing over the relatively stable ulna as the radius rotates around the longitudinal axis.



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In supination, the radius and ulna lie parallel to one another (Fig. 8–24A), whereas the radius crosses over the ulna in pronation (Fig. 8–24B). Proximally, the head of the radius spins within the osteoligamentous ring formed by the radial notch and the annular ligament, with the fovea of the radial head simultaneously spinning on the capitulum of

the humerus. Distally, the concave surface of the ulnar notch on the radius slides around the ulnar head, accompanied by the triangular fibrocartilage that sweeps beneath the ulnar head as it pivots around its attachment to the ulnar styloid.

Although the predominant motion at the radioulnar joints is motion of the radius, there is some motion of the distal ulna as well. Motion of the distal ulna is of less magnitude than that of the radius and opposite in direction to motion of the radius.[85] The ulnar head moves distally and dorsally in pronation, while moving proximally and medially in supination. Therefore, at the distal radioulnar joint, the ulnar head sits on the dorsal lip of the ulnar notch of the radius in pronation while it sits on the palmar aspect of the ulnar notch in full supination.[72] Contact between the head of the ulna and the ulnar notch of the radius is minimal at these end-ranges, and maximal when the forearm is midway between supination and pronation.[86]

Radioulnar Range of Motion

A total ROM of 150° has been ascribed to the radioulnar joints.[87,88] In the clinic, the ROM of pronation and supination is assessed with the elbow held against the trunk in 90° of flexion (Fig. 8–25). This position of the elbow stabilizes the humerus so that radioulnar joint rotation may be distinguished from rotation that is occurring at the glenohumeral joint. Shaaban and colleagues found supination and pronation ROM to vary based on the elbow position.[88] Controlling for all humeral motion, these investigators found a significant increase in supination with the elbow fully flexed and, conversely, greater pronation with the elbow extended. As shown in Fig. 8–26, there is approximately 90° supination and 60° pronation with the elbow flexed. When the elbow is extended, the opposite occurs with greater pronation motion (100°) and less supination motion

(50°).[88] From a function standpoint, it is advantageous to have more supination when putting the hand to the mouth to eat, generally accompanied by elbow flexion.[70] Pronation in all elbow positions is limited by bony approximation of the radius and ulna and by tension in the dorsal radioulnar ligament and the posterior fibers of the medial collateral ligament of the elbow.[89] Supination is limited by passive tension in the palmar radioulnar ligament and the oblique cord. The quadrate ligament that attaches to the radius immediately below the radial head limits spin of the radial head in both pronation and supination. The annular ligament does not limit motion because the radial head rotates within the annular ring but does help maintain stability of the proximal radioulnar joint by holding the radius in close approximation to the radial notch.

FIGURE 8-25

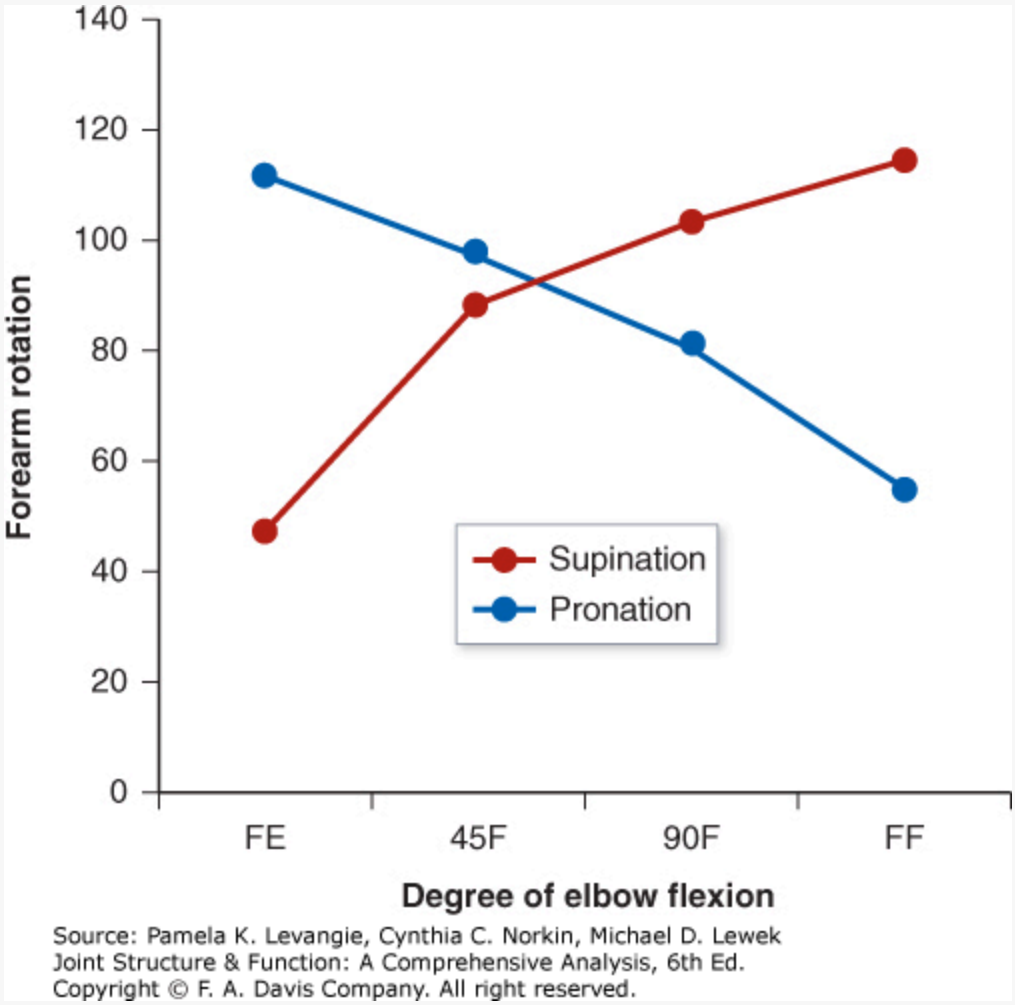
Clinical measurement of radioulnar motion. Measurement of the end range of supination with a goniometer. The subject's left elbow is flexed to 90° and the upper arm is held close to the trunk. The same position is used for measuring the end range in pronation.



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FIGURE 8-26

The relationship between the angle of flexion at the elbow and its effect on range of active pronation and supination of the forearm. The range of pronation is maximal with the elbow fully extended (FE) and the range of supination is maximal with the elbow fully flexed (FF). (*Reproduced with permission from: Shaaban H et al. J Hand Surg Eur Vol 2008; 33: 3–8. Sage Publications Ltd. All rights reserved.)*



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Muscle Action

The pronator teres has its major action at the radioulnar joints, but the long head also crosses the elbow joint. Lying very close to the elbow joint axis, the pronator teres plays no more than a slight role in elbow flexion but can contribute to stabilization of the proximal radioulnar and humeroradial joints. The pronator quadratus, a one-joint muscle, is unaffected by changing positions at the elbow. The pronator quadratus is active in unresisted and resisted pronation and in both slow and fast pronation. The pronator quadratus is thought to act as a dynamic stabilizer to maintain compression of the distal radioulnar joint.[84]

In a mechanical study on cadavers, the pronators were found to be most efficient around the neutral position of the forearm when the elbow was flexed to 90°.[90] The pronating effect of the pronator teres and pronator quadratus were found to be maximal between 40° of supination and 40° of pronation, with a decline at the extremes of pronation and supination.[91]

The supinator muscle, as a one-joint muscle, may act alone during unresisted slow supination in all positions of the elbow or forearm. It can also act alone during unresisted fast supination when the elbow is extended. However, activity of the biceps brachii is always evident when supination is performed against resistance, or during fast supination when the elbow is flexed to 90°. Bremer found that the moment arms of all major supinators exhibited peak torque values in 40° to 50° of pronation.[91] The biceps brachii has been found to exert four times as much supination torque with the forearm in the pronated position than with other forearm positions.[90] A mean maximum supination torque of 16 Nm was recorded with the forearm 75% pronated, in comparison with a mean maximum supination torque of 13.1 Nm for the neutral forearm position, with the elbow at 45° of flexion.[92]

Gordon and colleagues found supination and pronation torques to be essentially similar when the forearm was in a neutral position. However, supination torque generation was greatest with the forearm in pronation, and pronation torque generation was greatest with the forearm in supinated positions.[93] The brachioradialis also plays a role in supination and pronation. Naito and colleagues demonstrated in healthy subjects that the brachioradialis showed high levels of activity during rapid alternating supination/pronation motions with the elbow held at 90° of flexion. Higher

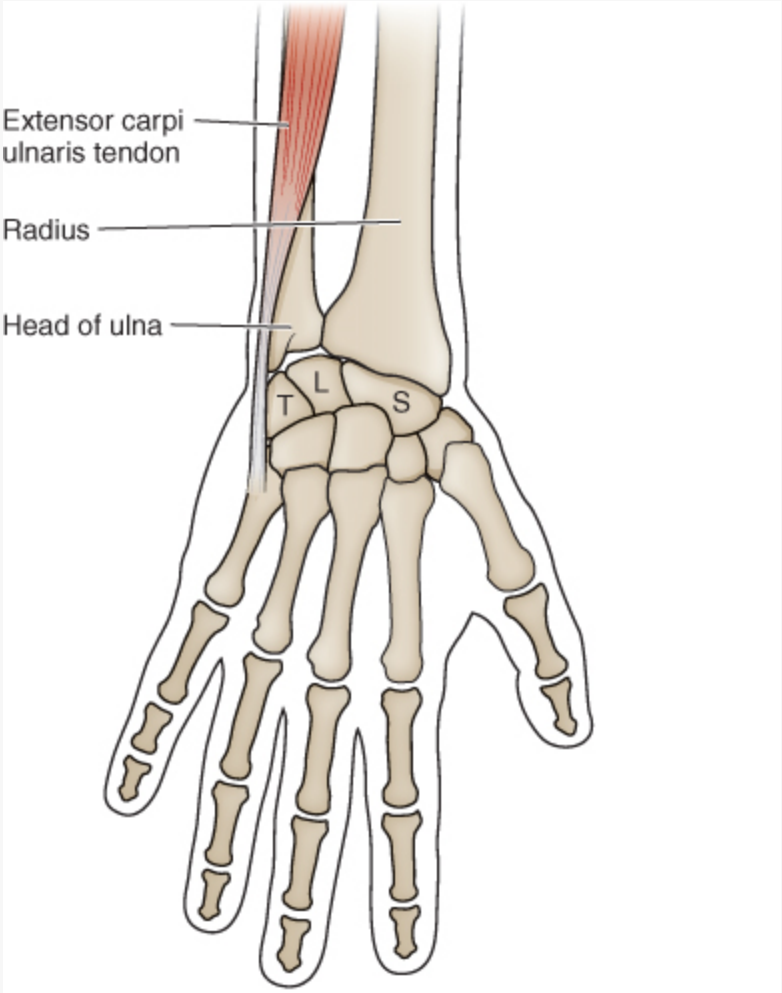
levels of activity were noted when the forearm was pronated than when it was supinated.[63] Bremer suggested that the brachioradialis acts as a pronator in supination and as a supinator in pronation.[91]

Stability

Muscular support of the distal radioulnar joint is attributed to the pronator quadratus[84,94,95] and the extensor carpi ulnaris tendon.[70,80] The deep head of the pronator quadratus is active throughout supination and pronation, and therefore is thought to provide dynamic stabilization for the distal radioulnar joint.[96] Activity in the extensor carpi ulnaris muscle (Fig. 8–27) exerts a depressive force on the dorsal aspect of the ulnar head as the tendon is stretched over the ulnar head during supination. Tension in the tendon helps to maintain the position of the ulnar head during both supination and pronation[70]

FIGURE 8-27

Extensor carpi ulnaris. This dorsal (posterior) view of the right forearm and hand shows the extensor carpi ulnaris tendon as it passes distally through the notch in the ulnar head to its attachments on the base of the 5th metacarpal. The muscle is attached proximally from the lateral epicondyle of the humerus. The distal tendon can stabilize the head of the ulna during pronation and supination. L, lunate; S, scaphoid; T, triquetrum.



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The extensor carpi radialis brevis, like the extensor carpi ulnaris, appears to provide support for radioulnar joints, as evidenced by the magnitude of motor unit activity during radioulnar motion. Maximum voluntary effort (MVE) is a measure of electrical activity in a muscle during a maximal contraction. During active supination, the extensor carpi radialis brevis showed activity equivalent to 26% to 43% of its MVE, with active pronation showing 27% to 55% of the muscle's MVE. The extensor carpi radialis brevis also appears to act both as a stabilizer to the forearm for gripping during forceful

pronation (depending on forearm angle) and as a prime mover for wrist extension during supination torques.[92]

Patient Application 8–3

Belinda Chambliss, a 47-year-old female textile worker, reports to physical therapy complaining of left lateral elbow pain for the past 2 months with no history of trauma. Her job consists of sitting at a sewing machine for half her day, with the remainder of her workday spent folding, packing, and lifting boxes of clothes. She does not report any change in her daily activity over the past couple of months. She has had a history of neck pain but indicated that it is not bothering her at this time. Key objective findings are as follows:

- Pain with palpation to the extensor carpi radialis brevis tendon
- Elbow pain with resisted wrist extension
- Patient prefers to sit in a slumped manner with forward head and rounded shoulders
- Cervical ROM is limited with extension, right rotation, and right side-bending, and pain is provoked along the right upper trapezius with cervical provocation tests
- Slight decrease in elbow pain with cervical distraction

This is a very common presentation of elbow pain seen in many outpatient physical therapy clinics. It is not uncommon to find poor postural habits and cervical dysfunction in combination with repetitive upper extremity activity that results in complaints of elbow pain of insidious onset.

O'Sullivan and Gallwey found a direct link between gripping during forearm rotations and high muscular activity in the extensor carpi radialis brevis.[92] Research has also shown a poor prognosis in cases of lateral epicondylagia (tennis elbow) associated with manual job employment with a high level of physical strain at work and a high level of pain at baseline.[97] Treatment focused on patient education and ergonomic instruction combined with specific treatment to the spine and elbow will give the therapist and patient the best chance for success. If only localized treatment to the elbow is implemented, results can vary.

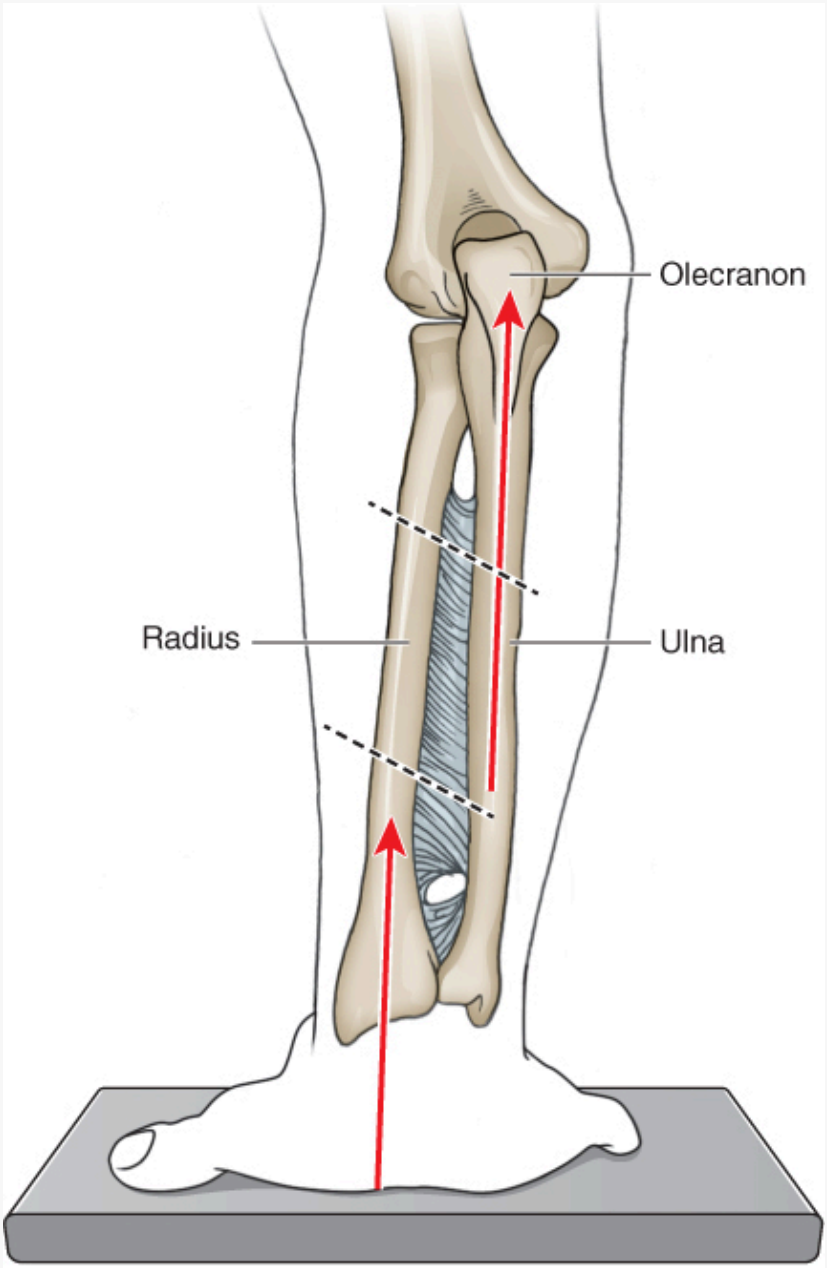
Passive support of the distal radioulnar joint is provided by the dorsal and palmar

radioulnar ligaments, the interosseous membrane, and the triangular fibrocartilage. The dorsal radioulnar ligament becomes taut in pronation, whereas the palmar radioulnar ligament becomes taut in supination.[70,95,98] The radioulnar ligaments have limited cross-sectional areas and low structural stiffness, but they are able to prevent separation of the radius from the ulna during loading and may also contribute to force transmission from the radius to the ulna through the distal radioulnar joint. However, these ligaments do not augment longitudinal stability. They allow approximately 5 mm of play between the radius and ulna before providing resistance to further distraction.[72] The interosseous membrane and triangular fibrocartilage provides stability for the distal joint by binding the radius and ulna together. However, they also provide important longitudinal stabilization.[79,80]

The interosseous membrane protects the proximal radioulnar joint and head of the radius by transferring some of the compressive loads at the distal radius to the proximal ulna.[99] At the wrist, the base of the radius is substantially larger than the head of the ulna and the ulna is also somewhat protected from compressive forces by the soft tissues of the triangular fibrocartilage complex (see Fig. 8–20B). If not attenuated, the force imposed on the distal radius would potentially cause large compressive stress at the small articulation between the radial head and the capitulum proximally. When bearing weight on the wrist and hand, the direction of the central fibers of the interosseous membrane limits the proximal shift of the radius on the ulna (**Fig. 8–28**), limiting compression of the radial head and shifting a proportion of the force imposed on the distal radius to the ulna that has a much larger surface at the humeroulnar joint.

FIGURE 8-28

Interosseous membrane in force transmission. In bearing weight on the right hand (posterior view), the greater proportion of the ground reaction force is borne by the large base of the radius. As the interosseous membrane tightens, superiorly directed motion of the radius is checked and transferred to the ulna and, from there, on to the larger humeroulnar surfaces.



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DeFrate and colleagues determined that the average proportion of total load transmission in the radius and ulna differed with forearm position. In supination, load distribution was 68% in the distal radius and 32% in the distal ulna. Proximally, the proportions shifted to 51% in the proximal radius, and 49% in the proximal ulna.[100] The

axial force transmitted through the radius was found to be least in full supination, increasing as the forearm moved toward pronation. Axial force transmission through the ulna was least at the extreme of pronation and gradually increased as the forearm moved into supination.[101]

Markolf and associates studied radioulnar load sharing at the wrist and elbow, with the elbow in varus, valgus, and neutral positions.[102] When the elbow was in a relative varus position (no contact between the radial head and capitulum), force was transmitted from the distal radius through the interosseous membrane to the proximal ulna. When the elbow was in a relative valgus position (with contact between the radial head and the capitulum), the force was transmitted through the radius. When the forearm was in a neutral varus/valgus position, the mean force in the distal end of the ulna averaged 7% of the applied wrist load, whereas the force in the proximal ulna averaged 93% of the load applied to the wrist.[102]

The triangular fibrocartilage acts as a cushion in allowing compression force transmission from the carpals to the ulna, and acts as a stabilizer of the ulnar side of the carpals.[70,103] Also, like the interosseous membrane, the triangular fibrocartilage assists in the transmission of compressive forces from the radius to the ulna.[102,104] Adams and Holley used a distractive force to simulate the effects of the separation of articulating surfaces that accompanies a power grip.[105] These authors found that strain distribution in the triangular fibrocartilage was dependent on forearm position.[105] Tension increased in the radial portion of the fibrocartilage in pronation, but decreased across the entire triangular fibrocartilage in supination. The authors concluded that the triangular fibrocartilage regularly bears both compressive and tensile strains.[105]

Compressive forces are transmitted through the central portion of the disc, and some of the load is converted to tensile loading within the peripheral margins.[87,106]

A summary of ligamentous and muscular support for both radioulnar joints is presented in Table 8–2.

Table 8-2

Ligamentous and Muscular Contributions to Stability at the Proximal and Distal Radioulnar Joints

Mobility and Stability of the Elbow Complex

Functional Activities

The joints and muscles of the elbow complex are used in almost all activities of daily living, such as dressing, eating, carrying, keyboarding, and lifting. They are also used in tasks such as splitting firewood and hammering nails, and in sports such as tennis, golf, basketball, and baseball. Most of the activities of daily living require combinations of motion at the elbow and radioulnar joints. Morrey and associates measured elbow and forearm motion in 33 healthy subjects during 15 activities.[107] The authors concluded that a total arc of about 100° of elbow flexion (between 30° and 130°) and about 100° of forearm rotation (50° supination and 50° pronation) were sufficient to accomplish simple tasks such as eating, drinking, brushing hair, brushing teeth, and dressing (Fig. 8–29). Therefore, mobility of the complex is necessary for normal functioning in most

areas of activity. As can be seen in Table 8–3, among the 10 activities listed, using a phone requires the largest arc of motion in flexion (92.8°) and pronation/supination (63.5°).[107–110] Cutting with a knife requires the smallest arc of flexion and of pronation/supination.

FIGURE 8-29

Functional range of motion. According to Safaee-Rad[110] (see Table 8–4), drinking from a cup requires a range of elbow flexion between 72° and 129° and a range of pronation/supination between 3° and 31°. (*From Norkin CC, White DJ: Measurement of Joint Motion: A Guide to Goniometry [ed. 5]. Philadelphia, FA Davis, 2016, with permission.)*



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Table 8-3

Elbow and Forearm Motion During Functional Activities: Mean Values in Degrees

Relationship to the Hand and Wrist

The design of the radioulnar joints enhances the mobility of the hand. In primitive mammalian species, the ulna was a major weightbearing structure and was connected directly to the carpals through a dense immobile syndesmosis.[111] The complete separation of the ulna from the carpals by the articular disc and the formation of a true diarthrodial joint lined with articular cartilage are features that permit pronation and supination to occur in every position of the hand in relation to the forearm. The mobility afforded the hand is achieved at the expense of stability; the movable forearm is unable to provide a stable base for attachment of the wrist and hand muscles. Therefore, many of the muscles that act on the wrist and hand are attached on the distal end of the humerus rather than on the forearm.

The location of the hand and wrist muscles on the distal humerus and the fact that these muscles cross the elbow create close structural and functional relationships between the elbow and wrist/hand complexes. Anatomically, the hand and wrist muscles help reinforce the elbow joint capsule and contribute to stability of the elbow complex. Davidson and coworkers found that the flexor carpi ulnaris muscle is the only muscle that lies directly over the anterior portion of the medial collateral ligament when the elbow is flexed between 90° and 120°.[112] Because the medial elbow is subjected to the largest valgus stress during the cocking and acceleration phases of throwing, which occur between 80° and 120° of elbow flexion, the flexor carpi ulnaris

muscle has the potential to provide significant reinforcement for the medial collateral ligament during throwing activities.[112] Lin and colleagues suggested that the flexor carpi ulnaris, flexor digitorum superficialis, and flexor carpi radialis function as dynamic stabilizers, with the flexor carpi ulnaris being the primary stabilizer for elbow valgus stability.[113] The flexor carpi ulnaris, flexor digitorum superficialis, and flexor carpi radialis significantly reduced medial collateral ligament strain at 45° and 90°. All extensor muscles increased the valgus strain on the medial collateral ligament.[113]

Life Span and Clinical Considerations

Like other joints in the body, the joints and muscles of the elbow complex may be subject to the effects of age, injury, and immobilization. Knowledge of these effects will significantly improve a clinician's ability to evaluate and treat elbow problems in patients of all ages.

Ossification Centers

Diagnosing elbow injuries in children is challenging due to the large discrepancy between clinical presentation and radiographic findings.[114] Knowledge of the anatomy, specifically the ossification centers and their presentation, is very important. The ossification centers of the elbow are unique in that they ossify late and in a predictable manner according to the child's age and sex.[114–116] A common mnemonic for the ossification centers is **CRITOE:**

Capitulum

Radial head

Internal epicondyle

Trochlea

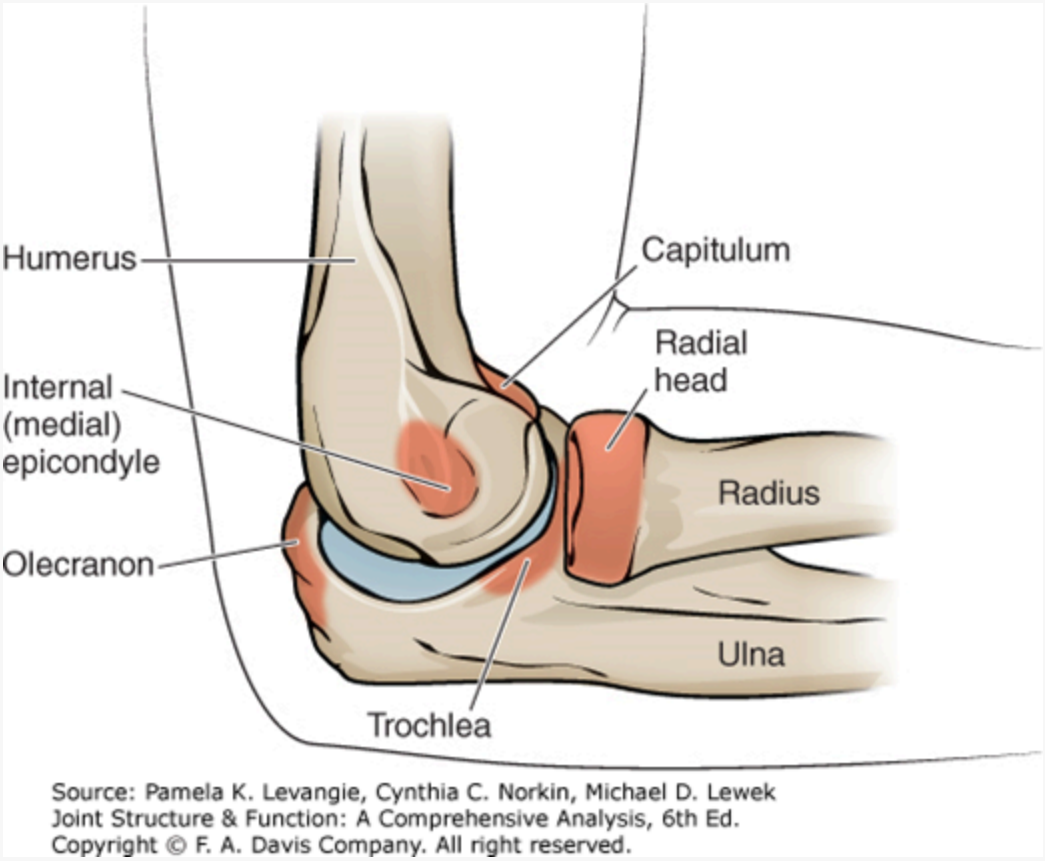
Olecranon

External epicondyle

Knowledge of these growth centers and their location (Fig. 8–30) is important because these growth centers may be vulnerable to injury until fused.[117] An understanding of these centers and the predictable progression of ossification may prevent missed diagnoses that can result in long-term complications in the pediatric population.[114] Refer to Table 8–4 for a list of the ossification centers and their typical progression of ossification.

FIGURE 8-30

Ossification centers at the elbow. The growth centers at the elbow (lateral epicondyle center not shown) are possible areas for apophysitis and avulsion fractures until fused. See Table 8–4 for ages at which each center fuses.



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Table 8-4

CRITOE: Ossification Centers of the Elbow

Effects of Gender

Wood investigated isokinetic elbow torque longitudinally over a 3-year period in boys and girls initially averaging 13 years of age.[118] Isokinetic concentric and eccentric elbow extensor peak torques did not differ significantly at any test occasion for boys and girls. As one might expect, eccentric elbow flexor torques were significantly greater than

concentric elbow flexor torques. Deighan et al measured the maximum cross-sectional area of the elbow flexors and extensors in children (ages 9–10), teenagers (ages 16–17), and adults (> 21 years old).[119] A greater physiological cross-sectional area was found in males than in females except in the youngest group; there was a greater increase in physiological cross-sectional area with increasing age among males than among females. Tonson and colleagues also found that mean physiological cross-sectional area ratios were significantly higher in adults than in children and adolescents.[120]

Effects of Age

As can be seen in the sampling of experimental findings presented in Table 8–5, the decrease in muscle strength that accompanies increasing age appears to be affected by the type of muscle action involved (eccentric/concentric), the muscle group involved, and gender, along with other factors such as level of physical activity.[121–125] Also, the elderly have been found to have smaller pennation angles and decreased fascicle lengths, which may account for as much of 50% of the loss of muscle function. Fortunately, resistance training may be able to increase fascicle length and, therefore, the number of sarcomeres.[126]

Table 8-5

Effects of Aging on Elbow Muscles

Lynch and colleagues investigated peak concentric and eccentric torque generation for

elbow flexor and extensor muscles in a large sample of individuals from 19 to 93 years of age. They confirmed age-related declines in concentric and eccentric peak torques in both genders.[127] John and colleagues determined that younger subjects produce significantly greater relative joint torques than older subjects across all effort levels except for the light effort level.[128] Valour and Pousson found that maximal isometric force and series elastic component compliance of the elbow flexors were significantly less in the elderly than in younger groups.[129] Toji and Kaneko concluded that the effects of aging on muscle shortening velocity become apparent after age 50.[130] Isokinetic testing conducted by Runnels et al in a group of 75 volunteers ages 20 to 83 years demonstrated that the elbow flexor and extensor muscle performance remained relatively constant from ages 20 to 59 before declining at all angular velocities.[131] Chronological age affected the performance of muscles independent of muscle mass and regardless of contraction type; however, isometric contraction was the least affected in 75 volunteers aged 20 to 83 years.[131]

Klein and associates found that the area of type I fibers in the biceps brachii muscle and maximum voluntary strength of the elbow flexors is lower in the elderly than in young persons, but the percentages of type II fibers and type I fiber areas are not different between young and old persons.[132]

Injury

Injuries to the elbow are fairly common. An understanding of the mechanisms of elbow injuries and their relation to elbow joint structures is necessary for determining the effects of the injuries on joint function.

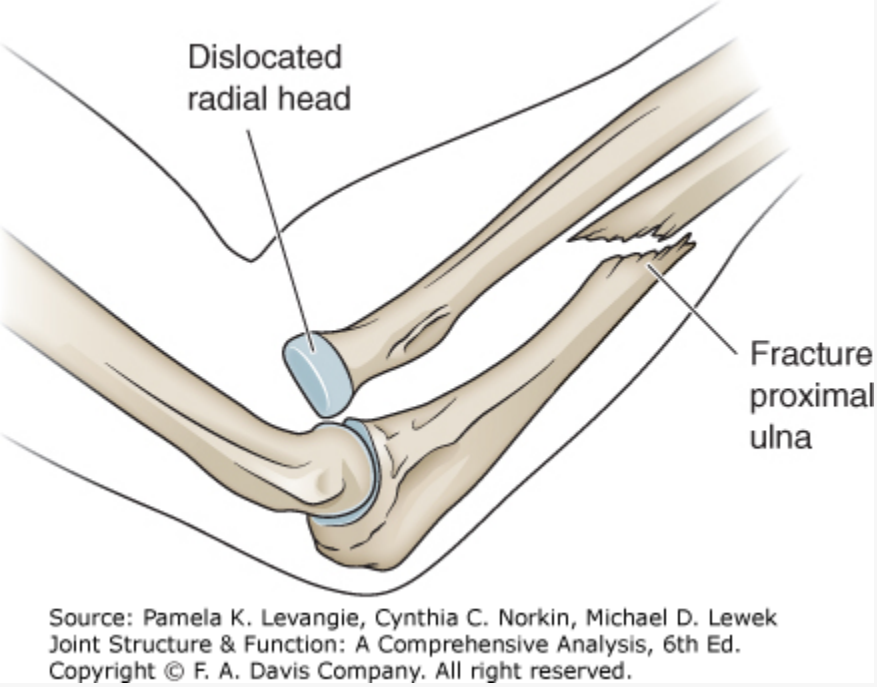
Compression Injuries

Resistance to longitudinal compression forces at the elbow is provided mainly by the contact of bony components; therefore, excessive compression forces at the elbow often result in bony failure. Falling on the hand when the elbow is in a close-packed (extended) position may result in the transmission of forces through the distal radius and ulna to the elbow, leading to fractures of one or both bones depending on the direction of forces (Fig. 8–31). If the forces are transmitted through the radius, as may happen with a concomitant valgus stress, a fracture of the radial head may result from impact of the radial head on the capitulum. A fracture of the radial head may be accompanied by a tear of the central band of the interosseous membrane given that one of the functions of the interosseous membrane is to limit compressive forces at the radial head. The central band has little physiological ability to heal, so may result in longitudinal instability if the injuries are not addressed surgically.[133] If the force from the fall is transmitted to the ulna, a fracture of either the coronoid or olecranon processes may occur from the impact of the ulna on the humerus. If neither the radius nor the ulna absorbs the excessive force by fracturing, then the force may be transmitted to the humerus, resulting in a fracture of the supracondylar area or more proximally in the humerus.

FIGURE 8-31

A fall on an outstretched hand with the elbow in extension (close-packed position) and the forearm pronated will result in the transmission of forces through the radius, the ulna or both. When the force is sufficient, a common pattern is a fracture of the proximal ulna

and anterior dislocation of the radial head



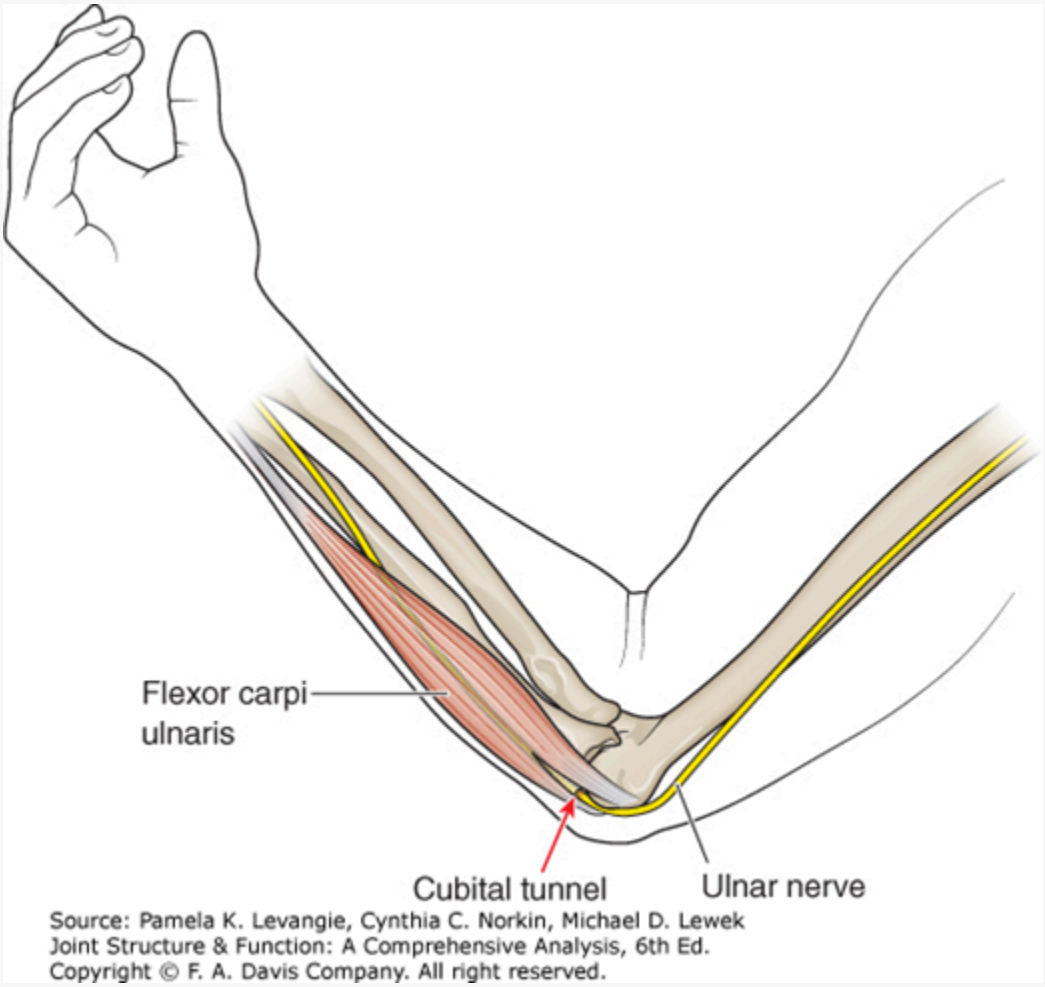
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Muscle contractions also may cause high compression forces at the elbow. For example, during the acceleration and deceleration phases of baseball pitching, the compression forces at the elbow can reach 90% of body weight.[134] Repetitive forceful contractions of the flexor carpi ulnaris muscle may compress the ulnar nerve as it passes through the cubital tunnel between the medial epicondyle of the humerus and olecranon process of the ulna (Fig. 8–32).[135,136] Such compression may be worsened with increasing elbow flexion as both the cubital tunnel and ulnar nerve cross-sections decrease with increased flexion.[137]

FIGURE 8-32

Ulnar nerve in the right cubital tunnel. The ulnar nerve passes

through the cubital tunnel formed by the two heads of the flexor carpi ulnaris muscle. A contraction of flexor carpi ulnaris muscle can cause compression of the ulnar nerve between the two heads of the muscle, which are located on either side of the ulnar nerve at the elbow.



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Patient Application 8–4

Margaret Blevins is a 46-year-old nurse who slipped backwards on a wet floor and attempted to cushion the fall with her right hand extended behind her. She immediately noted pain in her right wrist and elbow, but did not report the injury until the end of her shift. She was seen by an occupational health nurse practitioner who referred her to physical therapy the next day for an elbow contusion. Upon

examination in physical therapy, the patient complained primarily of right lateral elbow pain and was holding her right elbow in a flexed position. She was found to have noticeable swelling at the radioulnar joint line and tenderness at the joint with palpation. Excess fluid had accumulated within the joint that can stretch the joint capsule and cause pain. Patients will typically attempt to reduce the pain by assuming a position of about 80° of flexion, considered to be the elbow position at which the least amount of tension is present in the joint capsule.[138] Therefore, it is a position of relative comfort for patients with intra-articular swelling. The patient in this case was referred for imaging that revealed an intra-articular radial head fracture. The patient was subsequently placed in an immobilizer to allow the radial head fracture to heal.

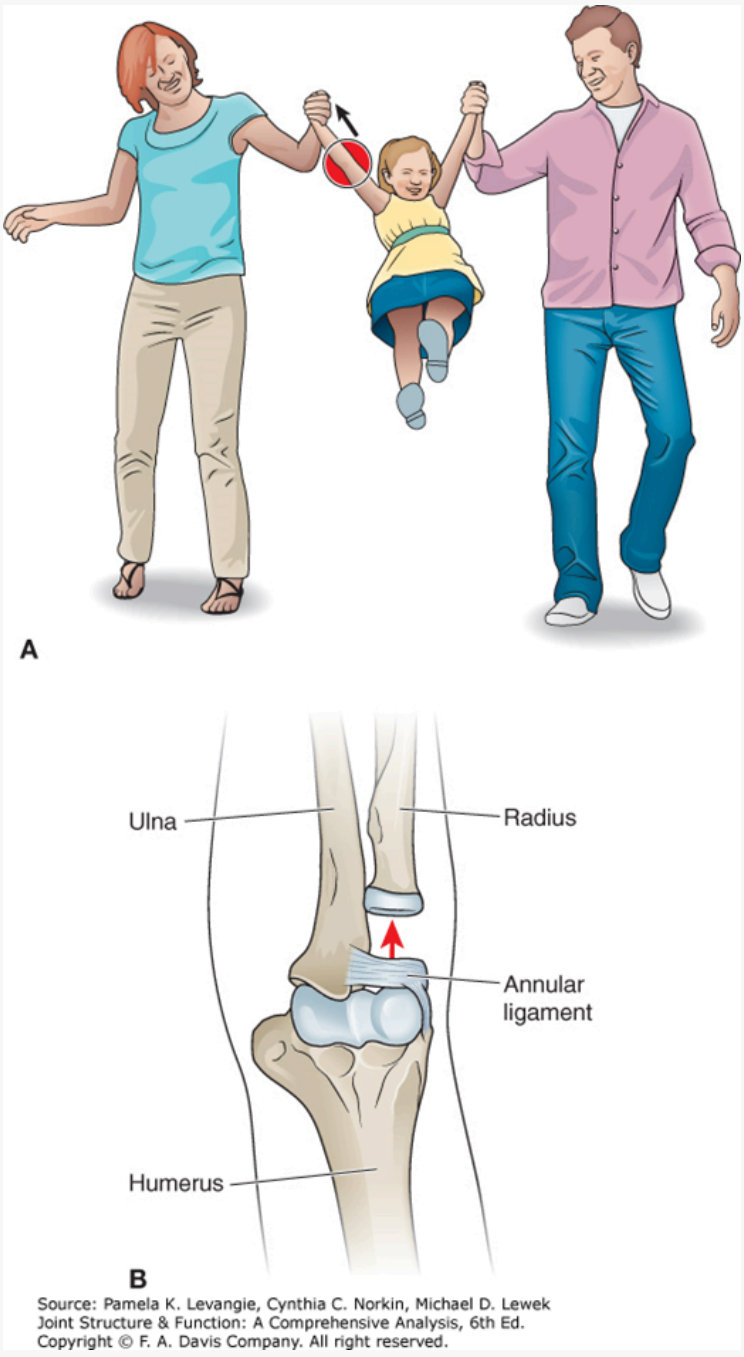
Distraction Injuries

Both ligaments and muscles at the elbow provide resistance to longitudinal traction forces. A tensile force of sufficient magnitude exerted on a pronated and extended forearm may cause the radius to be pulled inferiorly out of the annular ligament. This injury is common in children younger than 5 years, females more so than males, and is rare in adults.[139,140] The predominance in young children is attributed to the laxity or elasticity of the annular ligament in the young. Lifting a small child up into the air by one or both hands or yanking a child by one hand is the usual causative mechanism, and therefore the injury is referred to as **nursemaid's elbow** (Fig. 8–33).[139]

FIGURE 8-33

Nursemaid's elbow: **A.** An upward longitudinal force on the child's hands can create distraction at the humeroradial joint. **B.** The right radial head can slip out of the annular ligament, especially if the child is relaxed and active muscle forces crossing the elbow aren't

producing an offsetting joint compression.



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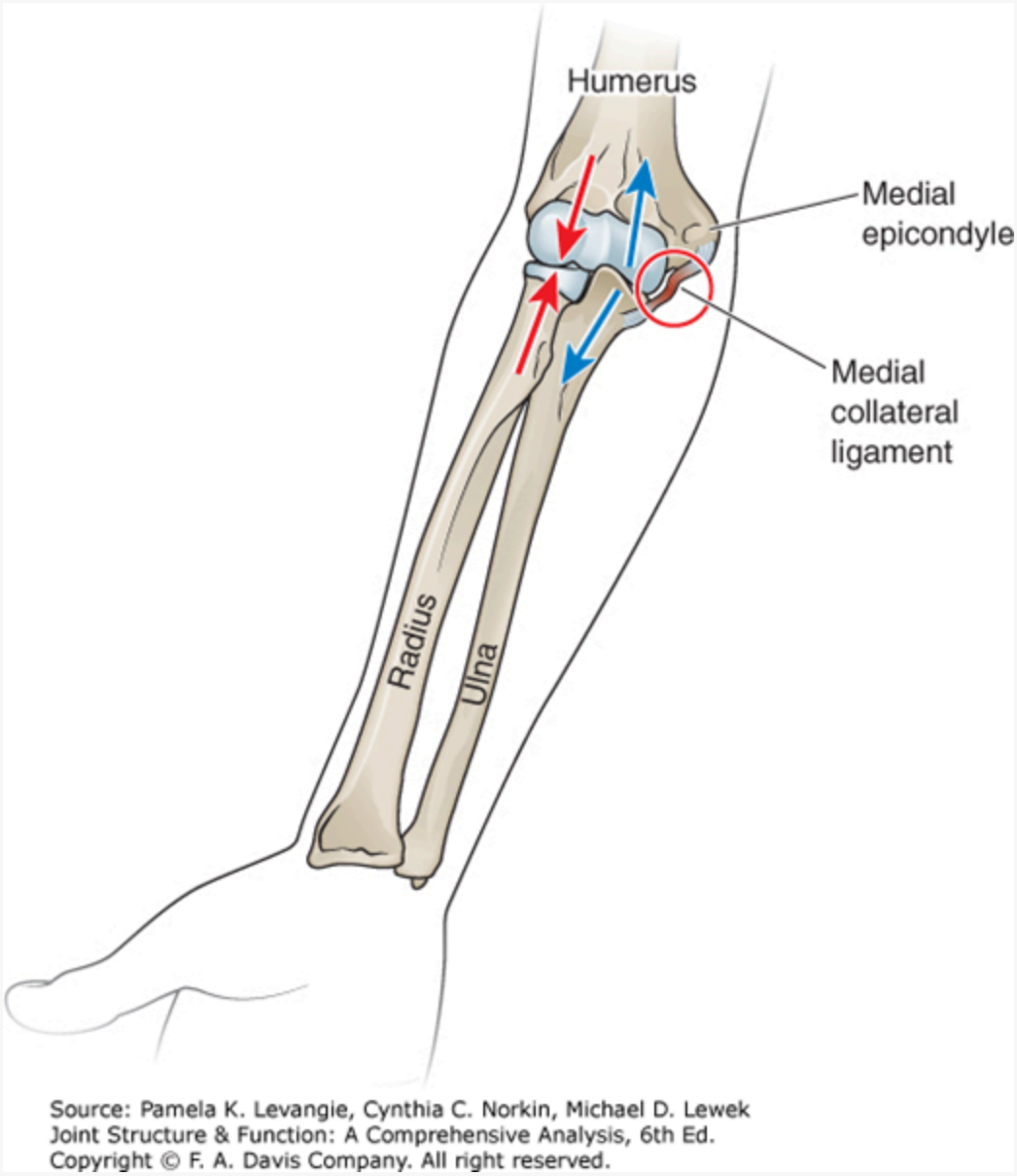
Varus/Valgus Injuries

Valgus and varus stresses at the elbow occur in many activities. When such stresses occur, one side of the joint is subjected to distraction forces whereas the other side is

subjected to compression forces. In the more commonly occurring valgus stress at the elbow, the radial head and capitulum are compressed while the proximal ulna and trochlea are distracted (Fig. 8–34). With repeated valgus stresses, the medial collateral ligament may become overstretched or torn, which then allows greater distraction medially and results in even greater compression between the radial head and capitulum. The opposite circumstances will occur with the less common varus stresses at the elbow.

FIGURE 8-34

Valgus injuries. The application of a valgus stress to the forearm produces compression at the humeroradial joint and distraction at the humeroulnar joint. With repeated valgus stress, the medial collateral ligament that ordinarily checks joint distraction may become stretched and inflamed



During the backswing or “cocking” phase of pitching a baseball (Fig. 8–35), the stress on the medial collateral ligament is repetitive, leading to ligamentous laxity and inability of the ligament to reinforce the medial aspect of the joint.[12,117] The resulting medial instability may cause an increase in the normal carrying angle and excessive compression between the radial head capitulum.[141] If the abnormal compression forces are prolonged, these forces may interfere with the blood supply of the humeroradial

joint cartilage and result in **avascular necrosis** of the capitulum.[117]

FIGURE 8-35

During the cocking phase of a baseball pitch, the valgus stress on the elbow is high. Repetitive valgus stresses to the elbow of a pitcher may lead to laxity or inflammation of the medial collateral ligament and elbow joint instability.



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Patient Application 8–5

Steve Cho is a 13-year-old male who joined two baseball teams during the summer to

practice his pitching on a more frequent basis. Three weeks into the summer season, Steve noticed medial elbow soreness in his throwing arm after pitching and that his pitching velocity and control were declining. When the elbow became painful with casual throwing of the ball, his parents sought medical advice. Palpation along the medial collateral ligament was painful. Excessive laxity was found with a valgus stress applied to the elbow in comparison with his non-throwing arm; the valgus stress reproduced the sharp pain that he was experiencing during pitching. It was determined that Steve had sprained his medial collateral ligament, so he was pulled out of the pitching rotation until complete healing occurred.

This scenario is very common as evidenced by the substantial increase in the number of youth baseball players seeking medical attention for elbow injuries, specifically medial collateral ligament sprains and tears.[117] This has led to new recommendations for youth baseball pitching such as count limit guidelines per week and at least 3 months of not pitching during the year, among others, to reduce the risk of injury.

Garrison et al examined baseball players who were diagnosed with a medial collateral ligament tear and compared these players with age-and position-matched healthy control players.[142,143] Their studies suggest that alterations in motion, strength, or proximal motor control are associated with medial collateral ligament injury, most likely due to increased valgus stress at the elbow.

Other conditions that may occur in the throwing elbow include ulnar neuritis, flexor-pronator muscle strain or tendinitis, and medial epicondylitis.[12] Medial tendinitis and medial epicondylitis may be caused by forceful repetitive contractions of the pronator teres, the flexor carpi radialis, and, occasionally, the flexor carpi ulnaris. These muscles are involved both in pitching and in a tennis serve, where combined motions of elbow extension, pronation, and wrist flexion are used. Medial epicondylitis, often referred to as golfer's elbow, is thought to result from traction-based insults to the elbow from the wrist and hand flexors, and the forearm pronators at their insertion into the medial

epicondyle. Overuse or a sudden deceleration of the golf club head hitting the ground instead of the ball is the usual cause.[144]

Peripheral Neuropathy

The elbow has among the highest incidences of peripheral neuropathies in the body.[145] Factors such as superficial positioning of nerves, unique anatomical structures, and high mechanical and repetitive stress to the elbow make the nerves susceptible to injury. Classic examples of peripheral entrapments around the elbow are excessive or repetitive valgus traction stress to the ulnar nerve with throwing or a compressive stress to the median nerve from repetitive gripping and pronation when using a screwdriver.[146–148] Even simple activities such as resting an elbow on the armrest or back of a chair for a prolonged period of time could result in localized nerve ischemia to the ulnar or radial nerves.



Patient Application 8–6

Nijee Warfield, a 21-year-old male college student, reports to the emergency department complaining of wrist weakness and forearm pain for unknown reasons upon waking. The patient was referred to the physical therapist for consultation and key objective findings were as follows:

- Localized tenderness with palpation to the radial tunnel on the dorsal aspect of his forearm
- One-fifth strength of his wrist extensors
- Negative cervical provocation

Upon further inquiry, patient admitted to drinking excessive alcohol and waking up in an "awkward" position in which his head was resting on his forearm without a pillow. Patient was diagnosed with radial nerve entrapment, also known as "Saturday night palsy," and was discharged home with a splint and instructions for self-management.[149]

Neuropathies are traditionally diagnosed on the basis of the findings from the clinical history, physical examination, and electrodiagnostic study.[150] Other pathologies, such as lateral epicondylalgia, medial collateral ligament pathology, and apophysitis, can mimic peripheral neuropathies around the elbow and must be ruled out during the examination process.

A “double crush” syndrome refers to multiple sites of compression along the course of a peripheral nerve. Individually, each mechanical stress is not enough to produce symptoms, but collectively they can result in clinical findings. A thorough examination of the nerve throughout the entire kinetic chain is often needed to appropriately diagnose and treat neuropathies.[151] Refer to Table 8–6 for a list of common elbow entrapments and mechanical causes.

Table 8-6

Common Sites of Peripheral Neuropathies of the Elbow

Summary

- The interrelationship between the elbow complex and the wrist and hand complex makes normal functioning of the elbow vitally important. If elbow function is impaired, function of the hand or joints, such as the shoulder and trunk, may be impaired as well. For example, if the elbow cannot be flexed, it is impossible for the

hand to bring food to the mouth. Alternatively, if the shoulder is limited in motion, then injury related to valgus stress at the elbow may result.

- Because many important vascular and neural structures that supply the hand are closely associated with the elbow, it is important to prevent excessive stress and to protect the elbow from injury. If the radial nerve is injured at the level of the epicondyle, the wrist extensors, supinator, and thumb and finger extensors will be affected. If the median nerve is injured at the level of the elbow, the pronators, flexor carpi radialis, finger flexors, thenar muscles, and lumbricals will be affected. In the following chapter, we will discuss the specific functions of the hand muscles and will be better able to appreciate the significance of injury to some of the muscles associated with the elbow complex.

- Some of the interrelationships between the structure and function of elbow, shoulder, wrist, and hand have been introduced in this chapter. Muscles that have their primary actions at the wrist and hand also cross the elbow and contribute to its stability and function, whereas the stability and ROM at the shoulder and elbow help to enhance the function of the wrist and hand.

- Compensations at the elbow complex often are necessary when the ROM is limited at the shoulder or wrist. New relationships for the joints and muscles of the upper extremity will be introduced in the detailed study of the wrist and hand that follows in the next chapter.

Study Questions

1. Name and locate all of the articulating surfaces of the joints of the elbow complex and describe the method of articulation at each joint, including axes of motion and degrees of freedom.

2. Describe the articulation at the humeroradial joint between the radial head, capitulum, and annular ligament in supination and pronation, with the elbow flexed and extended.

3. Explain the stabilizing function of the brachioradialis by diagramming the translatory and rotatory components for this muscle at different elbow joint angles.

4. Explain why active elbow flexion is more limited than passive flexion. Which structures limit extension?

5. Describe the “carrying angle” of the elbow and explain why it is present.

6. Which structures limit supination and pronation?

7. If slow pronation of the forearm is attempted without resistance, which muscle(s) will be used?

8. How does the structure and function of the annular ligament differ from that of the other elbow joint ligaments?

9. Describe the activity of the biceps brachii during the entirety of a chin-up exercise and the activity of the triceps during the entirety of a push-up exercise.

10. Describe a common mechanism of injury in what is commonly called tennis elbow.

11. Provide a rationale for considering joints proximal to the elbow when treating lateral epicondylalgia.

12. Explain why the term *epicondylosis* or *epicondylalgia* may be a more appropriate diagnostic term than *epicondylitis* for tennis elbow.

13. Compare the medial and lateral collateral ligaments on the basis of structure and function.

14. Compare the biceps brachii and the brachialis on the basis of structure and function.

15. List the six ossification centers of the elbow in their chronological order of ossification.

16. Describe the mechanism of injury involved in cubital tunnel syndrome.

17. Explain the function of the interosseous membrane.

18. According to Table 8–3, what activity requires the most pronation?

19. What ligaments provide primary support to the distal radioulnar joint?

20. In what position(s) of the shoulder, elbow, and forearm might you expect the biceps brachii to be passively insufficient? What is the consequence of that passive insufficiency?

21. What is the role of the triangular fibrocartilage at the distal radioulnar joint?

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Chapter 9: The Wrist and Hand Complex

Noelle M. Austin

Anatomy Overview

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MUSCLE ACTIONS OF THE WRIST AND FINGERS (DIGITS 2 – 5)

TABLE KEY: Primary movers Secondary movers

Sagittal Plane	Joint	Flexion	Extension
	Radiocarpal Midcarpal Carpometacarpal	Flexor carpi radialis Flexor carpi ulnaris Palmaris longus Flexor digitorum superficialis Flexor digitorum profundus Flexor pollicis longus Abductor pollicis longus	Extensor carpi radialis brevis Extensor carpi radialis longus Extensor carpi ulnaris Extensor digitorum communis Extensor indicis Extensor digiti minimi Extensor pollicis

			longus
	Metacarpophalangeal	Interossei (palmar and dorsal) Lumbricals Flexor digitorum superficialis Flexor digitorum profundus Flexor digiti minimi Opponens digiti minimi	Extensor digitorum communis Extensor indicis Extensor digiti minimi
	Proximal interphalangeal	Flexor digitorum superficialis Flexor digitorum profundus	Extensor digitorum communis Extensor indicis Extensor digiti minimi Lumbricals
	Distal interphalangeal	Flexor digitorum profundus	Extensor digitorum

			communis Extensor indicis Extensor digiti minimi Lumbricals
Frontal Plane		Radial Deviation/ Abduction	Ulnar Deviation/ Adduction
	Radiocarpal Midcarpal Carpometacarpal	Extensor carpi Radialis longus Extensor carpi radialis brevis Extensor indicis Flexor carpi radialis Flexor pollicis longus	Extensor carpi ulnaris Flexor carpi ulnaris Abductor pollicis longus Extensor pollicis brevis Extensor pollicis longus
	Metacarpophalangeal	Dorsal interossei Abductor digiti minimi	Palmar interossei

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Muscles	Proximal Attachment	Distal Attachment
Flexor carpi radialis (FCR)	Medial epicondyle of humerus (common flexor origin)	Base of 2nd metacarpal
Palmaris Longus	Medial epicondyle of humerus (common flexor origin)	Distal half of flexor retinaculum and apex of palmar aponeurosis
Flexor carpi ulnaris (FCU)	Radial head: medial epicondyle of humerus (common flexor origin) Ulnar head: Olecranon and posterior border of ulna (via aponeurosis)	Pisiform, hook of hamate, 5th metacarpal
Extensor carpi radialis longus (ECRL)	Lateral supracondylar ridge of humerus.	Dorsal aspect of base of 2nd metacarpal
Extensor carpi radialis brevis (ECRB)	Lateral epicondyle of humerus (common extensor origin)	Dorsal aspect of base of 3rd metacarpal
Extensor carpi ulnaris (ECU)	Lateral epicondyle of humerus; posterior border of ulna via a shared aponeurosis	Dorsal aspect of base of 5th metacarpal

Flexor digitorum superficialis (FDS)	Humeroulnar head: Medial epicondyle of humerus, ulnar collateral ligament, and coronoid process of ulna Radial head: Superior half of anterior border of radius	Bodies of middle phalanges of medial four digits
Flexor digitorum profundus (FDP)	Proximal three quarters of medial and anterior surfaces of ulna and interosseous membrane	Bases of distal phalanges of medial four digits
Extensor digitorum communis	Lateral epicondyle of humerus (common extensor origin)	Extensor expansions of 2nd–5th digits
Extensor indicis	Posterior surface of distal one-third of ulna and interosseous membrane	Extensor expansion of 2nd digit
Extensor digiti minimi	Lateral epicondyle of humerus (common extensor origin)	Extensor expansion of 5th digit
Abductor pollicis longus	Posterior surface of proximal half of ulna, radius, and interosseous membrane	Base of 1st metacarpal
Extensor pollicis longus	Posterior surface of middle one-third of ulna and interosseous membrane	Dorsal aspect of base of distal phalanx of thumb
Extensor pollicis brevis	Posterior surface of distal one-third of radius and interosseous membrane	Dorsal aspect of base of proximal phalanx of thumb

Flexor pollicis longus	Anterior surface of radius	Base of distal phalanx of thumb
Flexor pollicis brevis	Flexor retinaculum and tubercles of scaphoid and trapezium	Lateral side of base of proximal phalanx of thumb
Adductor pollicis	Oblique head: Bases of 2nd and 3rd metacarpals, capitate, and adjacent carpals Transverse head: Anterior surface of shaft of 3rd metacarpal	Medial side of base of proximal phalanx of thumb
Abductor pollicis brevis	Flexor retinaculum and tubercles of scaphoid and trapezium	Lateral side of base of proximal phalanx of thumb
Opponens pollicis	Flexor retinaculum and tubercles of scaphoid and trapezium	Lateral side of 1st metacarpal
Abductor digiti minimi	Pisiform	Medial side of base of proximal phalanx of 5th digit
Flexor digiti minimi brevis	Hook of hamate and flexor retinaculum	Medial side of base of proximal phalanx of 5th digit
Opponens digiti minimi	Hook of hamate and flexor retinaculum	Medial border of 5th metacarpal
Lumbricals, 1st and 2nd	Lateral two tendons of flexor digitorum profundus (as unipennate muscles)	Lateral sides of extensor expansions of

		2nd–5th digits
Lumbricals, 3rd and 4th	Medial three tendons of flexor digitorum profundus (as bipennate muscles)	Lateral sides of extensor expansions of 2nd–5th digits
Dorsal interossei, 1st–4th	Adjacent sides of two metacarpals (as bipennate muscles)	Bases of proximal phalanges; extensor expansions of 2nd–4th digits
Palmar interossei, 1st–3rd	Palmar surfaces of 2nd, 4th, and 5th metacarpals (as unipennate muscles)	Bases of proximal phalanges; extensor expansions of 2nd, 4th, and 5th digits



Note to Students: It is recommended that you review the pretest questions before reading the chapter to better understand the critical elements of the text. As you complete each section, stop and answer the corresponding questions to test your comprehension. The pretest does not cover all the material in each chapter and should not be used as a sole means of self-assessment of knowledge.

Introduction

The human hand may well surpass all body parts but the brain as a topic of universal interest. The human hand has been characterized as a symbol of power, an extension of intellect, and the seat of the will.[1–3] The symbiotic relation of the mind and hand is exemplified by sociologists' claim that although the brain is responsible for the design

of civilization, the hand is responsible for its formation. The hand cannot function without the brain to control it; likewise, the encapsulated brain needs the hand as a tool of expression. The entire upper limb is subservient to the hand. Any loss of function in the upper limb, regardless of the segment, ultimately translates into diminished function of its most distal joints. The significance of this potential loss has led to the detailed study of the finely balanced intricacies of the normal upper limb and hand.

This chapter will systematically explore the joints of the wrist and hand, delving first into joint structure. Specific anatomical structures will be highlighted, including the bones of the region, the joints that are formed by their interconnections, and the soft tissue structures that support these articulations—including the capsule and ligaments. The osteo- and arthrokinematics will highlight how these structures move, followed by a discussion of how the various muscles in the area interact to enable functional use. Finally, the chapter culminates with a discussion of how all of these elements integrate to provide an incredibly well adapted tool that allows for functional interaction with the environment.

Structure and Function of the Wrist Complex

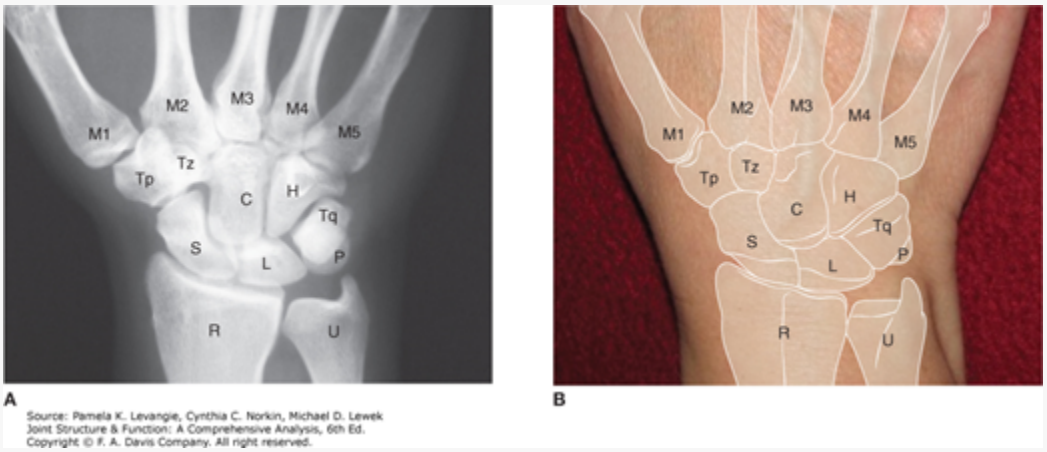
The wrist (**carpus**) consists of two compound joints: the **radiocarpal** and the **midcarpal joints,** referred to collectively as the **wrist complex** (Fig. 9–1; KIA video – Palpation Tutorial Wrist). Each joint proximal to the wrist complex serves to broaden the placement of the hand in space and to increase the degrees of freedom available to the hand. The shoulder serves as a dynamic base of support; the elbow allows the hand to approach or extend away from the body; and the forearm adjusts the approach of the

hand to an object. The carpus, unlike the more proximal joints, serves placement of the hand in space to only a minor degree. The major contribution of the wrist complex is to control length–tension relationships in the multiarticular hand muscles and to allow fine adjustment of grip (KIA video – Active/Passive Insufficiency).[4,5] The wrist muscles appear to be designed for balance and control rather than for maximizing torque production.[6] The adjustments in the length–tension relationship of the extrinsic hand muscles that occur at the wrist cannot be replaced by compensatory movements of the shoulder, elbow, or forearm (radioulnar joint). The wrist has been called the most complex joint of the body, from both an anatomical and physiological perspective.[7] The intricacy and variability of the interarticular and intra-articular relationships within the wrist complex are such that the wrist has received a large amount of attention with agreement on relatively few points. Two points on which there appears to be consensus are (1) that the structure and biomechanics of the wrist, as well as of the hand, vary tremendously from person to person; and (2) that even subtle variations can produce differences in the way a given function occurs. The intent of this chapter, therefore, is less to provide details on what is "normal" and more to describe the wrist complex and hand in such a way that general structure is clear and a conceptual framework is developed within which normal function and pathology can be understood.

FIGURE 9-1

A. Wrist complex as shown on radiograph and **B.** in a schematic representation. The radiocarpal joint is composed of the radius and the radioulnar disc, with the scaphoid (S), lunate (L), and the triquetrum (Tq). The midcarpal joint is composed of the scaphoid,

lunate, and triquetrum with the trapezium (Tp), the trapezoid (Tz), the capitate (C), and the hamate (H).

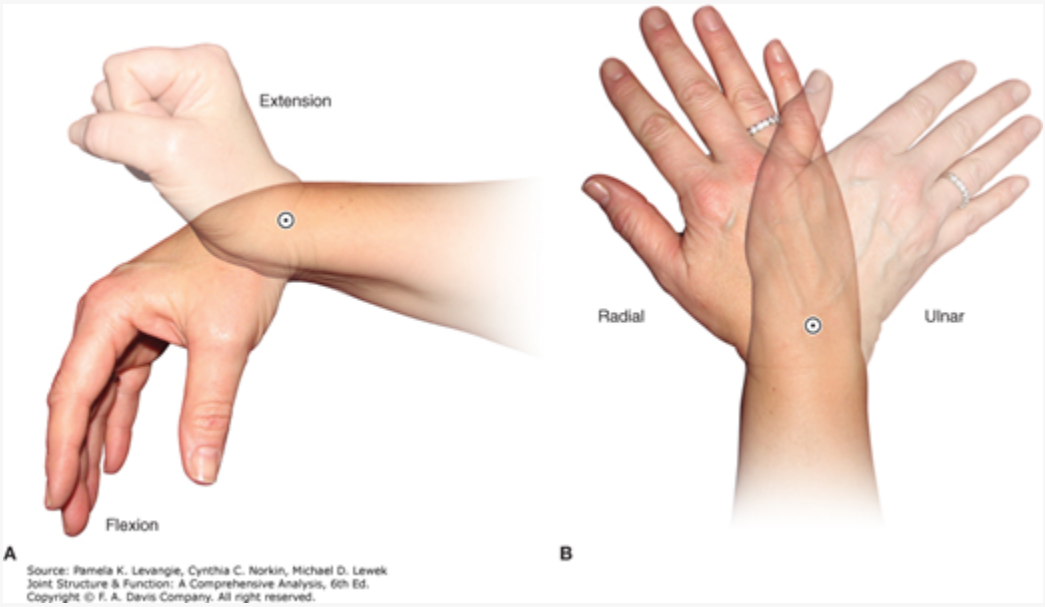


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The wrist complex as a whole is considered biaxial, with motions of **flexion/extension** around a coronal axis and **radial deviation/ulnar deviation** around an anteroposterior axis (Fig. 9–2). A few investigators argue that some degree of **pronation/supination** may also be found, especially at the radiocarpal joint.[8] The ranges of motion (ROMs) of the entire complex are variable and reflect the differences in carpal kinematics that arise from such factors as ligamentous laxity, the shape of articular surfaces, and the constraining effects of muscles.[9] Normal ranges of the wrist complex are cited as a varying 65° to 85° of flexion, 60° to 85° of extension, 15° to 21° of radial deviation, and 20° to 45° of ulnar deviation.[10–13] The ranges are contributed in various proportions by the compound radiocarpal and midcarpal joints. Gilford and colleagues proposed that the two-joint, rather than single-joint, system of the wrist complex: (1) permitted large ROMs with less exposed articular surface and tighter joint capsules, (2) had less tendency for structural pinch at extremes of ranges, and (3) allowed for flatter multijoint surfaces that are more capable of withstanding imposed pressures.[14]

FIGURE 9-2

A. Wrist flexion/extension around a coronal axis; **B.** radial deviation and ulnar deviation around an anteroposterior axis.



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Foundational Concepts 9–1

Nomenclature

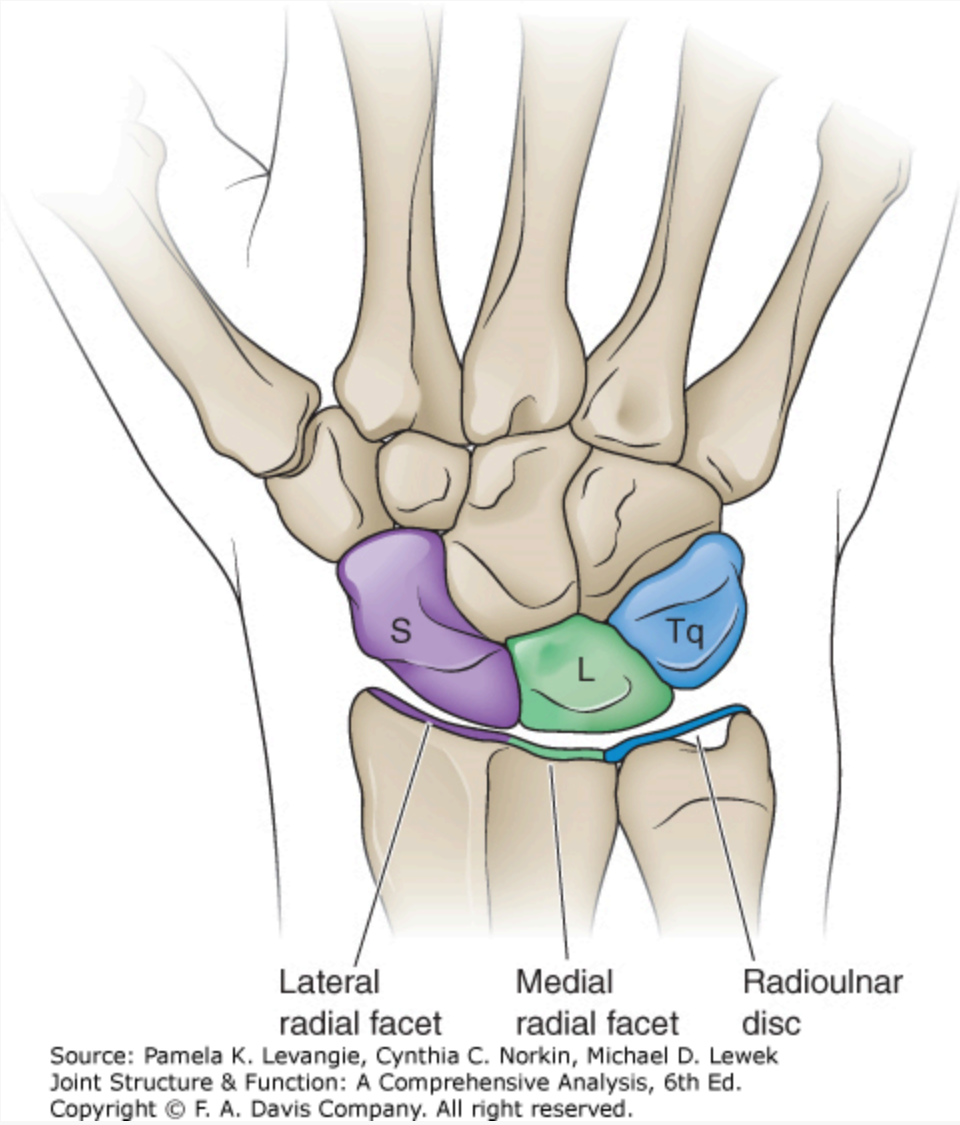
As is true of many other joints of the body, there are variations in nomenclature for the wrist and hand. Flexion/extension of the wrist may also be termed **volar** (**palmar**) **flexion/dorsiflexion,** respectively. Radial/ulnar deviation of the wrist may also be called abduction/adduction, respectively. At the wrist and with joints and structures in the hand, the terms **volar** and **palmar** are used virtually interchangeably, whereas reference to the posterior aspect of the hand is more consistently referred to as the **dorsum.** The terms **medial** and **lateral** may be used in lieu of **ulnar** and **radial.** We will use flexion/extension and radial/ulnar deviation for the wrist motions, although coronal plane motions of the fingers are referred to most commonly as abduction/adduction—a convention that we will follow. The terms **volar** and **palmar** will be used interchangeably to accurately represent terms found in the cited literature.

Radiocarpal Joint Structure

The radiocarpal joint is formed by the **radius** and **radioulnar disc** as part of the **triangular fibrocartilage complex (TFCC)** proximally, and by the **scaphoid, lunate,** and **triquetrum** distally (Fig. 9–3).

FIGURE 9-3

Radiocarpal joint is the articulation of the scaphoid (S) with the lateral radial facet, the lunate (L) with the medial radial facet, and the triquetrum (Tq) with the triangular fibrocartilage complex.



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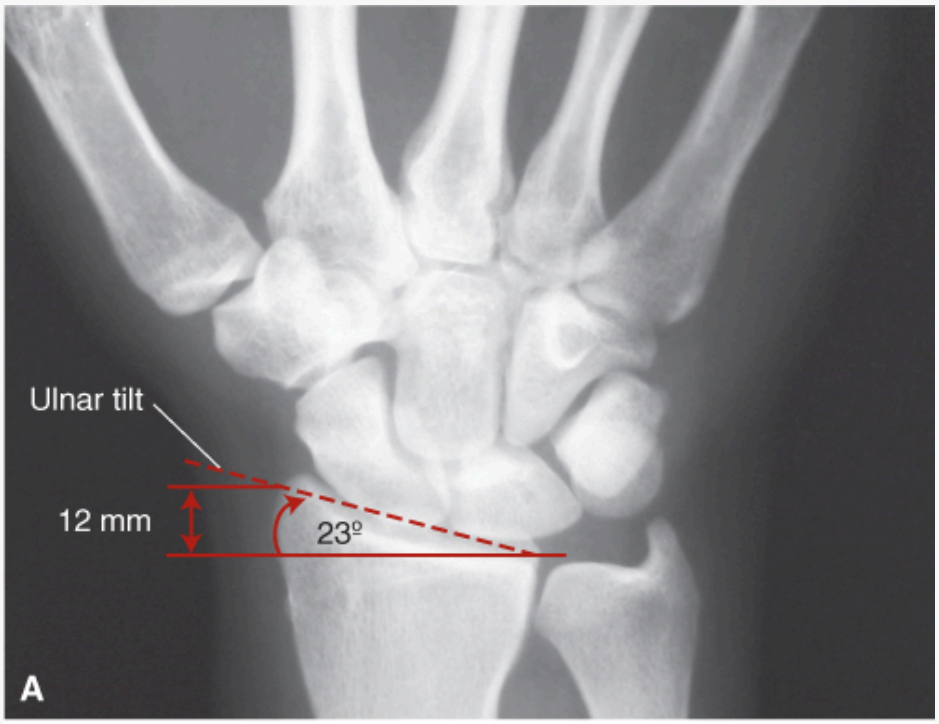
Proximal and Distal Segments of the Radiocarpal Joint

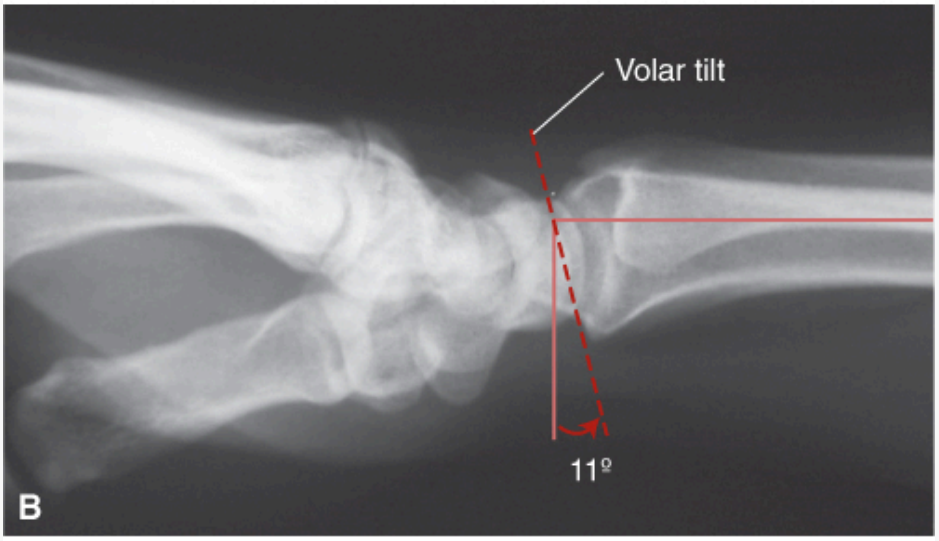
The distal radius has a single, continuous, biconcave curvature that is long and shallow from side to side (in the frontal plane) and shorter and sharper anteroposteriorly (in the sagittal plane). The proximal joint surface is composed of (1) the lateral radial facet that articulates with the scaphoid; (2) the medial radial facet that articulates with the lunate; and (3) the triangular fibrocartilage complex that articulates predominantly with the triquetrum, although it also has some contact with the lunate in the neutral wrist (see

Fig. 9–3). The radioulnar disc, a component of the triangular fibrocartilage complex, also serves as part of the distal radioulnar joint, as discussed in the previous chapter. As a whole, the compound proximal radiocarpal joint surface is oblique and angled slightly volarly and ulnarly. The average ulnar tilt of the distal radius is 23° (**Fig. 9–4A**). This inclination occurs because the radial length (height) is 12 mm greater on the radial side than on the ulnar side.[15] The distal radius is also tilted 11° volarly (**Fig. 9–4B**), with the dorsal radius slightly longer than the volar radius.[15]

FIGURE 9-4

A. A normal angle of 23° of inclination of the radius in the frontal plane (ulnarly), with the distal radius about 12 mm longer on the radial side than on the ulnar side. **B.** A normal angulation of inclination of about 11° of the radius volarly in the sagittal plane (volarly).





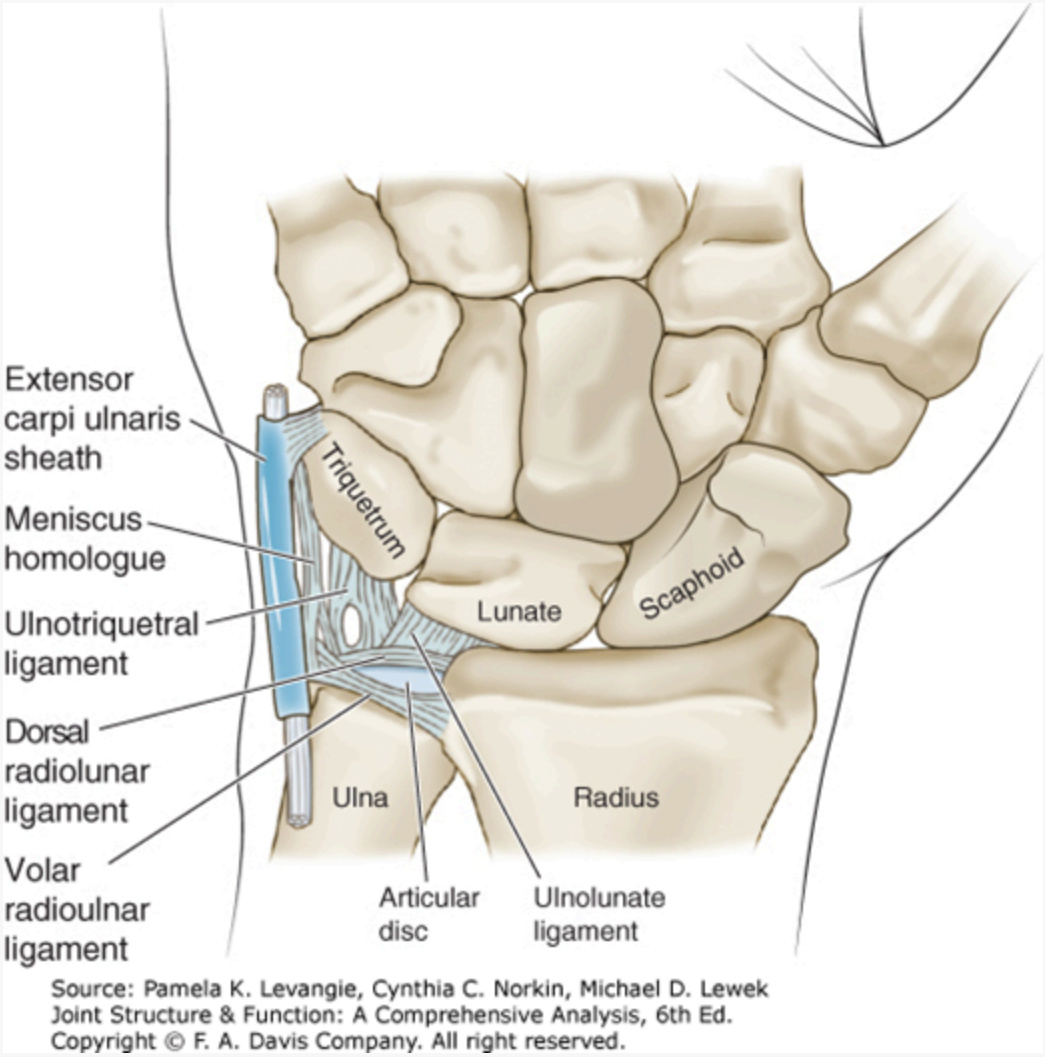
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The triangular fibrocartilage complex consists of the radioulnar disc and the various fibrous attachments that provide the primary support for the distal radioulnar joint (Fig. 9–5).[16] Although the attachments attributed to the triangular fibrocartilage complex vary somewhat, Mohiuddin and Janjua, Benjamin and colleagues, and Palmer provided

descriptions that represent a reasonable consensus.[17–19] The articular disc is a fibrocartilaginous continuation of the articular cartilage of the distal radius. The disc is connected medially via two dense, fibrous connective tissue laminae. The upper laminae include the **dorsal** and **volar radioulnar ligaments** that attach to the ulnar head and ulnar styloid. The lower lamina has connections to the sheath of the **extensor carpi ulnaris (ECU)** tendon and to the triquetrum, **hamate,** and the base of the fifth metacarpal through fibers from the **ulnar collateral ligament.** The so-called **meniscus homolog** is a region of irregular connective tissue that lies within and is part of the lower lamina, traversing volarly and ulnarly from the dorsal radius to insert on the triquetrum. Along its path, the meniscus homolog has fibers that insert into the ulnar styloid and contribute to the formation of the **prestyloid recess.**[20] The medial (ulnar) connective tissue structures may exist in lieu of more extensive fibrocartilage because connective tissue is more compressible than fibrocartilage and thus less likely to limit ROM.[17] Overall, the triangular fibrocartilage complex should be considered to function at the wrist as an extension of the distal radius, just as it does at the distal radioulnar joint.

FIGURE 9-5

The triangular fibrocartilage complex, including the articular disc with its various fibrous attachments, provides support to the distal radioulnar joint.



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The scaphoid, lunate, and triquetrum compose the proximal carpal row that articulate with the distal radius (see Figs. 9–1A and 9–3; KIA video – Carpal Bones of the Wrist). The proximal carpals are interconnected by two ligaments that, like the carpals themselves, are covered with cartilage proximally.[21] These ligaments are the **scapholunate interosseous** and the **lunotriquetral interosseous ligaments.** The proximal carpal row and ligaments together appear to be a single biconvex cartilage-covered joint surface that, unlike a rigid segment, can change shape somewhat to accommodate to the varying; space between the forearm and hand.[22] The **pisiform,** anatomically part of the

proximal row, does not participate in the radiocarpal articulation and functions entirely as a sesamoid bone, presumably to increase the moment arm of the **flexor carpi ulnaris (FCU)** tendon that envelops it.

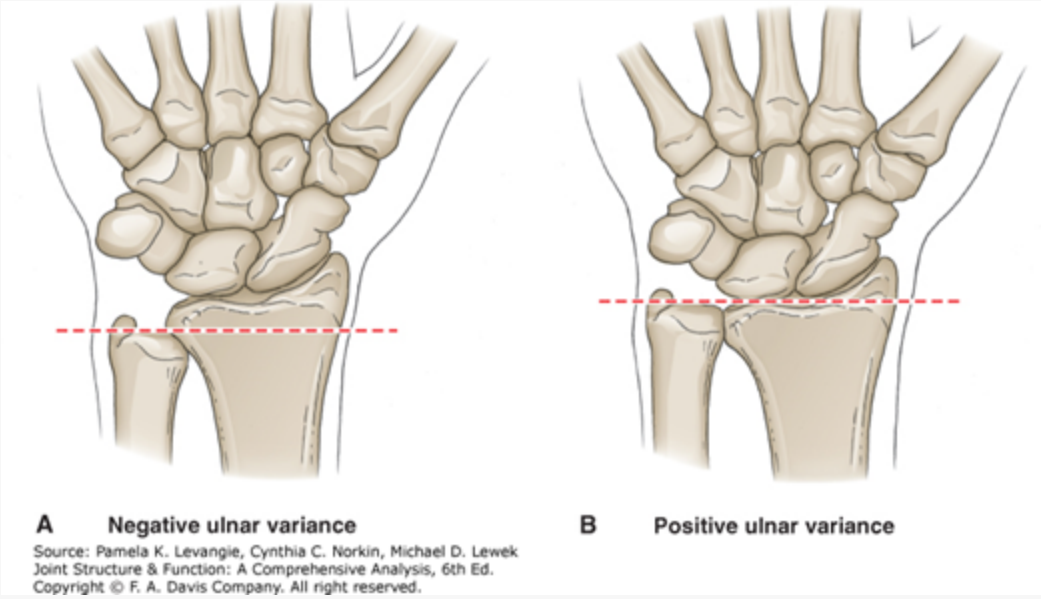
The curvature of the distal radiocarpal joint surface is sharper than the proximal joint surface in both the sagittal and coronal planes, which makes the radiocarpal joint somewhat incongruent. The concept of articular incongruence is supported by the finding that the overall contact between the proximal and distal radiocarpal surfaces is typically only about 20% of the available surface, with never more than 40% of available surface in contact at any one time.[23] Joint incongruence and the angulation of the proximal joint surface result in a greater range of radiocarpal flexion than extension and in greater radiocarpal ulnar deviation than radial deviation.[21,24] The total range of wrist flexion/extension is greater than the total range of wrist radial/ulnar deviation. Incongruence and ligamentous laxity may account for as much as 45° of combined passive pronation/supination at the radiocarpal and midcarpal joints together, although this motion is rarely considered to be an additional degree of freedom available to the wrist complex.[8] When an axial (longitudinal compressive) load is applied to the typical wrist, the scaphoid and lunate receive approximately 80% of the load, whereas the triangular fibrocartilage complex receives approximately 20%.[16,23,25,26] At the distal radius, 60% of the contact is made with the scaphoid and 40% with the lunate.[23]

Not only do the curvature and inclination of the radiocarpal surfaces affect function, but the length of the ulna in relation to the radius is also a factor.[27,28] **Ulnar negative variance** is described as a short ulna in comparison with the radius at the distal end,

whereas in **ulnar positive variance,** the distal ulna is long in relation to the distal radius (Fig. 9–6).[29] An ulnar positive variance has been associated with changes in the triangular fibrocartilage complex thickness.[30] With an ulnar positive variance, there is a potential for impingement of the triangular fibrocartilage complex structures between the distal ulna and the triquetrum.[27] Palmer and colleagues found an inverse relationship between the thickness of the triangular fibrocartilage complex and ulnar variance, with positive ulnar variance associated with a thinner triangular fibrocartilage complex and negative ulnar variance with a relatively thicker triangular fibrocartilage complex.[31] A relatively "long" ulna may be present after a fracture of the distal radius that healed in a shortened position. Pain is commonly present at end range pronation and ulnar deviation because these motions increase the likelihood of impingement of the ulnar structures. Surgical intervention may include a joint-leveling procedure, such as ulnar shortening, to unload the ulnar side of the wrist.[32]

FIGURE 9-6

Ulnar variance. **A.** Negative and **B.** positive

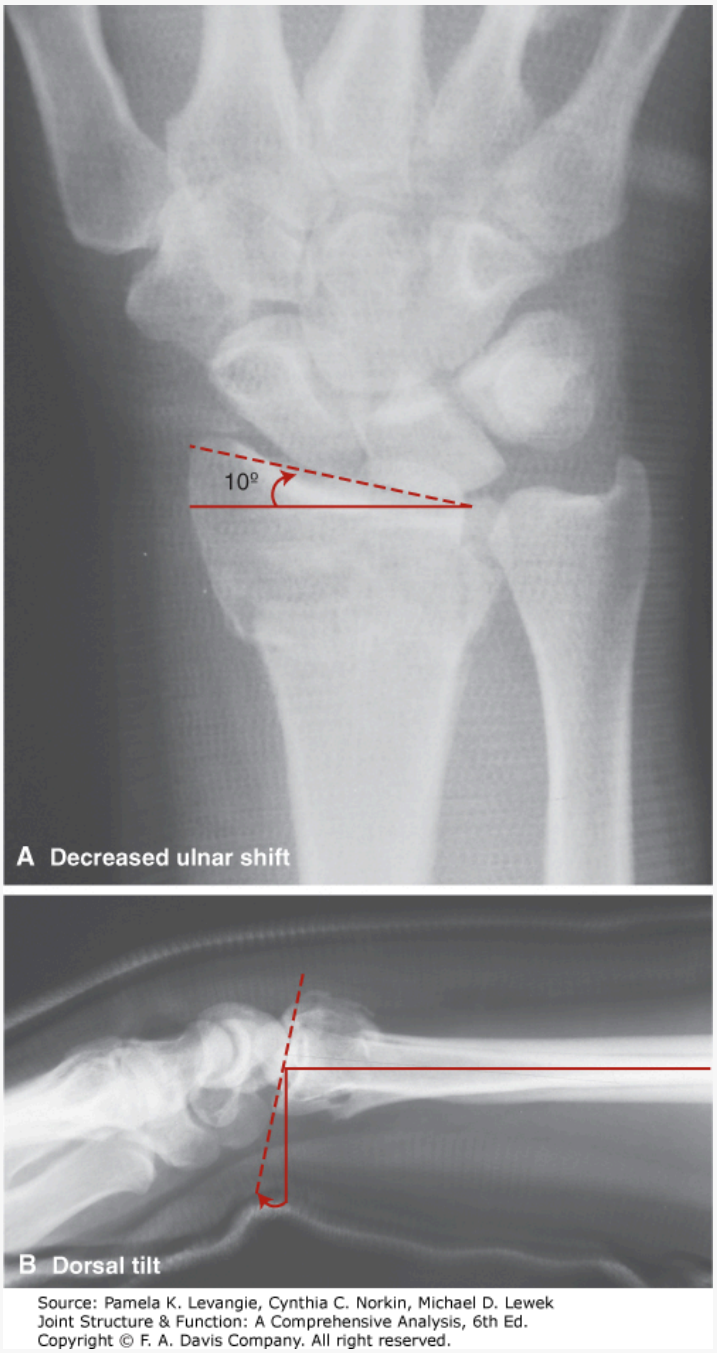


Patient Application 9–1

Gail Antanasi is a 26-year-old who sustained a right distal radius fracture after a **f**all **o**n an **o**utstretched **h**and (known by the acronym **FOOSH**). A posteroanterior radiograph demonstrated a loss in length of the radius and a reduction in the normal ulnar tilt (Fig. 9–7A). The normal volar tilt of the distal radius is now dorsally angulated in the postreduction lateral radiograph (Fig. 9–7B). These changes would be likely to result in a loss of ROM and the likelihood of future joint degeneration. Restoring the articular surfaces to near anatomical position (and correcting the relative lengths of the radius and ulna) would most likely require open reduction and internal fixation (ORIF) with plate and screws.

FIGURE 9-7

A. A radial fracture from a fall on an outstretched hand (FOOSH) resulting in diminished ulnar angulation and length of the distal radius (ulnar positive variance). **B.** Relative shortening of the radius also results in a reversal of the normal volar inclination of the radius.



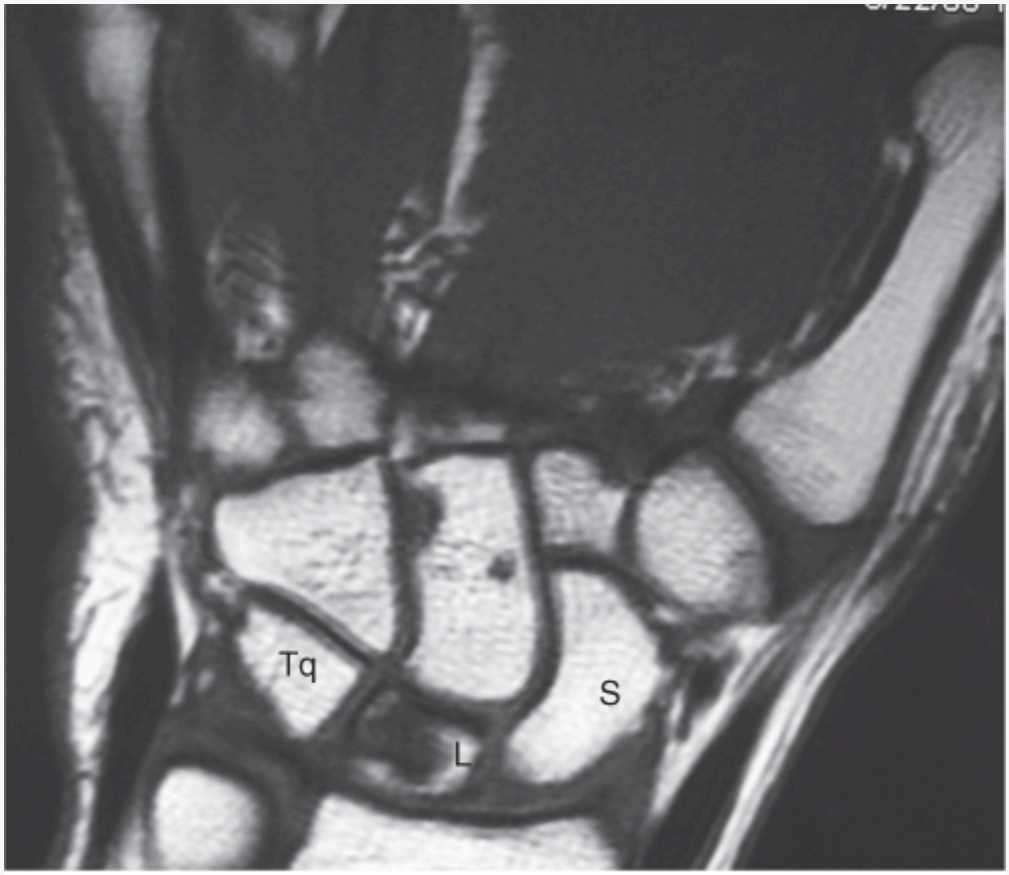
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In contrast to ulnar positive variance, ulnar negative variance (a relatively short ulna) may result in abnormal force distribution across the radiocarpal joint with potential degeneration at the radiocarpal joint.[33] **Avascular necrosis** of the lunate, **Kienbock's disease** (Fig. 9–8), has been associated with ulnar negative variance.[32,33] Treatment

options include unloading of the radiocarpal joint by lengthening the ulna, shortening the radius, or fusing select carpal bones.[32,34]

FIGURE 9-8

In this magnetic resonance image of the wrist, the dark area seen on the lunate demonstrates avascular nectosis, known as Kienbock's disease, and has been associated with negative ulnar variance. L, lunate; S, scaphoid; Tq, triquetrum.



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Radiocarpal Capsule and Ligaments

The radiocarpal joint is enclosed by a strong but somewhat loose capsule and is

reinforced by capsular and intracapsular ligaments. Most ligaments that cross the radiocarpal joint also contribute to stability at the midcarpal joint, and so all the ligaments will be presented together after introduction of the midcarpal joint. Similarly, the muscles of the radiocarpal joint also function at the midcarpal joint. In fact, the radiocarpal joint is not crossed by any muscles that act on the radiocarpal joint alone. The flexor carpi ulnaris is the only muscle that crosses the radiocarpal joint and attaches to any of the bones of the proximal carpal row. Although fibers of the flexor carpi ulnaris tendon end on the pisiform, the pisiform is only loosely connected to the triquetrum below it.[35] Consequently, forces applied to the pisiform by the flexor carpi ulnaris muscle are translated not to the triquetrum on which the pisiform sits but to the **hamate** and fifth metacarpal via pisiform ligaments. Motions occurring at the radiocarpal joint are a result of forces applied by the abundant passive ligamentous structures and by muscles that are attached to the distal carpal row and metacarpals. Consequently, movements of the radiocarpal and midcarpal joints must be examined together.

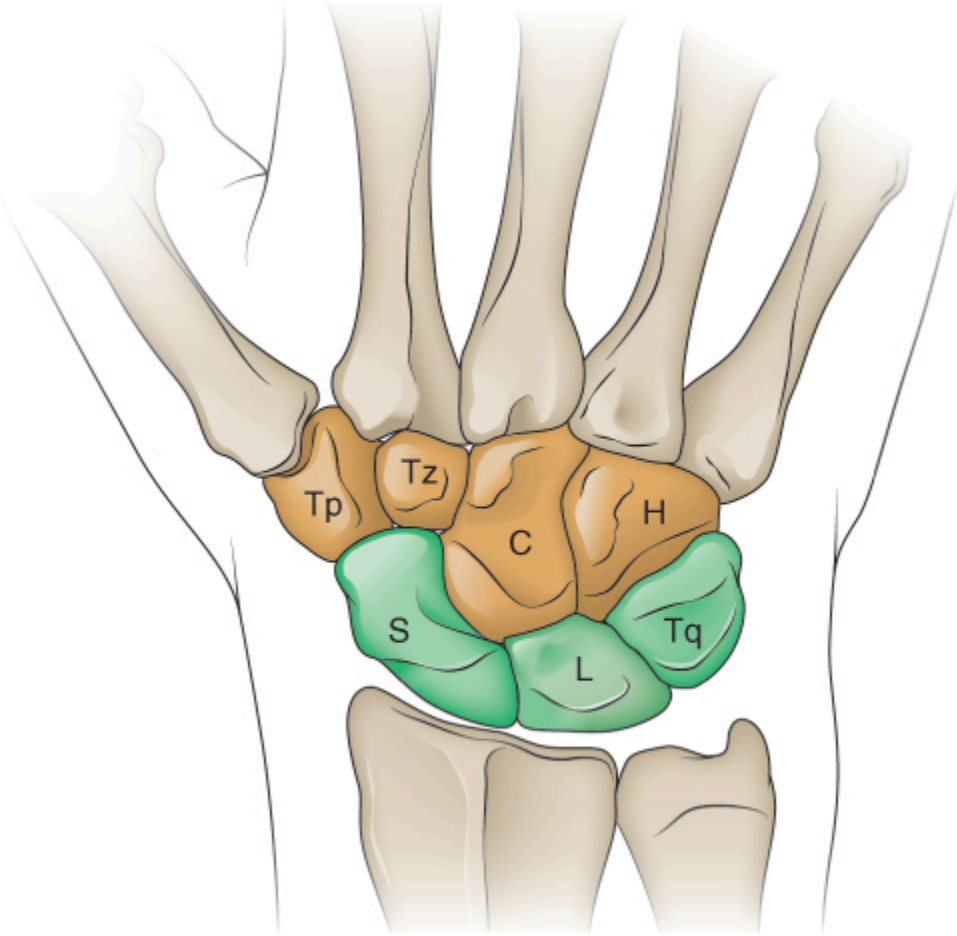
Midcarpal Joint Structure

The midcarpal joint is the articulation between the scaphoid, lunate, and triquetrum proximally and the distal carpal row composed of the **trapezium, trapezoid, capitate,** and hamate (Fig. 9–9). The midcarpal joint is a functional rather than an anatomical unit because it does not form a single uninterrupted articular surface. However, it is anatomically separate from the radiocarpal joint and has a fibrous capsule and synovial lining that is continuous with each intercarpal articulation and may be continuous with some of the **carpometacarpal (CMC) joints.**[21] The midcarpal joint surfaces are complex, with an overall reciprocally concave-convex configuration. The complexity of surfaces

and ligamentous connections, however, requires simplification of the conceptualization of its movements.

FIGURE 9-9

Midcarpal joint is formed proximally by the scaphoid (S), lunate (L), and triquetrum (Tq), and distally by the trapezium (Tp), trapezoid (Tz), capitate (C), and hamate (H).



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Functionally, the carpals of the distal row (with their attached metacarpals) move as an almost fixed unit. The capitate and hamate are most strongly bound together with, at

most, a small amount of play between them.[36–38] The union of the distal carpals also results in nearly equal distribution of loads across the articulations of the scaphoid with the trapezium and lunate, the scaphoid with the capitate, the lunate with the capitate, and the triquetrum with the hamate.[23,39] Together the bones of the distal carpal row contribute two degrees of freedom to the wrist complex, with varying amounts of radial/ulnar deviation and flexion/extension credited to the joint. The excursions permitted by the articular surfaces of the midcarpal joint generally favor the range of extension over flexion and radial deviation over ulnar deviation—the opposite of what was found for the radiocarpal joint.[21,24,40] The functional union of the distal carpals with each other and with their contiguous metacarpals not only serves the wrist complex but also is the foundation for the transverse and longitudinal arches of the hand, which will be addressed in detail later.[37]

Ligaments of the Wrist Complex

The tremendous individual differences that exist in the structure of the carpus can perhaps best be appreciated after a review of the ligaments of the wrist. There are substantive differences in names, anatomical descriptions, and ascribed functions from investigator to investigator.[41–44] We will present the work of Taleisnik to organize and describe the wrist ligamentous anatomy.[45,46] Although there may not be universal agreement on the structure and function of individual ligaments, there is consensus that the ligamentous structure of the carpus is responsible not only for articular stability but also for guiding and checking motion between and among the carpals.[47] When we examine the function of the wrist complex, we shall see that the variability of ligaments will, among other factors, translate into substantial and widely acknowledged differences among individuals in movement of the joints of the wrist

complex.

In general, the dorsal wrist ligaments are described as thin, whereas the more numerous volar ligaments are thicker and stronger.[45,46] Both volar and dorsal ligaments are designated as either extrinsic or intrinsic.[41,45,46] The **extrinsic ligaments** are those that connect the carpals to the radius or ulna proximally or to the metacarpals distally; the **intrinsic ligaments** are those that interconnect the carpals themselves and are also known as **intercarpal** or **interosseous ligaments.** Nowalk and Logan found the intrinsic ligaments to be stronger and less stiff than the extrinsic ligaments.[41] They concluded that the intrinsic ligaments lie within the synovial lining and, therefore, must rely on synovial fluid for nutrition rather than contiguous vascularized tissues, as do the extrinsic ligaments. The extrinsic ligaments, therefore, are more likely to fail but also have better potential for healing and help protect the slower-to-heal intrinsic ligaments by accepting forces first.[41]

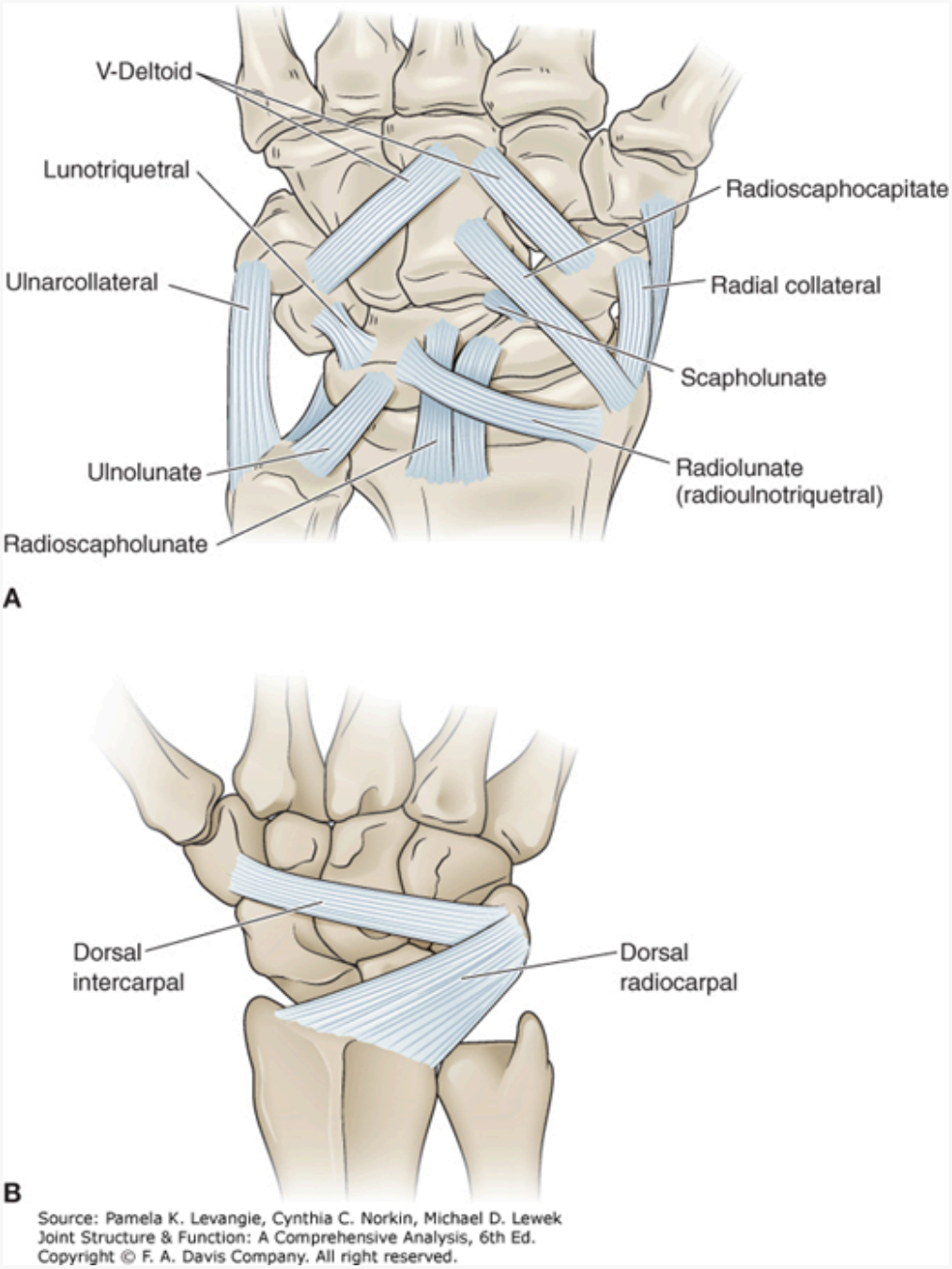
VOLAR CARPAL LIGAMENTS

On the volar surface of the wrist complex, the numerous intrinsic and extrinsic ligaments are variously described by either composite or separate names, depending on the investigator. Taleisnik organized the volar extrinsic ligaments into two groupings: the radiocarpal and the ulnocarpal ligaments (Fig. 9–10A). The composite ligament known as the **volar radiocarpal ligament** is described most commonly as having three distinct bands: the **radioscaphocapitate (radiocapitate),** short and long **radiolunate (radiolunotriquetral),** and **radioscapholunate ligaments.**[45–47] The radioscapholunate ligament was once described as the most important stabilizer of the proximal pole of the scaphoid, and disruption of it may lead to scaphoid instability; however, more

recent research reveals that this structure offers little support to the joint but acts as a conduit for neurovascularity to the scapholunate joint.[46,47] The **radial collateral ligament** may be considered an extension of the volar radiocarpal ligament and capsule.[48] Nowalk and Logan identified the radiocapitate as an extrinsic ligament, whereas Blevens and colleagues identified it as part of the "palmar intracapsular radiocarpal ligaments."[41,43] The **ulnocarpal ligament complex** is composed of the triangular fibrocartilage complex (including the articular disc and meniscus homolog), the **ulnolunate ligament,** and the ulnar collateral ligament.[20,48]

FIGURE 9-10

A. Volar ligaments of the wrist complex, including the three bands of the volar radiocarpal ligament: radioscaphocapitate, radiolunate, and radioscapholunate. The two intrinsic ligaments (scapholunate and lunotriquetral) are credited with maintaining scaphoid stability. **B.** Dorsal wrist ligaments form a horizontal V adding to radiocarpal stability.



Two volar intrinsic ligaments have received particular attention and acknowledgment of their importance to wrist function. The first of these, the **scapholunate interosseous ligament,** is generally, although not universally, credited with being a key factor in maintaining scaphoid stability and, therefore, stability of much of the wrist.[43, 49–52]

Injury to this ligament appears to contribute primarily to scaphoid instability and, therefore, to one of the most common wrist problems.[47,53] As an intrinsic ligament, however, the scapholunate interosseous ligament is largely avascular, therefore may be susceptible to degenerative change.[54] The second key intrinsic ligament is the **lunotriquetral interosseous ligament.** This ligament is credited with maintaining stability between the lunate and triquetrum. Injury to this ligament appears to contribute to lunate instability, another problematic wrist pathology.[55,56] However, this instability pattern probably doesn't occur without concomitant injury to the extrinsic ligaments. In general, the volar wrist ligaments are placed on stretch with wrist extension.[57]

DORSAL CARPAL LIGAMENTS

Dorsally, the major wrist ligament is the **dorsal radiocarpal ligament** (Fig. 9–10B)**.** This ligament, as is true of the volar radiocarpal, varies somewhat in description but is obliquely oriented.[45,46] Essentially, the ligament as a whole converges on the triquetrum from the distal radius, with possible attachments along the way to the lunate and lunotriquetral interosseous ligament.[44,58,59] Garcia-Elias suggested that the obliquity of the volar and dorsal radiocarpal ligaments helps offset the sliding of the proximal "carpal condyle" on the inclined radius.[60] A second dorsal ligament is the **dorsal intercarpal ligament,** which courses horizontally from the triquetrum to the lunate, scaphoid, and trapezium.[48,58] The two dorsal ligaments together form a horizontal V (see Fig. 9–10B) that contributes to radiocarpal stability, notably stabilizing the scaphoid during wrist ROM.[45,58,59] The dorsal wrist ligaments are taut with wrist flexion.[57]

Foundational Concepts 9–2

Summary of Ligaments

- Extrinsic Ligaments
  - Radiocarpal
    - Radial collateral
    - Volar collateral
      - Superficial
      - Deep
        - Radioscaphocapitate
        - Radiolunate (radiolunotriquetral)
        - Radioscaphoid-lunate
    - Ulnocarpal
      - Triangular fibrocartilage complex
      - Meniscus homolog
      - Ulnolunate
      - Ulnar collateral
    - Dorsal radiocarpal (radiotriquetral)
  - Intrinsic Ligaments
    - Short
      - Volar
      - Dorsal
      - Interosseous
    - Intermediate
      - Lunotriquetral
      - Scapholunate
      - Scaphotrapezium
    - Long

Volar intercarpal (v-ligament, deltoid)

Dorsal intercarpal

Function of the Wrist Complex

Movements of the Radiocarpal and Midcarpal Joints

Motions at the radiocarpal and midcarpal joints are caused by a rather unique combination of active muscular, passive ligamentous, and joint reaction forces. Although there are abundant passive forces on the proximal carpal row, no muscular forces are applied directly to the articular bones of the proximal row, given that the flexor carpi ulnaris muscle applies its force via the pisiform to the more distal bones. The proximal carpals, therefore, are effectively a mechanical link that lies between the radius and the distal carpals and metacarpals, to which the muscular forces are actually applied. Gilford and colleagues suggested that the proximal carpal row is an **intercalated segment,** a relatively unattached middle segment of a three-segment linkage.[14] Ruby and associates concurred, hypothesizing that the proximal carpal row functions as an intercalated segment between the distal radius/triangular fibrocartilage complex and the relatively less mobile distal carpal row.[61] When compressive forces are applied across an intercalated segment, the middle segment tends to collapse and move in the opposite direction from the segments above and below. For example, compressive muscular extensor forces would be applied directly to the distal row that then extends, whereas the relatively unstable interposed proximal scaphoid would collapse into flexion. An intercalated segment requires some type of stabilizing mechanism to normalize combined midcarpal/radiocarpal motion and prevent collapse of the middle segment (the proximal carpal row). The stabilization mechanism appears to involve the scaphoid's functional and anatomical (ligamentous) connections to the

adjacent lunate and to the distal carpal row.

Garcia-Elias supported the hypothesis that the stability of the proximal carpal row depends on the interaction of two opposite tendencies when the carpals are axially loaded (compressed across a neutral wrist); the scaphoid tends to flex, whereas the lunate and triquetrum tend to extend.[60] These opposite rotations within the proximal row are prevented by the ligamentous structure, including the key scapholunate interosseous and lunotriquetral interosseous ligaments. Linking the scaphoid to the lunate and triquetrum through ligaments will, according to Garcia-Elias, cause the proximal carpals to "collapse synchronously" into flexion and pronation while the distal carpals move into extension and supination.[60] Garcia-Ellis proposed that the counter rotations between proximal and distal carpal rows and the resulting ligamentous tension increase coaptation of midcarpal articular surfaces and add to stability.[60]

Although the carpal stability mechanism proposed by Garcia-Elias appears to hold as a conceptual framework, findings of other investigators differ in detail if not in substance. Advances in technology, including computer modeling, suggest that intercarpal motion is far more complex and individualistic than was once thought.[62,63] There is general agreement that the three bones of the proximal carpal row do not move as a unit but that motions of the three carpals vary both in magnitude and in direction—whether with axial loading, with radiocarpal flexion/extension, or with radial/ulnar deviation.[7,46,60,64-66] In fact, Short and colleagues found that carpal motions differed not only with individual osteoligamentous configuration and position but also with direction of motion; that is, relations within the carpus differed when the wrist reached neutral position, depending on whether the position was reached from full flexion, full

extension, or deviation.[67]

Controversy remains in terms of the existence of an actual **center of rotation** of the wrist complex. Much of the literature proposes that the head of the capitate, frequently referred to as the **keystone** of the wrist, may serve as the location of the coronal axis for wrist extension/flexion and the anteroposterior (A-P) axis for radial/ulnar deviation, as well as providing the rigid center of the fixed carpal arch.[40,57] Also of importance is the scaphoid and its movement with the capitate, most notably during wrist extension.[67] Neu and associates studied the kinematics of the capitate with wrist motion in both planes and concluded that the axes of motion are not constant, which further supports the premise that carpal kinematics are complex and vary depending on the individual.[68]

FLEXION/EXTENSION OF THE WRIST

During flexion/extension of the wrist, the scaphoid seems to show the greatest motion of the three proximal carpal bones, whereas the lunate moves least.[7,38] Some investigators found that flexion and extension at the radiocarpal joint occurs with more flexion and extension of the scaphoid than the lunate.[51] Others, however, found simultaneous but lesser amounts of radial/ulnar deviation and pronation/supination of two or all three proximal carpal bones during radiocarpal flexion/extension.[7,21,57] Motion of the more tightly bound distal carpals and their attached metacarpals during midcarpal flexion/extension appears to be fairly simple flexion/extension motions, with movement of these distal segments proportional to movement of the hand.[46]

In view of the apparent variability of findings, a conceptual framework for flexion/extension of the wrist is in order. The following sequence of events was proposed by Conwell and provides an explanation of the relative motions of the various segments

and of their interdependence.[69] It can easily be appreciated, however, that the conceptual framework is oversimplified and ignores some of the simultaneous interactions that occur among the key carpal bones.

1. The motion in this conceptual framework (**Fig. 9–11**) begins with the wrist in full flexion. Active extension is initiated at the distal carpal row and at the firmly attached metacarpals by the wrist extensor muscles attached to those bones. The distal carpals (capitate, hamate, trapezium, and trapezoid) glide on the relatively fixed proximal row (scaphoid, lunate, and triquetrum). Although the surface configurations of the midcarpal joint are complex, the distal carpal row effectively glides posteriorly in the same direction as motion of the hand. When the wrist complex reaches neutral (the long axis of the third metacarpal in line with the long axis of the forearm), the ligaments spanning the capitate and scaphoid draw the capitate and scaphoid together into a close-packed position.

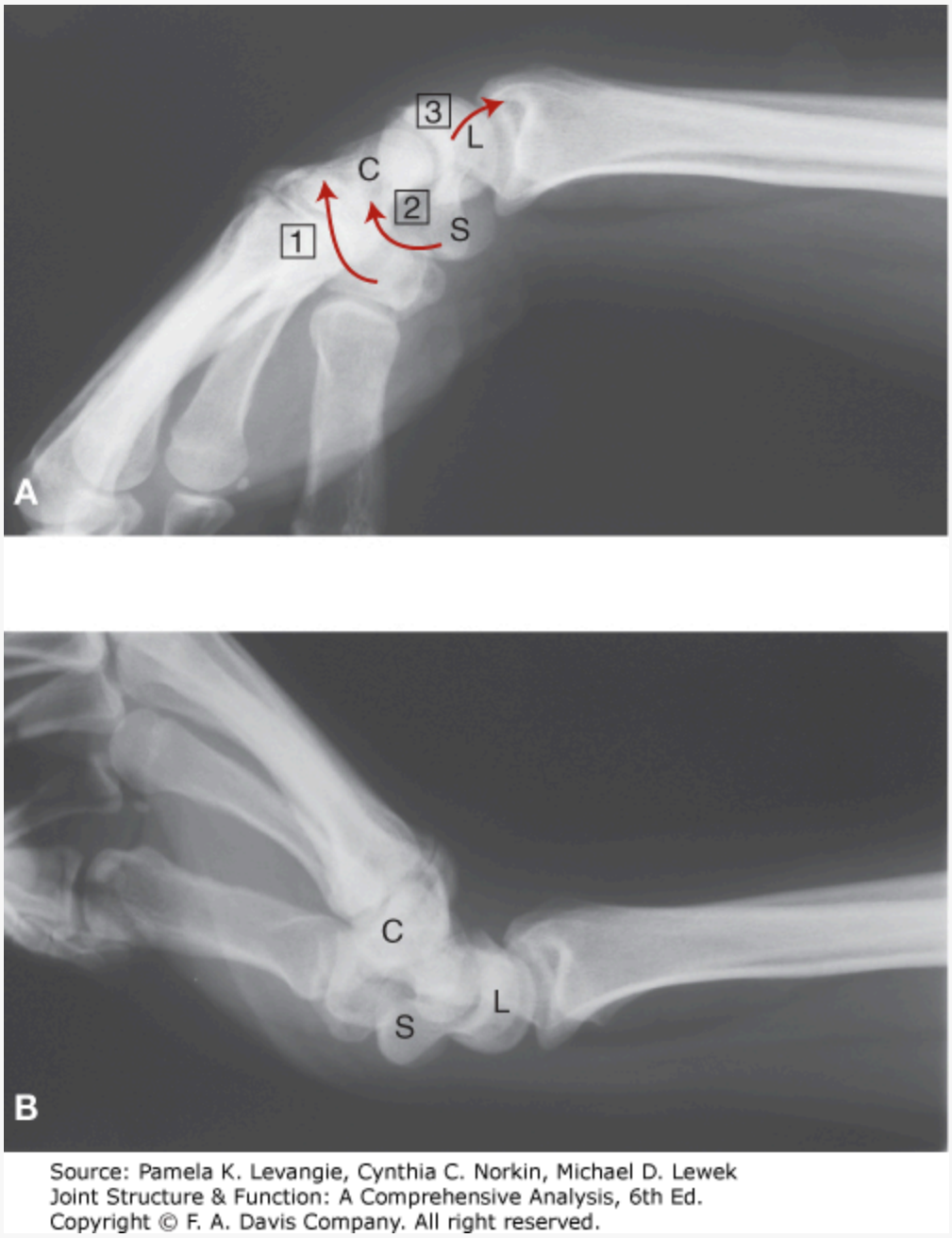
2. Continued extensor force now results in movement of the combined unit of the distal carpal row and the scaphoid on the relatively fixed lunate and triquetrum. At approximately 45° of extension of the wrist complex, the scapholunate interosseous ligament brings the scaphoid and lunate into close-packed position. This unites all the carpals and causes them to function (in the next phase) as a single unit.

3. Completion of wrist complex extension occurs as movement of the proximal carpal row (with the close-packed distal carpal row) moving as a relatively solid unit on the radius and triangular fibrocartilage complex. All ligaments become taut as full extension is reached (Fig. 9–10B) and the entire wrist complex is

close packed.[70]

FIGURE 9-11

A. As wrist extension is initiated from full flexion, (1) the distal carpal row moves on the proximal carpal row until neutral flexion/extension; (2) the scaphoid and distal row move on the lunate/triquetrum from neutral to 45° of extension; and (3) the carpals move as a unit on the radius and triangular fibrocartilage complex to achieve the full wrist extension shown in **B.** C, capitate; L, lunate; S, scaphoid.



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Wrist motion from full extension to full flexion occurs in the reverse sequence. In the context of this conceptual framework, the scaphoid (through mediation of the wrist ligaments) participates at different times in: (1) scaphoid/capitate, (2) scaphoid/lunate, or (3) radio/scaphoid motion. Crumpling of the proximal (intercalated) carpal row is

prevented, and full ROM is achieved. Interestingly, computer modeling and cadaver study of radiocarpal intra-articular contact patterns showed that radiocarpal extension is accompanied by increased contact dorsally. One would expect extension of the hand to be accompanied by sliding of the convex proximal carpal surface volarly in a direction opposite to hand motion. If this contact pattern exists in vivo, it likely reflects the complexity of radiocarpal motion and may contradict assumptions about movement between convex and concave surfaces.[23]

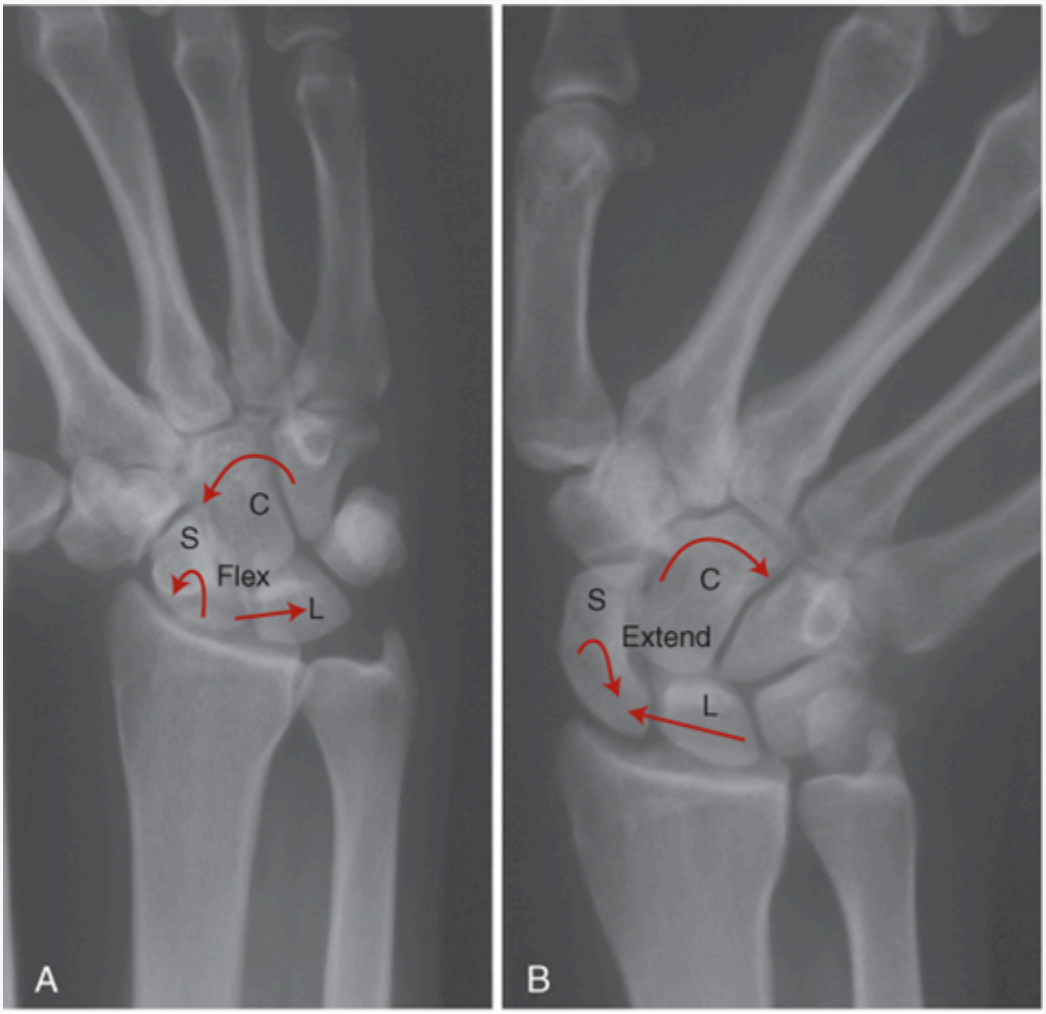
RADIAL/ULNAR DEVIATION OF THE WRIST

Radial and ulnar deviation of the wrist seems to be an even more complex, but perhaps less varied, motion than is wrist flexion/extension. The proximal carpal row displays a unique "reciprocal" motion with radial and ulnar deviation.[12] In radial deviation (**Fig. 9–12A**), the proximal carpal row not only slides ulnarly opposite to wrist motion, but there is also simultaneous flexion of the proximal carpals and extension of the distal carpals (with observations of accompanying pronation/supination components varying among investigators).[7,22,25,71] The opposite motions of the proximal and distal carpals occur with ulnar deviation (**Fig. 9–12B**). During radial/ulnar deviation, the distal carpals—once again—move as a relatively fixed unit whereas the magnitude of motion between the bones of the proximal carpal row may differ.[7,38] Garcia-Elias and colleagues found that the magnitude of scaphoid flexion during radial deviation (and scaphoid extension during ulnar deviation) was related to ligamentous laxity.[9] Volunteer subjects with demonstrated ligamentous laxity showed more scaphoid flexion/extension and less radial/ulnar deviation than did those with less ligamentous laxity. Ligamentous laxity was more common among women than men. The investigators proposed that ligamentous laxity led to less binding of the scaphoid to the

distal carpal row and, therefore, more out-of-plane motion for the scaphoid.

FIGURE 9-12

A. With radial deviation of the wrist (ulnar slide of the proximal carpal row), the flexion of the scaphoid makes the scaphoid appear shorter than **B.,** when the scaphoid extends during ulnar deviation (and radial slide of the proximal carpal row). C, capitate; L, lunate; S, scaphoid.



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In full radial deviation, the radiocarpal and midcarpal joints are in close-packed

position.[40,72–74] The ranges of wrist complex radial and ulnar deviation are greatest when the wrist is in neutral flexion/extension.[75] When the wrist is extended and is in close-packed position, the carpals are all locked, and very little radial or ulnar deviation is possible. In wrist flexion, the joints are loose packed and the bones are splayed. Further movement of the proximal row cannot occur, and, as in extreme extension, little radial or ulnar deviation is possible in the fully flexed position.[76]



Expanded Concepts 9–1

Functional ROM

What appears to be a redundancy in function at the midcarpal and radiocarpal joints ensures maintenance of the minimum ROM required for activities of daily living. Brumfield and Champoux found that a series of hand activities necessary for independence required a functional wrist motion of 10° of flexion and 35° of extension.[77] Ryu and colleagues included a wide range of hand functions in their test battery and determined that all could be completed with minimum wrist motions of 60° extension, 54° flexion, 40° ulnar deviation, and 17° radial deviation.[13] There is consensus that wrist extension and ulnar deviation are most important for wrist activities. Full wrist extension combined with ulnar deviation was also found to constitute the position of maximum scapholunate contact.[23] Given the key role of the scaphoid in wrist stability as a link between the proximal and distal carpal rows, this extended and ulnarly deviated wrist position will provide a stable base that allows for maximum hand function distally. When deciding on the position of fusion for the wrist, the surgeon commonly chooses an optimal functional position of approximately 20° of extension and 10° of ulnar deviation.[57] This extended position also positions the long digital flexors for maximal force generation in prehension activities.

Wrist Instability

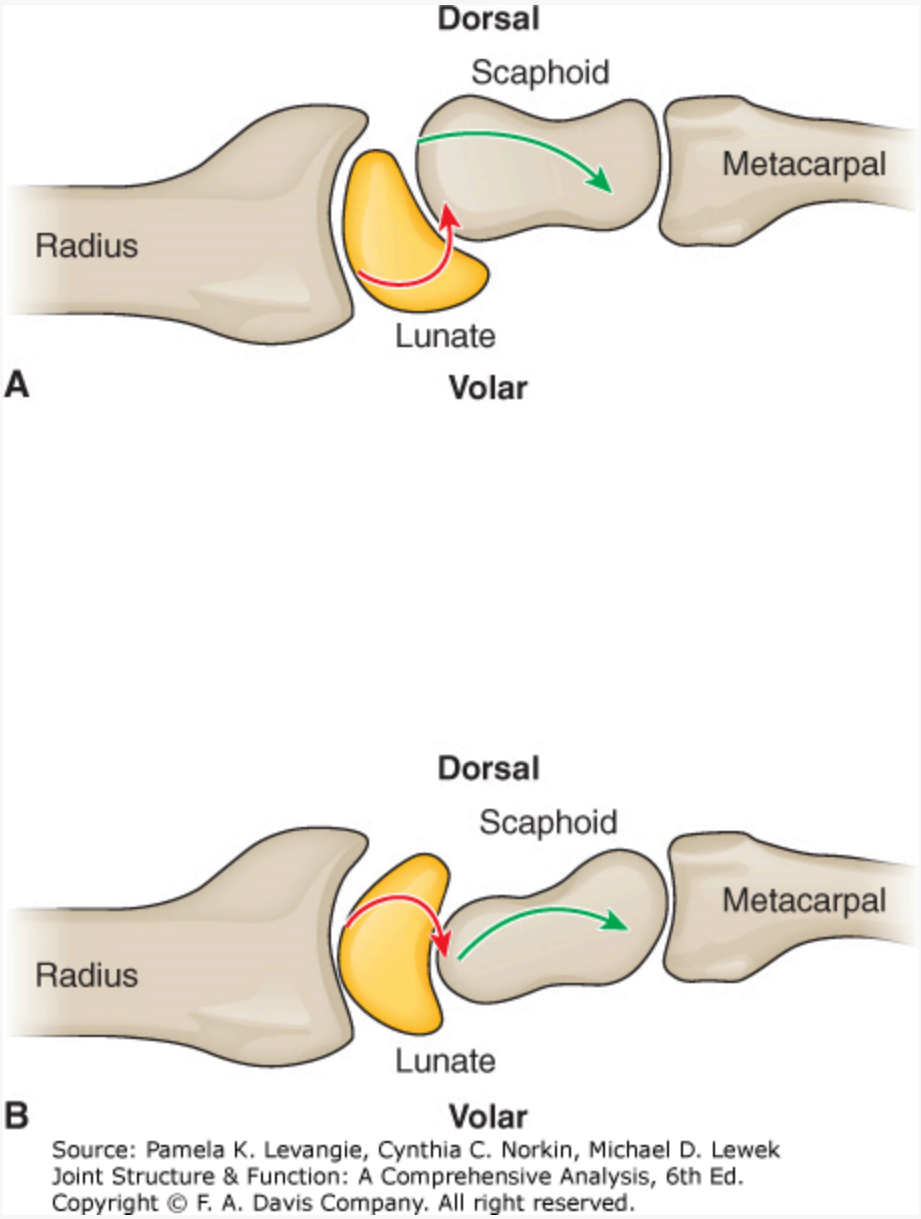
Injury to one or more of the ligaments attached to the scaphoid and lunate may diminish or remove the synergistic stabilization of the lunate and scaphoid.[78,79] When

this occurs, the scaphoid behaves as an unconstrained segment, following its natural tendency to collapse into flexion on the volarly inclined surface of the distal radius (potentially including some out-of-plane motion as well). The base of the flexed scaphoid slides dorsally on the radius and may sublux. Once released from ligamentous stabilization with the scaphoid, the lunate and triquetrum together act as an unconstrained segment, following their natural tendency to extend (rotate dorsally). The muscles applying forces to the distal carpals (none act on the proximal carpals) cause the distal carpals to flex on the extended lunate and triquetrum. The flexed distal carpals glide dorsally on the lunate and triquetrum, accentuating the extension of the lunate and triquetrum. This zigzag pattern of the three segments (the scaphoid, the lunate/triquetrum, and the distal carpal row) is known as **intercalated segmental instability** (Fig. 9–13).[22,25] When the lunate assumes an extended posture, the presentation is referred to as **dorsal intercalated segmental instability (DISI;** Fig. 9–13A**).** The scaphoid subluxation may be dynamic, occurring only with compressive loading of the wrist with muscle forces, or the subluxation may become fixed or static.[80] With subluxation of the scaphoid, the contact pressures between the radius and scaphoid increase because the contact occurs over a smaller area.[23,43] A dorsal intercalated segmental instability problem, therefore, may result over time in degenerative changes at the radioscaphoid joint and then, ultimately, at the other intercarpal joints.[42] With sufficient ligamentous laxity, the capitate may sublux dorsally off the extended lunate or, more commonly, migrate into the gap between the flexed scaphoid and extended lunate. The progressive degenerative problem from an untreated dorsal intercalated segmental instability is known as **scapholunate advanced collapse (SLAC)** wrist.[43,81] The progressive stages have been identified radiographically on the basis of the time lapse

from injury.[81,82] Although it is arguable whether the load between the scaphoid and the lunate increases or decreases with dorsal intercalated segmental instability, there is agreement that the radiolunate articulation is less likely to show degenerative changes than is the radioscaphoid joint.[23,35,51] The lesser tendency toward degenerative changes in the radiolunate joint has been attributed to a more spherical configuration of the radiolunate facets that better center applied loads across the articular surfaces.[43]

FIGURE 9-13

A. Dorsal intercalated segmental instability (DISI). The lunate, released by ligamentous laxity or a tear from the flexed scaphoid, extends (rotates dorsally) on the radius. **B.** Volar intercalated segmental instability (VISI). The lunate and scaphoid both flex (rotate volarly) on the radius when ligamentous laxity or tear separates the lunate from the triquetrum.



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The other common form of carpal instability occurs when the ligamentous union of the lunate and triquetrum is disrupted through injury.[22,80] The lunate and triquetrum together normally tend to move toward extension and offset the tendency of the scaphoid to flex. When the lunate is no longer linked with the triquetrum, the lunate and scaphoid together fall into flexion, and the triquetrum and distal carpal row extend (Fig. 9–13B). This ulnar perilunate instability is known as **volar intercalated segmental**

instability (VTSI).[46] This condition is not as common as dorsal intercalated segmental instability. The problems of VISI and DISI illustrate the importance of proximal carpal row stabilization to wrist function and of maintenance of the scaphoid as the bridge between the distal carpal row and the two other bones of the proximal carpal row.

Patient Application 9–2

Jon Chin is a 32-year-old glazier who plays in a men's softball league. When he was 27 years old, he sustained a fall on an outstretched hand FOOSH that resulted in pain and swelling on the dorsum of the wrist (Fig. 9–14). A radiograph taken in a walk-in clinic a week later showed a separation between his scaphoid and lunate (Fig. 9–14A) that was indicative of ligamentous damage, including tear of the scapholunate interosseous ligament. It was recommended that he see a hand specialist. A follow-up radiograph with the hand surgeon showed dorsal intercalated segmental instability or DISI (Fig. 9–14B). Jon made the decision not to pursue any kind of treatment beyond a period of immobilization. The problem appeared to resolve with time. However, he is now finding that pain in his wrist has increased to the point where he has decided once again to seek medical attention from the hand surgeon. The hand surgeon diagnosed SLAC. Repetitive loading of the wrist (grasping, lifting) caused wrist instability marked by proximal migration of the capitate into the space between the scaphoid and lunate and degenerative changes in the radioscaphoid and capitate-lunate joints (Fig. 9–14C). A partial wrist fusion (scaphocapitate arthrodesis) was recommended to improve stability (removing one source of pain) while allowing limited wrist mobility to minimize loss of function.

FIGURE 9-14

A. With disruption of the scapholunate ligaments through trauma, the scaphoid and lunate migrate apart, leaving a gap (diastasis). **B.** Dorsal intercalated segmental instability (DISI) results in dorsal tilt of the lunate (shown), as well as less evident volar tilt of the scaphoid and

capitate. **C.** Scapholunate advance collapse (SLAC) with migration of the capitate proxim ally and erosion of the radioscaphoid and capitate-lunate joints.



Muscles of the Wrist Complex

The primary role of the muscles of the wrist complex is to provide a stable base for the hand while permitting positional adjustments that allow for optimal length–tension relationships in the long finger muscles.[4,57] Information on a muscle's cross-sectional area and length of moment arm will help facilitate understanding of a muscle's specific

action, force, and torque potential. Many researchers investigated the peak force that could be exerted at the **interphalangeal (IP) joints** of the fingers by the long finger flexors during different wrist positions. Some studies found that the greatest interphalangeal flexor force occurs with ulnar deviation of the wrist (neutral flexion/extension), whereas the least force occurred with wrist flexion (neutral deviation).[2,83,84] Other studies concluded that 20° to 25° of wrist extension with 5° to 7° of ulnar deviation was the optimal range to maximize grip strength output.[85] The muscles of the wrist, however, are not structured merely to optimize the force of finger flexion. If optimizing finger flexor force outweighed other concerns, one might expect the wrist extensors to be stronger than the wrist flexors. Rather, the work capacity (ability of a muscle to generate force per unit of cross-section) of the wrist flexors is more than twice that of the extensors. Again, contrary to expectation if optimizing finger flexor force was the goal, the work capacity of the radial deviators slightly exceeds that of the ulnar deviators.[86] The function of the wrist muscles cannot be understood by looking at any one factor or function; it should be assessed by electromyograph (EMG) in various patterns of use against the resistance of gravity and external loads. We will describe the wrist muscles here, but their function is best understood in the context of later discussion of the synergies between hand and wrist musculature.

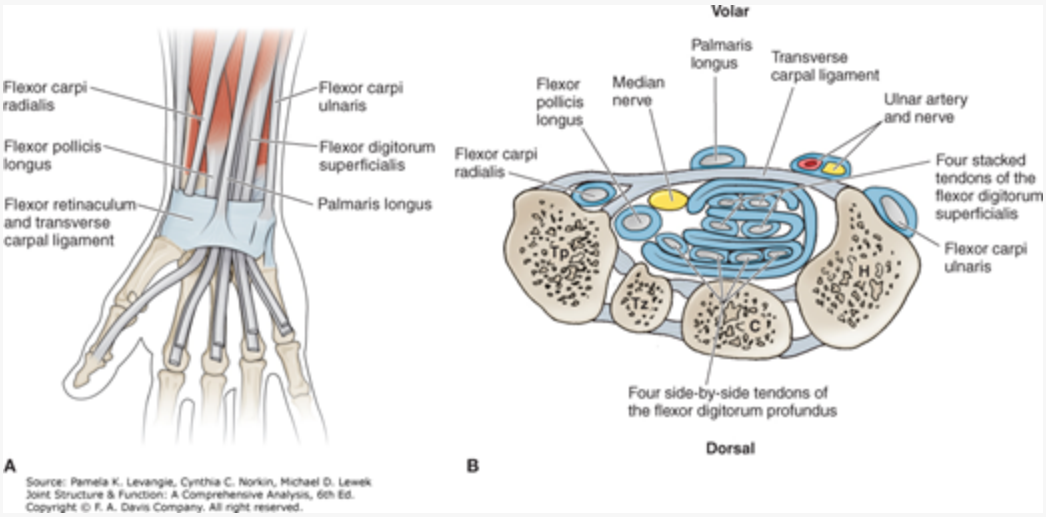
VOLAR WRIST MUSCULATURE

Six muscles have tendons crossing the volar aspect of the wrist and, therefore, are capable of creating a wrist flexion movement (Fig. 9–15). On the volar aspect, these are the **palmaris longus (PL),** the **flexor carpi radialis (FCR),** the flexor carpi ulnaris, the **flexor digitorum superficialis (FDS),** the **flexor digitorum profundus (FDP),** and the **flexor pollicis longus (FPL)** muscles (Fig. 9–15A). The first three of these muscles are primary

wrist muscles. The last three are flexors of the digits with secondary actions at the wrist. At the wrist level, all of the volar wrist muscles pass beneath the **flexor retinaculum** along with the median nerve except the palmaris longus and the flexor carpi ulnaris muscles (Fig. 9–15B). The flexor retinaculum prevents bowstringing of the long flexor tendons, thereby contributing to maintaining an appropriate length–tension relationship. The flexor retinaculum is often considered to have a proximal portion and a distal portion, with the distal portion more commonly known as the **transverse carpal ligament (TCL).**

FIGURE 9-15

A. The tendons of the primary and secondary wrist flexors lie on the volar aspect of the wrist. All but the palmaris longus tendon and the flexor carpi ulnaris muscle pass beneath the flexor retinaculum. **B.** On cross-section, the relationship of the tendons and nerves to the transverse carpal ligament is more evident. The flexor pollicis longus is encased in its own tendon sheath (or radial bursa), whereas the four deep tendons of the flexor digitorum profundus and the four more superficial stacked tendons of the flexor digitorum superficialis are wrapped by folds in the ulnar bursa. ***KIA***



The positions of the flexor carpi radialis and flexor carpi ulnaris tendons in relation to the axis of the wrist indicate that these muscles can, respectively, radially and ulnarly deviate, as well as flex, the wrist. However, the flexor carpi radialis muscle does not appear to be effective as a radial deviator of the wrist in an isolated contraction. Its distal attachment on the bases of the second and third metacarpals places it in line with the long axis of the hand. Along with the palmaris longus muscle, the flexor carpi radialis muscle functions as a wrist flexor with little concomitant deviation.[11] The flexor carpi radialis muscle is active during radial deviation, however. The flexor carpi radialis muscle either augments the strong radial deviating force of the **extensor carpi radialis longus (ECRL)** or offsets the extension also produced by the extensor carpi radialis longus muscle as it radially deviates the wrist. The palmaris longus muscle is a wrist flexor without producing either radial or ulnar deviation. The palmaris longus muscle and tendon are absent unilaterally or bilaterally in approximately 14% of people without any apparent strength or functional deficit.[87] Given its apparent redundancy with other muscles, the palmaris longus tendon (when present) may be “sacrificed” for

surgical reconstruction of other structures.[32]

The flexor carpi ulnaris muscle envelops the pisiform, a sesamoid bone that increases the moment arm of the flexor carpi ulnaris muscle for flexion. The flexor carpi ulnaris muscle can act on the hamate and fifth metacarpal indirectly through the pisiform's ligaments, effectively producing flexion and ulnar deviation of the wrist complex.[35] The flexor carpi ulnaris tendon crosses the wrist at a greater distance from the axis for wrist radial/ulnar deviation than does the flexor carpi radialis muscle, so the flexor carpi ulnaris muscle is more effective in its ulnar deviation function than is the flexor carpi radialis muscle is in its radial deviation function.[4] The flexor carpi ulnaris muscle is able to exert the greatest tension of all the wrist muscles, giving it particular functional relevance, especially with activities requiring high ulnar deviation forces, such as chopping wood.[4]

The flexor digitorum superficialis and flexor digitorum profundus muscles are predominantly flexors of the fingers, and the flexor pollicis longus muscle is predominantly the flexor of the thumb. As multijoint muscles, their capacity to produce an effective wrist flexion force depends on synergistic stabilization of the more distal joints that these muscles cross to prevent excessive shortening of the muscles over multiple joints. If these muscles attempt to shorten over both the wrist and the more distal joints, the muscles will become actively insufficient. The flexor digitorum superficialis and flexor digitorum profundus muscles show varied activity in wrist radial/ulnar deviation, as might be anticipated from the central location of the tendons. The flexor digitorum superficialis muscle seems to function more consistently as a wrist flexor than does the flexor digitorum profundus muscle.[88] This is logical because the

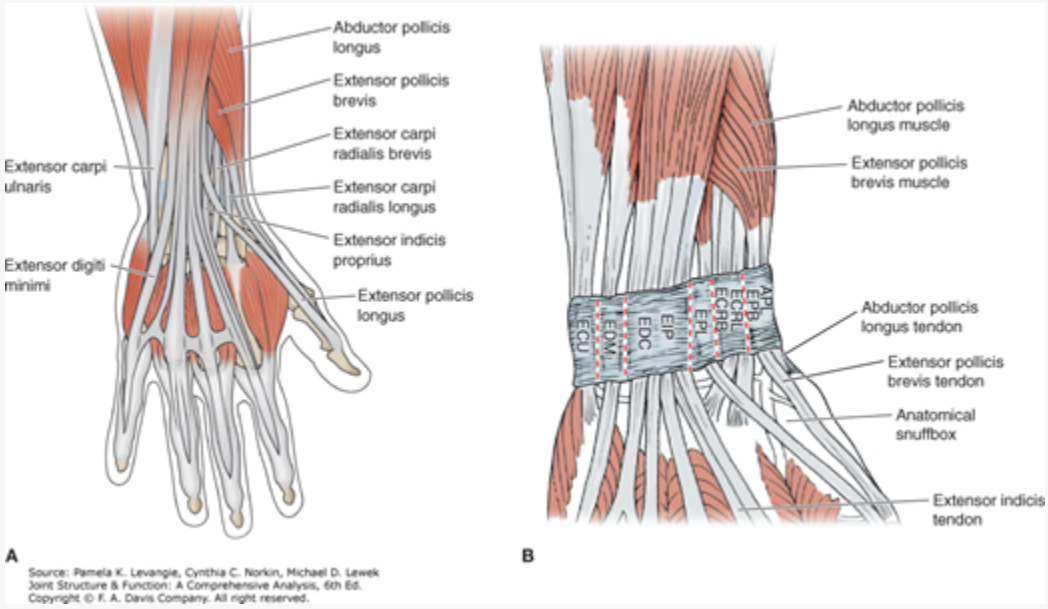
flexor digitorum profundus muscle is a longer, deeper muscle, crosses more joints, and is therefore more likely to become actively insufficient. The effect of the flexor pollicis longus muscle on the wrist has received relatively little attention. The position of this tendon at the wrist suggests the ability to contribute to flexion and radial deviation of the wrist if its more distal joints are stabilized.

DORSAL WRIST MUSCULATURE

The dorsum of the wrist complex is crossed by the tendons of nine muscles (Fig. 9–16). Three of the nine muscles are primary wrist muscles: the extensor carpi radialis longus, the **extensor carpi radialis brevis (ECRB),** and the extensor carpi ulnaris (Fig. 9–16A). The other six are finger and thumb muscles that may act secondarily on the wrist: the **extensor digitorum communis (EDC),** the **extensor indicis proprius (EIP),** the **extensor digiti minimi (EDM),** the **extensor pollicis longus (EPL),** the **extensor pollicis brevis (EPB),** and the **abductor pollicis longus (APL).** The extensor digitorum communis and the extensor indicis proprius muscles are also known, more simply, as the extensor digitorum and the extensor indicis, respectively. The tendons of all nine muscles pass under the **extensor retinaculum,** which is divided into six distinct tunnels by septa (Fig. 9–16B). As the tendons pass deep to the retinaculum, each tendon is encased within its own tendon sheath to prevent friction between the tendons and the retinaculum. The septa of the retinaculum through which the tendons pass are attached to the dorsal carpal ligaments and help maintain stability of the extensor tendons on the dorsum, as well as allowing those muscles to contribute to wrist extension and preventing bowstringing of the tendons with active contraction.[48,49]

FIGURE 9-16

Dorsum of the hand (dorsal retinaculum removed). **A.** The primary wrist extensors are the extensor carpi ulnaris (ECU) and the extensor carpi radialis longus (ECRL) and brevis (ECRB). Finger muscles that can act on the wrist include the extensor digitorum communis (ECU), extensor indicis proprius (EIP), extensor digiti minimi (EDM), extensor pollicus longus (EPL) and brevis (EPB), and the abductor pollicus longus (APL). **B.** The dorsally located extensor tendons pass beneath the extensor retinaculum, where the tendons are compartmentalized.



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The extensor carpi radialis longus and extensor carpi radialis brevis muscles together make up the predominant part of the wrist extensor mass.[90] The extensor carpi radialis brevis muscle is somewhat smaller than the extensor carpi radialis longus muscle but has a more central location as it inserts into the third metacarpal; it generally shows more activity during wrist extension activities than the extensor carpi radialis

longus.[11,91] One study found the extensor carpi radialis brevis muscle to be active during all grasp-and-release hand activities, except those performed in supination.[92] The extensor carpi radialis longus muscle inserts into the more radially located second metacarpal and has a smaller moment arm for wrist extension than does the extensor carpi radialis brevis muscle.[7] The extensor carpi radialis longus muscle shows increased activity when either radial deviation or support against ulnar deviation is required or when forceful finger flexion motions are performed.[70,91] The ongoing activity of the extensor carpi radialis brevis muscle makes it vulnerable to overuse and is more likely than the quieter extensor carpi radialis longus muscle to be inflamed in lateral epicondylitis.[93] The literature has also questioned the role of the extensor digitorum communis muscle in development of this pathology.[94,95]

The extensor carpi ulnaris muscle extends and ulnarly deviates the wrist. It is active not only in wrist extension but frequently in wrist flexion as well.[91] Backdahl and Carlsoo hypothesized that the extensor carpi ulnaris muscle activity in wrist flexion adds an additional component of stability to the structurally less stable position of wrist flexion.[88] This is not needed on the radial side of the wrist that has more plentiful ligamentous and bony structural checks. The connection of the extensor carpi ulnaris tendon sheath to the triangular fibrocartilage complex also appears to help tether the extensor carpi ulnaris muscle and prevent loss of excursion efficiency that would occur with bowstringing.[20] Tang and colleagues found a 30% increase in excursion of the extensor carpi ulnaris muscle after release of the triangular fibrocartilage complex from the distal ulna.[96] The effectiveness of the extensor carpi ulnaris muscle as a wrist extensor is also affected by forearm position. When the forearm is pronated, the crossing of the radius over the ulna causes a reduction in the moment arm of the

extensor carpi ulnaris muscle, making it less effective as a wrist extensor.[4,90,92]

The extensor digiti minimi and the extensor indicis proprius muscles insert into the tendons of the extensor digitorum communis muscle; therefore, they have a common function with the extensor digitorum communis muscle.[97] The extensor indicis proprius and extensor digiti minimi muscles are capable of extending the wrist, but wrist extension is credited more to the extensor digitorum communis muscle, a finger extensor muscle that also functions as an important wrist extensor (without radial or ulnar deviation). There appears to be some reciprocal synergy of the extensor digitorum communis muscle with the extensor carpi radialis brevis muscle in providing wrist extension, because less extensor carpi radialis brevis muscle activity is seen when the extensor digitorum communis muscle is active during finger extension.[91]

Three extrinsic thumb muscles cross the wrist. Both the abductor pollicis longus and the extensor pollicis brevis muscles are capable of radially deviating the wrist and may serve a minor role in that function.[87] However, radial deviation of the wrist may detract from their prime action on the thumb. A synergistic contraction of the extensor carpi ulnaris muscle may be required to offset the unwanted wrist motion when the abductor pollicis longus and extensor pollicis brevis muscles act on the thumb. When muscles producing ulnar deviation are absent, the thumb extrinsic muscles may produce a significant radial deviation deformity at the wrist. Little evidence has been found to indicate that the more centrally located extensor pollicis longus muscle has any notable effect on the wrist.

Now that we have examined the wrist complex, let us look at the hand complex that the wrist serves.

Structure and Function of the Hand Complex

The hand consists of five digits: four fingers and a thumb (Fig. 9–17). Each digit has a carpometacarpal joint and a **metacarpophalangeal (MP)** joint; the fingers each have two **interphalangeal (IP) joints**—the **proximal interphalangeal (PIP)** and the **distal interphalangeal (DIP) joints;** the thumb has one interphalangeal joint. Although the joints of the fingers and the joints of the thumb have structural similarities, function differs significantly enough that the joints of the fingers shall be examined separately from those of the thumb. In examining the joints of the fingers, however, one should be cautious about generalizations. Ranney pointed out that each digit of the hand is unique and that models proposed for and conclusions drawn about one finger may not be accurate for all.[98]

FIGURE 9-17

Bony anatomy of the thumb and fingers. DIP, distal interphalangeal; PIP, proximal interphalangeal; MP, metacarpophalangeal; CMC, carpometacarpal; M, metacarpal; P1, proximal phalanx; P2, middle phalanx; P3, distal phalanx; Tp, trapezium; Tz, trapezpoid; C, capitates; H, hamate.



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Carpometacarpal Joints of the Fingers

The carpometacarpal joints of the fingers are composed of the articulations between the distal carpal row and the bases of the second through fifth metacarpal joints (see Fig. 9–1). The distal carpal row, of course, is also part of the midcarpal joint. The

proximal portion of the four metacarpals of the fingers articulate with the distal carpals to form the second through fifth carpometacarpal joints (see Fig. 9–17). The second metacarpal articulates primarily with the trapezoid and secondarily with the trapezium and capitate. The third metacarpal articulates primarily with the capitate, and the fourth metacarpal articulates with the capitate and hamate. Last, the fifth metacarpal articulates with the hamate. Each of the metacarpals also articulates at its base with the contiguous metacarpal or metacarpals with the exception of the second metacarpal that does not articulate with the contiguous first metacarpal. All finger carpometacarpal joints are supported by strong transverse and weaker longitudinal ligaments volarly and dorsally.[99,100]

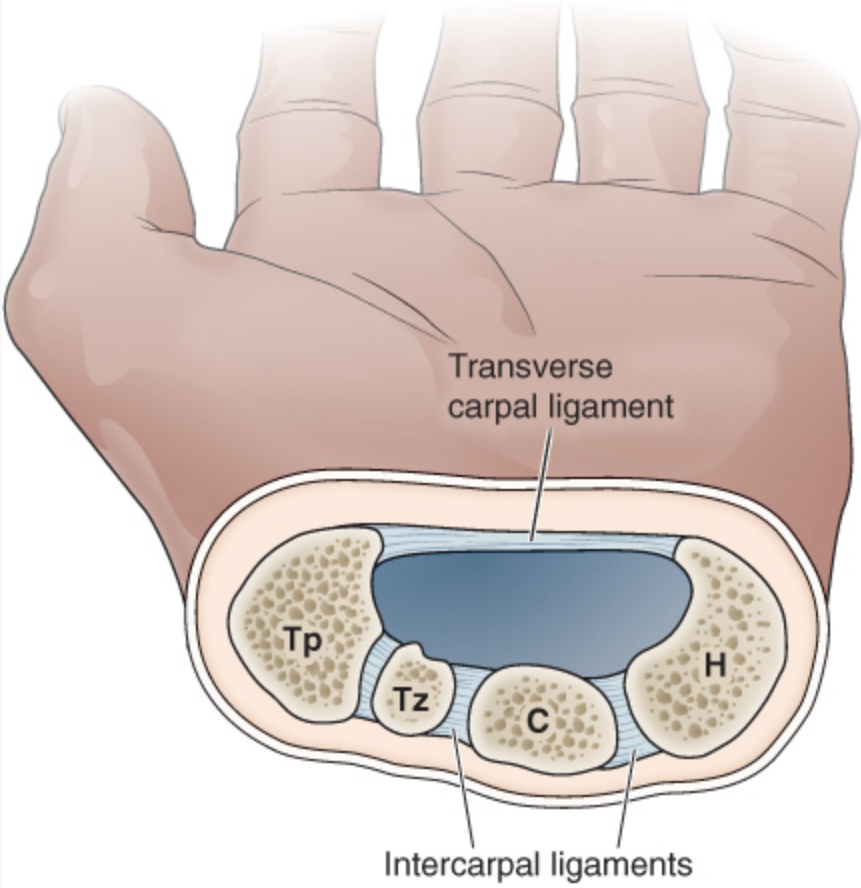
The **deep transverse metacarpal ligament** spans the heads (distal portion) of the second through fourth metacarpals volarly. The deep transverse metacarpal ligament tethers together the metacarpal heads and effectively prevents the attached metacarpals from any more than minimal abduction at the carpometacarpal joints. Although the transverse metacarpal ligament contributes directly to carpometacarpal stability, it is also structurally part of the metacarpophalangeal joints of the fingers and will be discussed again in that context. The capsuloligamentous structure is primarily responsible for controlling the total ROM available at each carpometacarpal joint, although some differences exist from articulation to articulation.

One attribute of the distal carpals that affects carpometa-carpal and hand function but not wrist function is the volar concavity, or **proximal transverse (carpal) arch,** formed by the trapezoid, trapezium, capitate, and hamate (**Fig. 9–18**). The carpal arch persists even when the hand is fully opened and is created not only by the curved shape of the

carpals but also by the ligaments that maintain the concavity. The ligaments that maintain the arch are the transverse carpal ligament and the transversely oriented **intercarpal ligaments.** The transverse carpal ligament is the portion of the flexor retinaculum that attaches to the pisiform and hook of the hamate medially and to the scaphoid and trapezium laterally; the more proximal portion of the flexor retinaculum is continuous with the fascia overlying the forearm muscles. The transverse carpal ligament and intercarpal ligaments that link the four distal carpals maintain the relatively fixed concavity that will contribute to the arches of the palm. These structures also form the **carpal tunnel.** The carpal tunnel contains the median nerve and nine extrinsic flexor tendons of the fingers and thumb (see Fig. 9–15B). A number of intrinsic hand muscles attach to the transverse carpal ligament and bones of the distal carpal row. These may also contribute to maintaining the carpal arch.

FIGURE 9-18

The proximal transverse arch, or carpal arch, forms the tunnel through which the median nerve and long finger flexors travel. The transverse carpal ligament and intercarpal ligaments assist in maintaining this concavity. C, capitate; H, hamate; Tp, trapezium; Tz, trapezoid.



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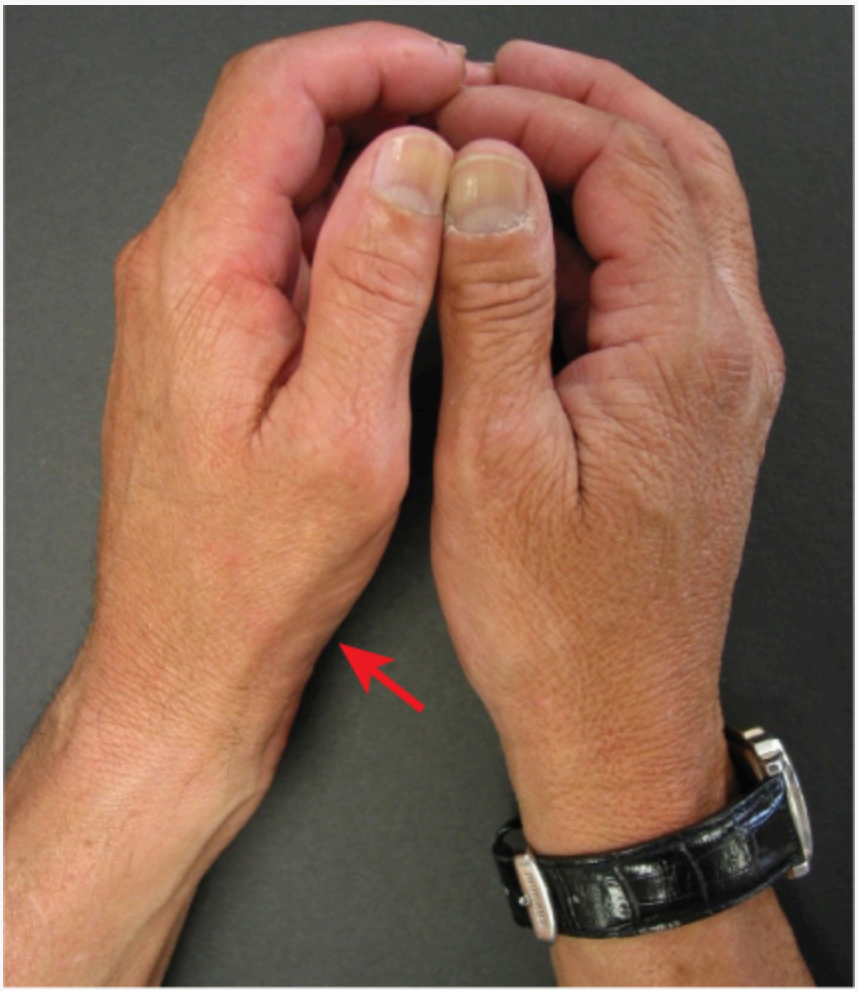
Patient Application 9–3

Jacob Tandesti is a 42-year-old who has been a computer programmer for over 20 years. He spends the majority of his day typing. He reports that he began waking with numbness in his right hand 5 years ago, specifically affecting the thumb as well as the index, middle, and radial half of the ring fingers. He was evaluated by a hand surgeon who found that tapping on the median nerve over the carpal tunnel reproduced his paresthesias [tingling] in the median nerve distribution (positive **Tinel's sign),** as did placing Jacob's wrist in sustained flexion for 1 minute (positive **Phalen's test).** The physician prescribed night splinting and patient education regarding proper ergonomics at work.[101] The orthosis held the wrist in a neutral position at night, which decreased the pressure on the median nerve.[102,103] In the subsequent 6 months, Jacob noted progressive difficulty with completing fine motor tasks such as

buttoning shirts and handling coins. He was referred to a neurologist who performed nerve conduction studies that revealed significant slowing in the median nerve conduction velocity, which was consistent with nerve compression at the wrist level. Also evident was atrophy of median nerve innervated thenar [thumb] muscles, a presentation commonly known as **ape hand** (Fig. 9–19).

FIGURE 9-19

Long-term median nerve compression can lead to atrophy of the median nerve innervated muscles in the thenar eminence, a presentation known as “ape hand” because of the flattening of the palm and the adducted position of the thumb. Notice atrophy on left hand in this clinical example.



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Expanded Concepts 9–2

Carpal Tunnel Syndrome

When the median nerve becomes compressed within the carpal tunnel, a neuropathy known as **carpal tunnel syndrome (CTS)** may develop. Cobb and colleagues proposed that the proximal edge of the transverse carpal ligament is the most common site for wrist flexion induced median nerve compression.[104] The tunnel is narrowest, however, at the level of the hook of the hamate, where median nerve compression is unlikely to be affected by changes in wrist position.[104] When the transverse carpal ligament is cut to release median nerve compression, the carpal arch may widen somewhat, but investigators found that the arch maintains its dorsovolar stiffness as

long as the stronger transverse intercarpal ligaments were intact.[105]

Carpometacarpal Joint Range of Motion

The range of carpometacarpal motion of the second through fifth metacarpals is observable most readily at the metacarpal heads, and shows increasing mobility from the radial to the ulnar side of the hand.[57,106] The second through fourth carpometacarpal joints are plane synovial joints with one degree of freedom: flexion/extension. Although structured to permit flexion/extension, the second and third carpometacarpal joints are essentially immobile and may be considered to have "zero degrees of freedom."[40,98] The fourth carpometacarpal joint has perceptible flexion/extension. The fifth carpometacarpal joint is a saddle joint—typically considered to have two degrees of freedom including flexion/extension and some abduction/adduction, with a limited amount of opposition.[11,98,107] The immobile second and third metacarpals provide a fixed and stable axis about which the fourth and fifth metacarpals and the very mobile first metacarpal (thumb) can move.[36,98,108] The motion of the fourth and fifth metacarpals facilitates the ability of the ring and little fingers to oppose the thumb.

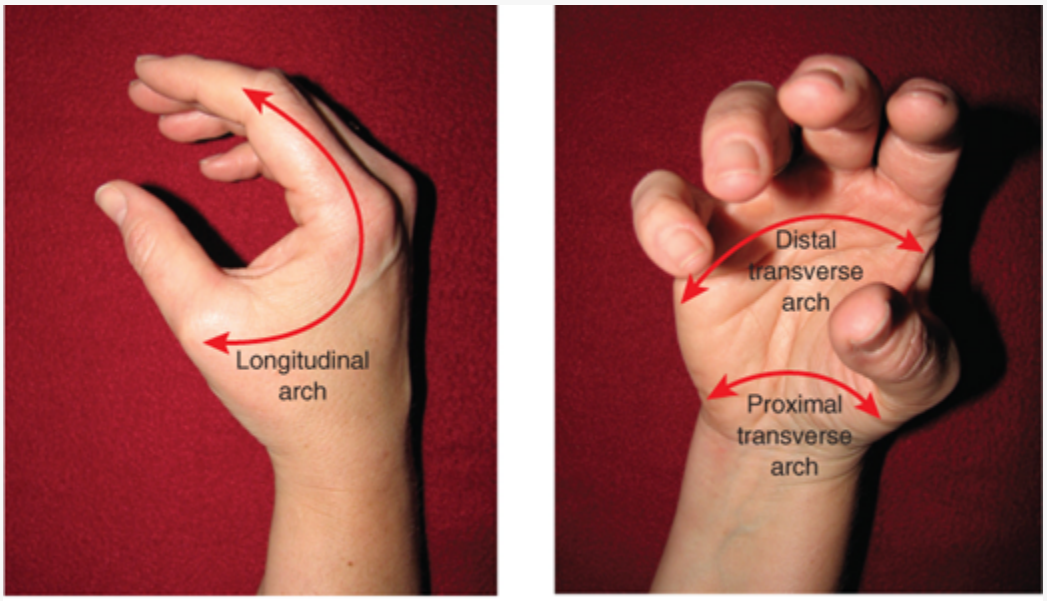
Palmar Arches

The function of the finger carpometacarpal joints and their segments overall is to contribute (with the thumb) to the **palmar arch system** (Fig. 9–20). The concavity formed by the carpal bones results in the proximal transverse arch of the palm of the hand. The adjustable positions of the first, fourth, and fifth metacarpal heads around the relatively fixed second and third metacarpals form a mobile **distal transverse arch** at the level of the metacarpal heads that augments the fixed proximal transverse arch of

the distal carpal row. The **longitudinal arch** traverses the length of the digits from proximal to distal and is inclusive of the fingers. The deep transverse metacarpal ligament contributes to stability of the mobile arches during grip functions.[109] The palmar arches allow the palm and the digits to conform optimally to the shape of the object being held.[110] This maximizes the amount of surface contact with the object, enhancing stability as well as increasing sensory feedback.

FIGURE 9-20

The palmar arch system assists with functional grasp. The proximal transverse arch is fixed, whereas the distal transverse arch and the longitudinal arch are mobile.



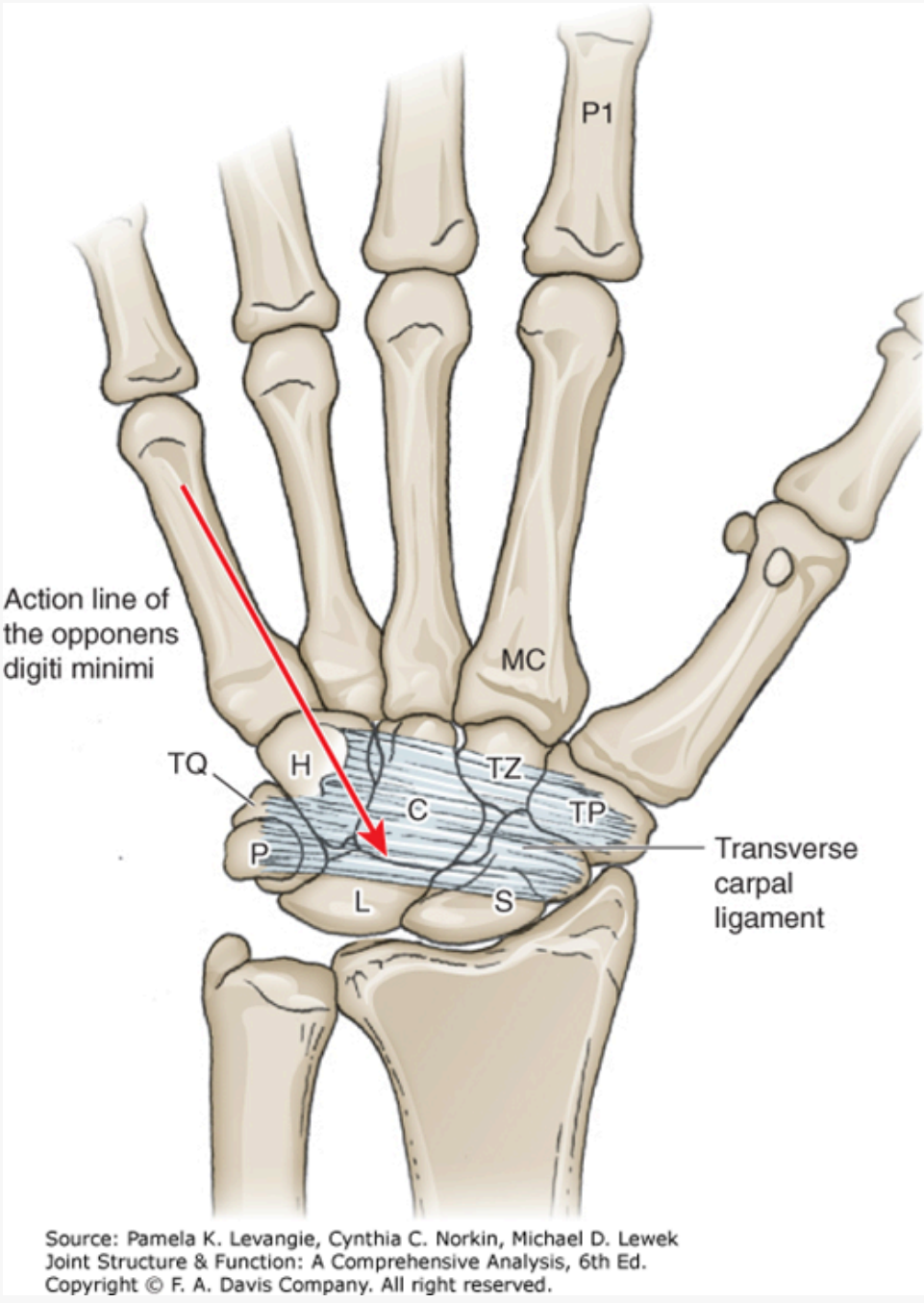
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Muscles that cross the carpometacarpal joints will contribute to palmar cupping (conformation of the palm to an object) by acting on the mobile segments of the palmar arches. Hollowing of the palm accompanies finger flexion, and relative flattening of the

palm accompanies finger extension. The fifth carpometacarpal joint is crossed and acted on by the **opponens digiti minimi (ODM)** muscle. This oblique muscle (Fig. 9–21) is optimally positioned to flex and rotate the fifth metacarpal about its long axis. No other muscles cross or act on the finger carpometacarpal joints alone. However, increased arching occurs with activity of the flexor carpi ulnaris muscle attached to the pisiform and with activity of the intrinsic hand muscles that insert on the transverse carpal ligament.[10,111] The radial wrist muscles (flexor carpi radialis, extensor carpi radialis longus, and extensor carpi radialis brevis) cross the second and third carpometacarpal joints to insert on the bases of those metacarpals but produce little or no motion at these relatively fixed articulations. The stability of the second and third carpometacarpal joints can be viewed as a functional adaptation that enhances the efficiency of the flexor carpi radialis, extensor carpi radialis longus, and extensor carpi radialis brevis muscles in their wrist functions. If the second and third carpometacarpal joints were mobile, the radial flexor and extensors would first move the carpometacarpal joints and, consequently, would be less effective at the midcarpal and radiocarpal joints, given the loss in length–tension.

FIGURE 9-21

The opponens digiti minimi is the only muscle that acts exclusively on a carpometacarpal joint. As indicated by its action line, it is effective at flexion of the fifth metacarpal joint and rotation of the metacarpal joint around its long axis. The opponens digiti minimi muscle's attachment to the transverse carpal ligament may also contribute to supporting the proximal palmar arch.



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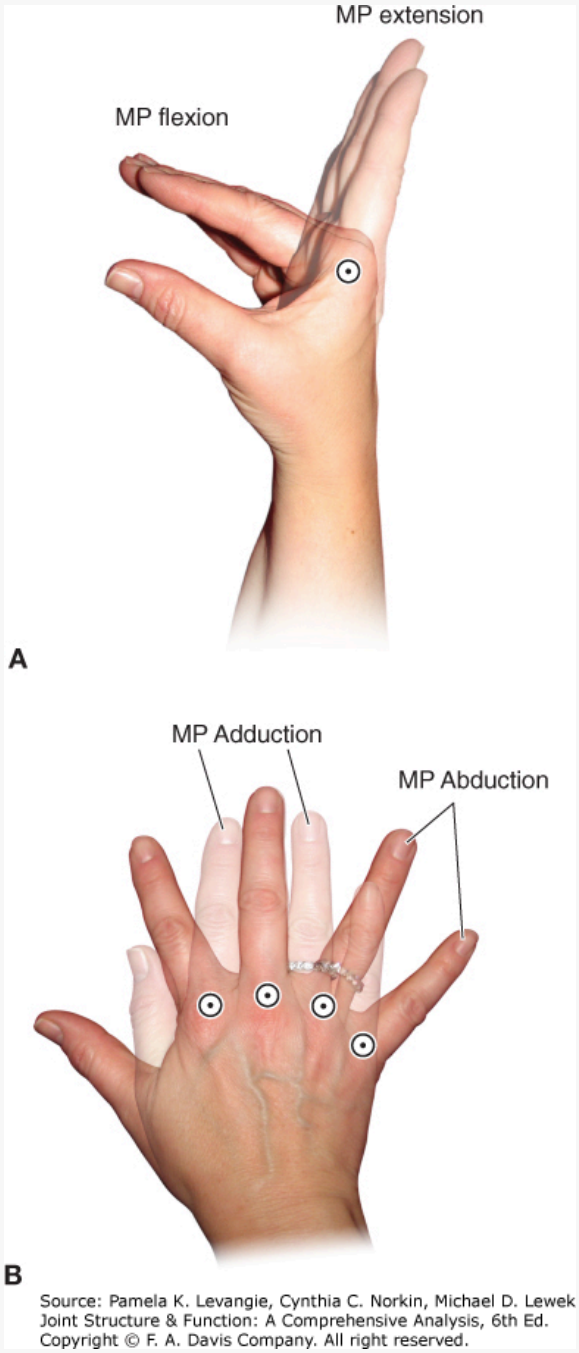
Metacarpophalangeal Joints of the Fingers

Each of the four metacarpophalangeal joints of the fingers is composed of the convex metacarpal head proximally and the concave base of the first phalanx distally. The metacarpophalangeal joint is condyloid with two degrees of freedom: flexion/extension

and abduction/adduction (**Fig. 9–22**). The large metacarpal head has 180° of articular surface in the sagittal plane, with the predominant portion lying volarly. This is apposed to approximately 20° of articular surface on the base of the phalanx (**Fig. 9–23**). In the frontal plane, there is less articular surface than in the sagittal plane, and the articular surfaces are more congruent (see Fig. 9–17).

FIGURE 9-22

Two degrees of freedom at the metacarpophalangeal joint: **A.** Flexion/extension around a coronal axis through the metacarpal heads. **B.** Abduction/adduction around sagittal axes.

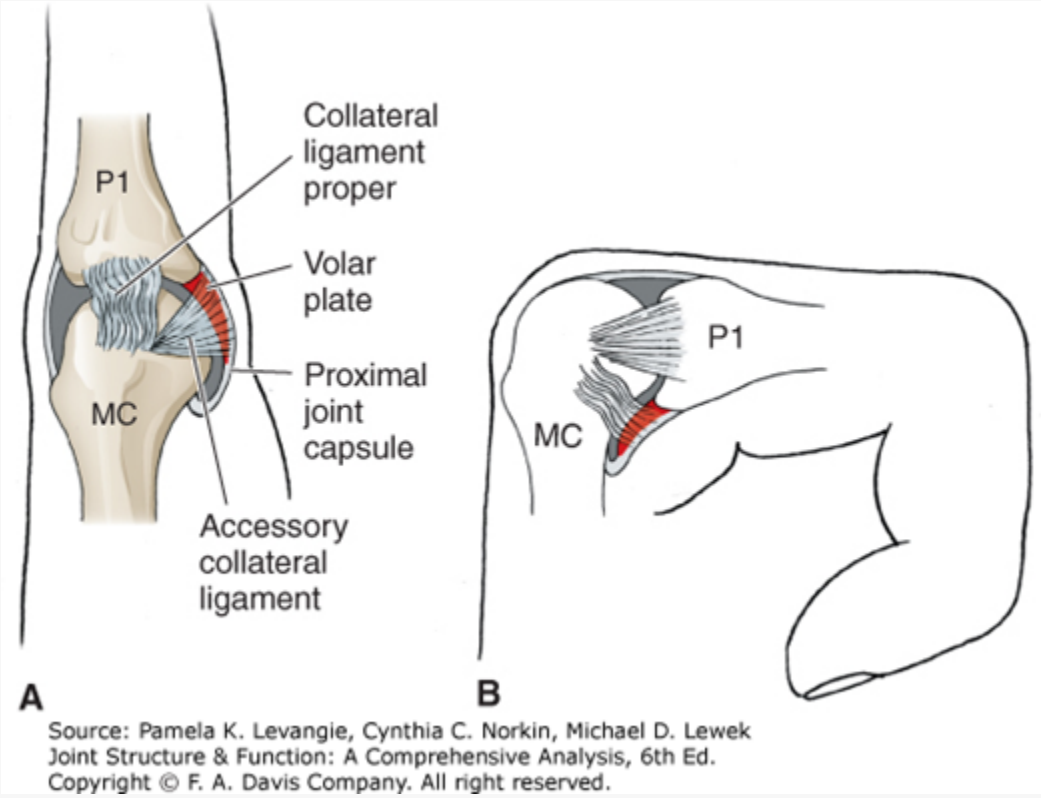


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FIGURE 9-23

A. The volar plate at the metacarpophalangeal joint attaches firmly to the base of the proximal phalanx, blending with the joint capsule to

attach loosely to the neck of the metacarpal below. **B.** In metacarpophalangeal joint flexion, the flexible attachments of the plate allow the plate to slide proximally on the metacarpal head without impeding motion.



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The metacarpophalangeal joint is surrounded by a capsule that is generally considered to be lax in extension. Given the incongruent articular surfaces, capsular laxity in extension allows some passive axial rotation of the proximal phalanx.[98] Two **collateral ligaments** and the volarly-located deep transverse metacarpal ligament enhance joint stability. As we noted previously, incongruent joints often have an accessory joint structure to enhance stability. At the metacarpophalangeal joint, this function is served by the **volar plate.**

Volar Plates

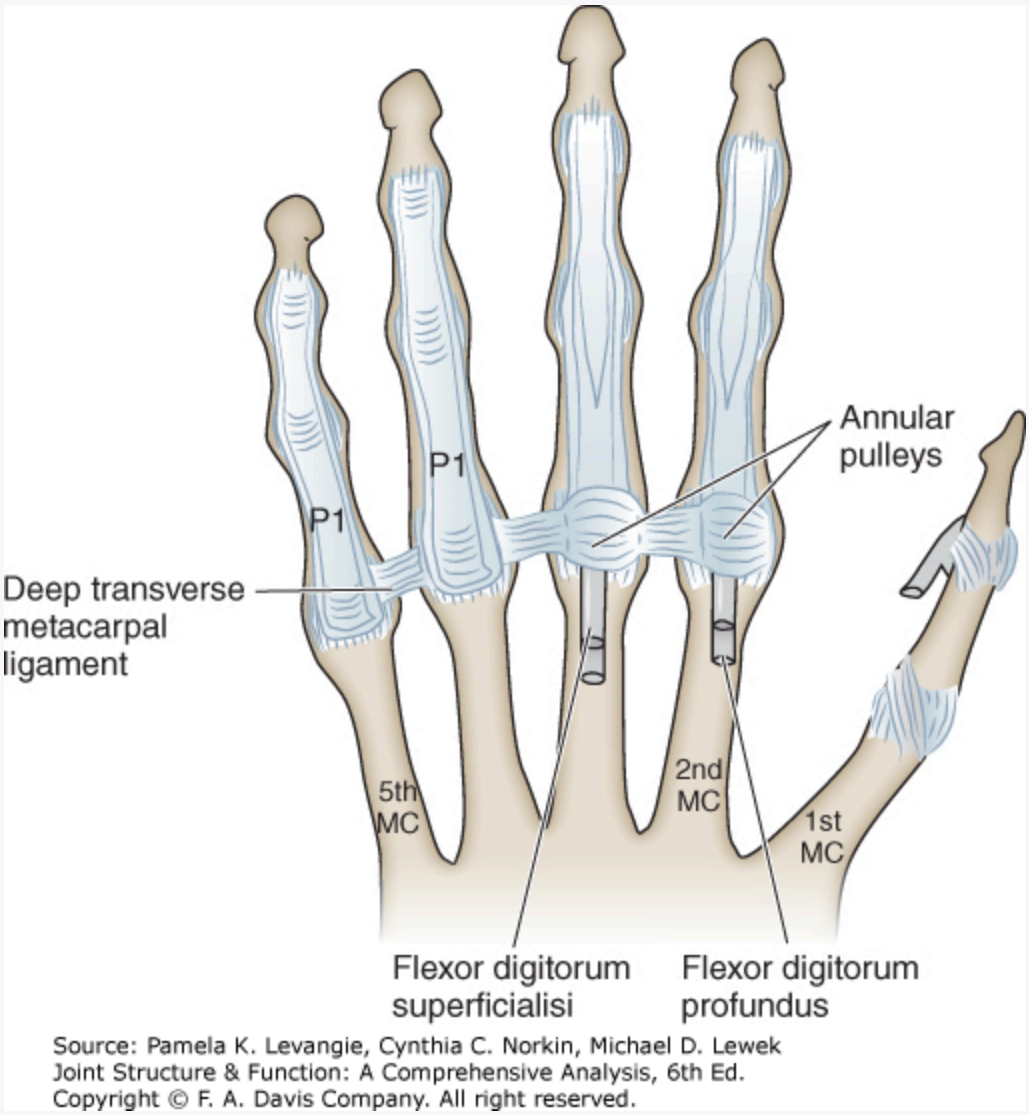
The volar plate (or **palmar plate**) at each of the metacarpophalangeal joints is a unique structure that increases joint congruence. It also provides stability to the metacarpophalangeal joint by limiting hyperextension and, therefore, provides indirect support to the longitudinal arch.[57] The volar plate is composed of fibrocartilage. The plate is firmly attached to the base of the proximal phalanx distally and becomes membranous proximally to blend with the volar capsule that attaches to the metacarpal head just proximal to the articular surface (Fig. 9–23A).[55] The volar plate can also be visualized as a fibrocartilage impregnation of the volar portion of the capsule just superficial to the metacarpal head. The inner surface of the volar plate is effectively a continuation of the articular surface of the base of the proximal phalanx. In metacarpophalangeal extension, the plate adds to the amount of surface in contact with the large metacarpal head. The fibrocartilage composition of the plate is consistent with its ability to resist both tensile stresses in restricting metacarpophalangeal hyperextension and compressive forces needed to protect the volar articular surface of the metacarpal head from objects held in the palm.[112] The flexible attachment of the plate to the phalanx permits the plate to glide proximally down the volar surface of the metacarpal head in flexion (Fig. 9–23B) without that restricting motion, while also preventing pinching of the long flexor tendons in the metacarpophalangeal joint.

In addition to their connection to their respective proximal phalanges, the four volar plates and their respective capsules of the metacarpophalangeal joints of the fingers also blend with and are interconnected superficially by the deep transverse metacarpal ligament that tethers together the heads of the metacarpals of the four fingers (Fig.

9–24). Dorsal to the deep transverse metacarpal ligament are **sagittal bands** on each side of the metacarpal head that connect each volar plate (via the capsule and deep transverse metacarpal ligament) to the extensor digitorum communis tendon and **extensor expansion** (Fig. 9–25). The sagittal bands help stabilize the volar plates over the four metacarpal heads.[21,98,109]

FIGURE 9-24

The deep transverse metacarpal ligament runs transversely across the four metacarpal heads. The volar aspect of the transverse metacarpal ligament is grooved to hold the long finger flexor tendons (shown on fourth and fifth metacarpophalangeal joints where the annular pulleys and tendons are removed).

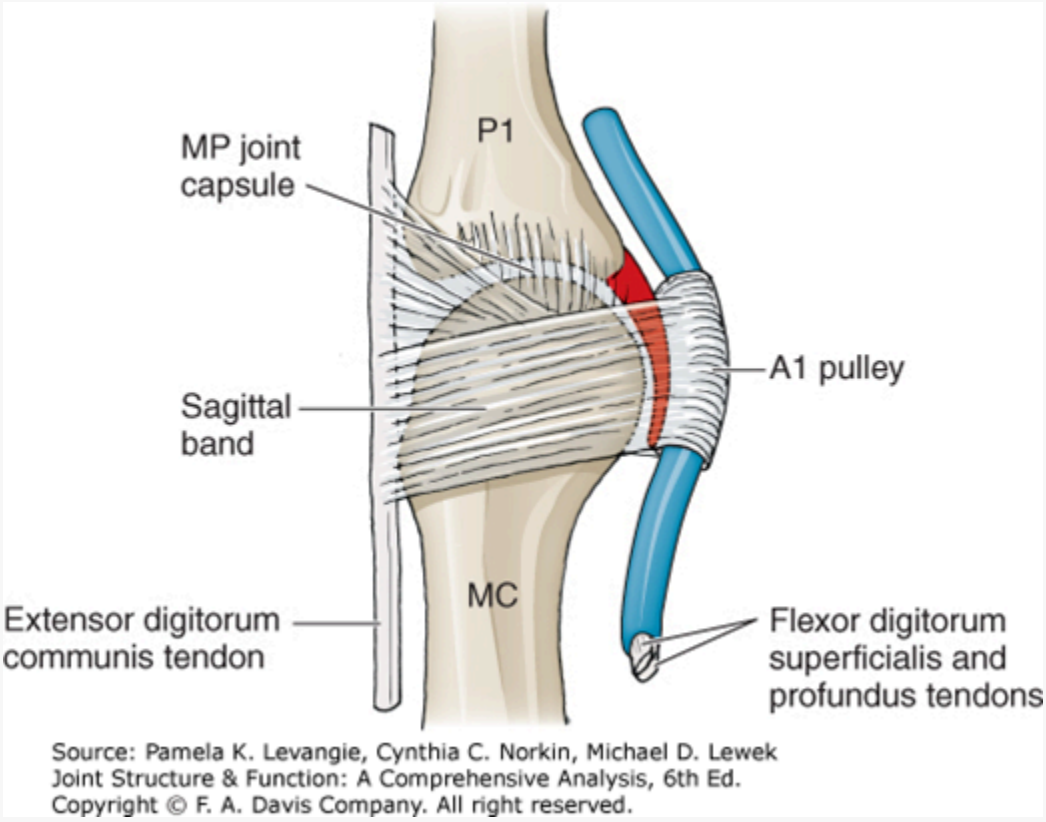


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FIGURE 9-25

The connections of the sagittal bands to each side of the volar plate, the collateral ligaments of the metacarpophalangeal joint (via the capsule), and the extensor digitorum communis muscle via the extensor expansion help stabilize the volar plates on the four metacarpal heads volarly and the extensor digitorum communis

tendons over the metacarpophalangeal joints dorsally.



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Collateral Ligaments

The radial and ulnar collateral ligaments of the metacarpophalangeal joint are composed of two parts: the **collateral ligament proper,** which is cordlike, and the **accessory collateral ligament** (see Fig. 9–23). Minami and associates quantified the length changes in the different parts of the collateral ligament at the metacarpophalangeal joint with varying degrees of motion.[113] They found that the more dorsally located collateral ligament proper was lengthened with metacarpophalangeal joint flexion from 0° to 80°, whereas the more volarly located accessory collateral ligament was shortened. Conversely, with metacarpophalangeal joint hyperextension, the accessory portion was lengthened and the proper portion was

placed on slack. Tension in the collateral ligaments at full metacarpophalangeal joint flexion (the close-packed position for the metacarpophalangeal joint) is considered to account for the minimal amount of abduction/adduction that can be obtained at the metacarpophalangeal joint in full flexion. Shultz and associates concluded that the collateral ligaments provided stability throughout the metacarpophalangeal joint ROM, with parts of the fibers taut at various points in the range.[114] Rather than attributing the limitation of abduction/adduction in metacarpophalangeal flexion to collateral ligamentous tension, they proposed that the bicondylar shape of the volar surface of the metacarpal head resulted in a bony block at about 70° of metacarpophalangeal joint flexion.

Fisher and associates completed a series of dissections of fingers, seeking an explanation for the relatively small incidence of osteoarthritis (OA) in metacarpophalangeal joints in comparison with the fairly common changes seen in the distal interphalangeal joints and, to a lesser extent, in the proximal interphalangeal joints.[115] They found fibrocartilage that projected into the metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints from the inner surface of the dorsally located **extensor hood,** from the volar plates, and from the collateral ligaments. The fibrocartilage projections were most impressive in the metacarpophalangeal joints and may, like the volar plate itself, increase the surface area on the small base of the phalange for contact with the large metacarpal (and phalangeal) heads.

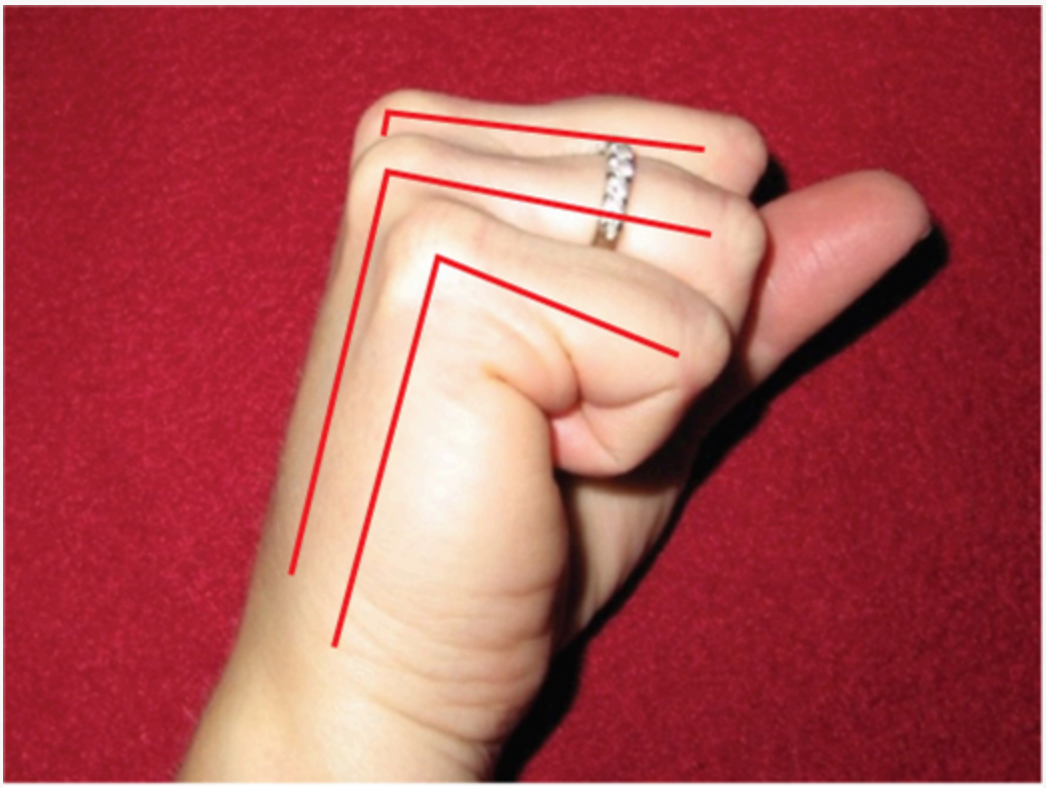
Range of Motion

The total ROM available at the metacarpophalangeal joint varies with each finger. Flexion/extension increases radially to ulnarly, with the index finger having

approximately 90° of metacarpophalangeal joint flexion and the little finger approximately 110° (Fig. 9–26).[57] Hyperextension is fairly consistent between fingers but varies widely among individuals. The range of passive hyperextension has been used as a measure of generalized body flexibility.[10] The range of metacarpophalangeal joint abduction/adduction is maximal in metacarpophalangeal joint extension. The index and little fingers have more frontal plane mobility than do the middle and ring fingers. As previously noted, metacarpophalangeal abduction/adduction is most restricted in full metacarpophalangeal joint flexion.[114] Some passive rotation of the metacarpophalangeal joints has been documented, which supports the contention that this mobility allows for adaptation of grasp for different sized objects.[116]

FIGURE 9-26

The available ROM at the metacarpophalangeal joints of the fingers increases from the radial side (second metacarpophalangeal joint not shown) to the ulnar side, with the greatest metacarpophalangeal finger range at the fifth metacarpophalangeal joint.



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Interphalangeal Joints of the Fingers

Each proximal interphalangeal and distal interphalangeal joint of the fingers is composed of the head of a phalanx and the base of the phalanx distal to it, forming a true synovial hinge joint with one degree of freedom (flexion/extension; Fig. 9–27). Each interphalangeal joint has a joint capsule, a volar plate, and two collateral ligaments (Fig. 9–28). The base of each middle and distal phalanx has two shallow concave facets with a central ridge. The distal phalanx sits on the pulley-shaped head of the phalanx proximal to it. The joint structure is similar to that of the metacarpophalangeal joint in that the proximal articular surface is larger than the distal articular surface. Unlike the metacarpophalangeal joints, there is little posterior articular surface at the proximal or

distal interphalangeal joint and, therefore, little hyperextension. The distal interphalangeal joint may have some passive hyperextension, but the proximal interphalangeal joint has essentially none in most individuals.

FIGURE 9-27

One degree of freedom (flexion/extension) at each of the proximal and distal interphalangeal joints around coronal axes.

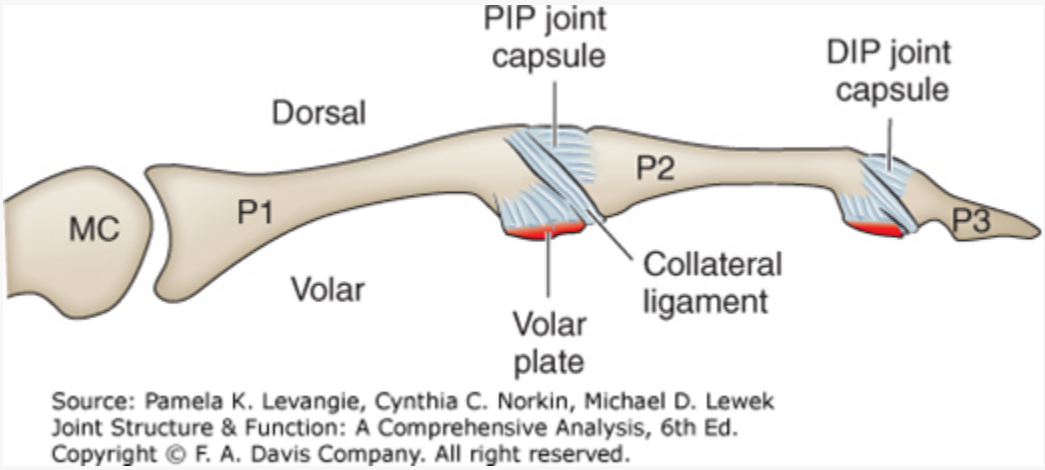


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FIGURE 9-28

The proximal interphalangeal and distal interphalangeal joints, like the metacarpophalangeal joints, have volar plates that blend with the

volar capsule portion of the capsule. The orientation of the collateral ligaments at the proximal and distal joints, however, differs from the orientation of the collateral ligaments at the metacarpophalangeal joints.



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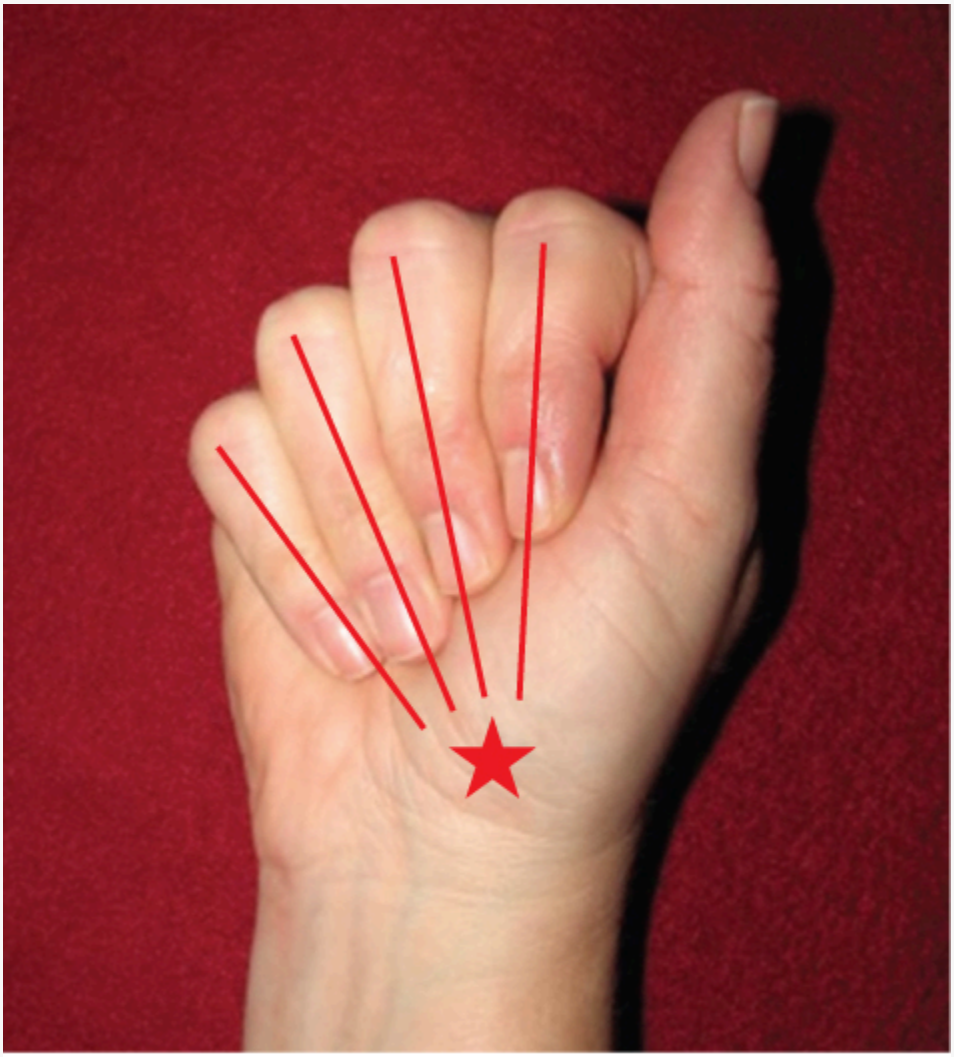
Volar plates also reinforce each of the interphalangeal joint capsules, enhance stability, and limit hyperextension.[117] The plates at the interphalangeal joints are structurally and functionally identical to those at the metacarpophalangeal joint, except that the plates are not connected by a deep transverse ligament. Fisher and associates found fibrocartilage projections from the **extensor mechanism,** the volar plate, and the collateral ligaments attached to the bases of the phalanges at both the proximal interphalangeal and the distal interphalangeal joints, with the structures more obvious at the proximal interphalangeal joints.[115] The collateral ligaments of the interphalangeal joints are not fully understood but are described as having cord and accessory parts similar to those of the metacarpophalangeal joint.[57] Stability is provided by this collateral ligament complex because some portions remain taut and provide support throughout proximal interphalangeal and distal interphalangeal joint

motion.[57,118,119] Injuries to the collateral ligaments of the proximal interphalangeal joint are common, particularly in sports and workplace injuries, with the radial (lateral) collateral twice as likely to be injured as the ulnar (medial) collateral.[120,121] Dzwierzynski and colleagues found the lateral collateral of the index finger to be the strongest of the proximal interphalangeal collateral ligaments, whereas the fifth proximal interphalangeal joint had the weakest collateral ligaments.[120] The relative strengths of the lateral collateral ligaments meet functional expectations because the thumb is most likely to oppose the lateral side of the index (creating a varus stress at the proximal interphalangeal joint), and least likely to do so at the fifth.

The total range of flexion/extension available to the index finger is greater at the proximal interphalangeal joint (100° to 110°) than it is at the distal interphalangeal joint (80°). The ranges for proximal interphalangeal and distal interphalangeal flexion at each finger increase ulnarly, with the fifth proximal interphalangeal and distal interphalangeal joints achieving 135° and 90°, respectively. The pattern of increasing flexion/extension ROM from the radial to the ulnar side of the hand is consistent at the carpometacarpal, metacarpophalangeal, and proximal interphalangeal joints and, less obviously, at the distal interphalangeal joints.[122] The additional range allocated to the more ulnarly located fingers as well as the underlying bony arrangement favors angulation of the fingers toward the scaphoid (termed **flexion convergence**) and facilitates opposition of the fingers with the thumb (**Fig. 9–29**). The greater available range ulnarly also produces a grip that is tighter, or has greater closure, on the ulnar side of the hand. Many objects are constructed so that the shape is narrower at the ring and small fingers and widens toward the middle and index fingers to fit this ROM pattern.

FIGURE 9-29

With flexion of digits to the palm, there is a convergence toward the scaphoid tubercle (star) and toward the thumb. This obliquity is due to the increasing flexion mobility of the metacarpophalangeal and proximal interphalangeal joints from the radial to the ulnar side of the hand.



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Expanded Concepts 9–3

Anti-Deformity Positioning

After trauma to the hand, a custom-fabricated orthosis (splint) is commonly provided to immobilize the injured structures. The purpose of this device is to provide support and protection to the injured region during the healing process, while attempting to minimize the potential problems at the joints created by immobilization (i.e., contractures). Because the collateral ligaments of the metacarpophalangeal joints are slack with extension, immobilization in metacarpophalangeal extension in an orthosis would place the collateral ligaments at risk for adaptive shortening. Adaptive shortening of the collateral ligaments would limit metacarpophalangeal joint flexion, with concomitant disruption of the longitudinal arch leading to impairments in grasp and functional use. Optimally, an immobilization (static) orthosis (Fig. 9–30) should place the metacarpophalangeal joints in some flexion so that the collateral ligaments are on stretch; the interphalangeal joints should be held in extension to reduce the risk of flexion contractures from adaptive shortening of the volar plates. The thumb should be placed in midrange of carpometacarpal palmar/radial abduction to prevent a first web space contracture. This position of metacarpophalangeal joint flexion with interphalangeal extension is known as the **anti-deformity position** or **intrinsic plus position.**[123]

FIGURE 9-30

Fabricating an immobilization orthosis with the hand in the “anti-deformity” position minimizes the risk of dysfunctional changes to the immobilized joints.



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Extrinsic Finger Flexors

The **extrinsic muscles** (also referred to as **motors)** of the fingers and thumb are those muscles that have attachments above (proximal to) the radiocarpal joint, whereas **intrinsic muscles** are those with all attachments distal to the radiocarpal joint. Functionally, the extrinsic muscles are also divided into flexors and extensors. The intrinsic muscles are typically not referred to as flexor or extensor groups because several muscles flex one joint while extending another joint simultaneously. We will first consider the extrinsic muscles of the fingers, then the intrinsic muscles of the fingers, and conclude with the extrinsic and intrinsic muscles of the thumb before discussing coordinated function of all the elements together.

There are two extrinsic muscles that contribute to finger flexion. These are the flexor digitorum superficialis (FDS) and the flexor digitorum profundus (FDP) muscles. The flexor digitorum superficialis muscle primarily flexes the proximal interphalangeal joint,

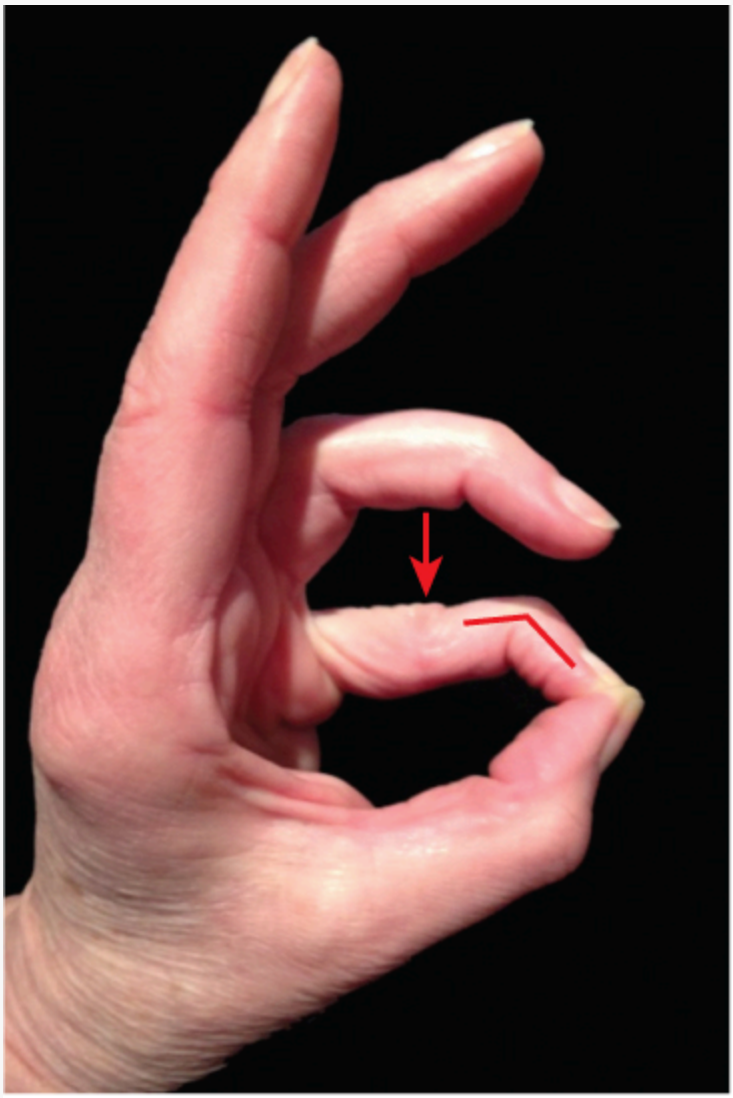
but it also contributes to metacarpophalangeal joint flexion. The flexor digitorum profundus muscle can flex the metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints and is considered to be the more active of the two extrinsic flexor muscles.[4] With gentle pinch or grasp, the flexor digitorum profundus muscle alone will be active. As greater flexor force is needed or when finger flexion with simultaneous wrist flexion is desired, the flexor digitorum superficialis muscle joins the flexor digitorum profundus muscle.[88,97,124,125]

The flexor digitorum superficialis muscle can produce more torque at the metacarpophalangeal joint than can the flexor digitorum profundus muscle. Not only does the flexor digitorum superficialis muscle cross fewer joints (making it less likely to lose tension as it shortens over multiple joints), but the flexor digitorum superficialis tendon is also superficial to the flexor digitorum profundus tendon at the metacarpophalangeal joint. Consequently, the flexor digitorum superficialis muscle has a greater moment arm for metacarpophalangeal joint flexion.[3] It is often thought that the flexor digitorum superficialis muscle is the stronger muscle during proximal interphalangeal flexion because the flexor digitorum superficialis muscle crosses fewer joints, but this is not the case. In contrast with the orientation of the flexor tendons at the metacarpophalangeal joint, the flexor digitorum superficialis tendon lies deep to the flexor digitorum profundus tendon at the proximal interphalangeal joint and, therefore, has a lesser moment arm at the proximal interphalangeal joint.[98] The switch in position between the flexor digitorum superficialis and flexor digitorum profundus tendons occurs just proximal to the proximal interphalangeal joint. At this point the flexor digitorum profundus tendon emerges through the split in the flexor digitorum superficialis tendon **(Camper's chiasma)** so that the flexor digitorum superficialis

tendon can attach to the base of the middle phalanx that is deep to the flexor digitorum profundus tendon. Although the moment arm of the flexor digitorum superficialis tendon may not be at its greatest at the proximal interphalangeal joint, the flexor digitorum superficialis tendon is important for balance at the proximal interphalangeal joint. When the flexor digitorum superficialis tendon is absent, forceful pinch (thumb to fingertip) activity of the flexor digitorum profundus muscle may create proximal interphalangeal *extension* along with distal interphalangeal flexion (Fig. 9–31), rather than flexion at both joints.[126] This phenomenon can be observed in many normal hands because the flexor digitorum superficialis tendon of the little finger is commonly absent or may have anomalous distal attachments.[126,127]

FIGURE 9-31

Forcefully pressing the pads of the thumb and little finger together activates the flexor digitorum profundus to flex both interphalangeal joints of the little finger against the flexed interphalangeal joint of the thumb. When the flexor digitorum superficialis is absent (as it often is in the little finger), a contraction of the profundus produces distal interphalangeal joint flexion with *proximal interphalangeal joint extension* (arrow). Without the stabilization of the proximal interphalangeal joint by the flexor digitorum superficialis muscle, the flexor digitorum profundus muscle is not able to flex both joints.



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Both the flexor digitomm superficialis and flexor digitorum profundus muscles are dependent on wrist position for an optimal length-tension relationship.[4] If there is no counterbalancing extensor torque at the wrist, the volarly located flexor digitorum superficialis and flexor digitoram profundus muscles will cause wrist flexion to occur. If the finger flexor muscles are permitted to shorten over the flexed wrist, there will be a concomitant loss of flexor tension at the more distal joints. In fact, it is almost

impossible to fully flex the fingers actively if the wrist is also flexed.[128] Although the poor length–tension relationship in the flexor digitorum superficialis and flexor digitorum profundus muscles accounts for some of this phenomenon, the inability to complete the flexion ROM with wrist flexion is also attributable to the concomitant passive tension in the stretched finger extensors. During active finger flexion (as in grasp activities), a counterbalancing wrist extensor force prevents wrist flexion and is usually supplied by an active wrist extensor such as the extensor carpi radialis brevis muscle or, in some instances, the extensor digitorum communis muscle.



Expanded Concepts 9–4

Finger Flexor Grasp

The greater available range of metacarpophalangeal and interphalangeal joint flexion in the ring and little fingers in comparison with the index or long fingers means that the long flexors of the ring and little fingers must shorten over a greater range, resulting in a loss of tension in the muscles of those fingers. If the object to be held by the fingers is heavy or requires a strong grip (e.g., hammer or electric drill), the object may be shaped so that it is wider ulnarly than radially, a so-called pistol grip (Fig. 9–32A). The pistol grip limits metacarpophalangeal/interphalangeal joint flexion in the ring and little fingers while the wrist extensors stabilize the wrist against a strong contraction of the finger flexors. The loss of tension in the long finger flexors is not a problem if strong grip is not required (e.g., holding a glass); then the object may be tapered at the ring and little fingers to accommodate the greater ROM (Fig. 9–32B). Notice that the wrist in both forceful and gentle grips tends to assume a position of ulnar deviation that maximizes efficiency of the long finger flexors.[2,83]

FIGURE 9-32

A. The so-called “pistol grip” of the hammer allows the flexor digitorum superficialis and flexor digitorum profundus muscles to

work more forcefully at the ring and little fingers because full flexion in these more mobile metacarpophalangeal and interphalangeal joints is prevented by the shape of the object (thicker toward the end of the handle). The shape also encourages wrist ulnar deviation that further enhances force production in the long finger flexors. **B.** When force is not needed, the shape of an object is often tapered to accommodate the greater range of the ring and little fingers, allowing the long finger flexors to close the fingers fully around the object. (KIA Video – Gripping Tasks Power, Gripping with Rheumatoid Arthritis)



A **B**

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Mechanisms of Finger Flexion

Optimal function of the flexor digitorum superficialis and flexor digitorum profundus muscles depends not only on stabilization by the wrist musculature but also on intact flexor gliding mechanisms.[129] The gliding mechanisms consist of the flexor retinacula, bursae, and digital tendon sheaths. The fibrous retinacular structures (proximal flexor retinaculum, transverse carpal ligament, and extensor retinaculum) tether the long flexor tendons to the hand; the bursae and tendon sheaths facilitate friction-free excursion of the tendons on the fibrous retinacula. The retinacula prevent bowstringing of the tendons that would result in loss of excursion and work efficiency in the contracting muscles that pass under them. The tendons must be anchored without interfering with their excursion and without creating frictional forces that would cause degeneration of the tendons over time.

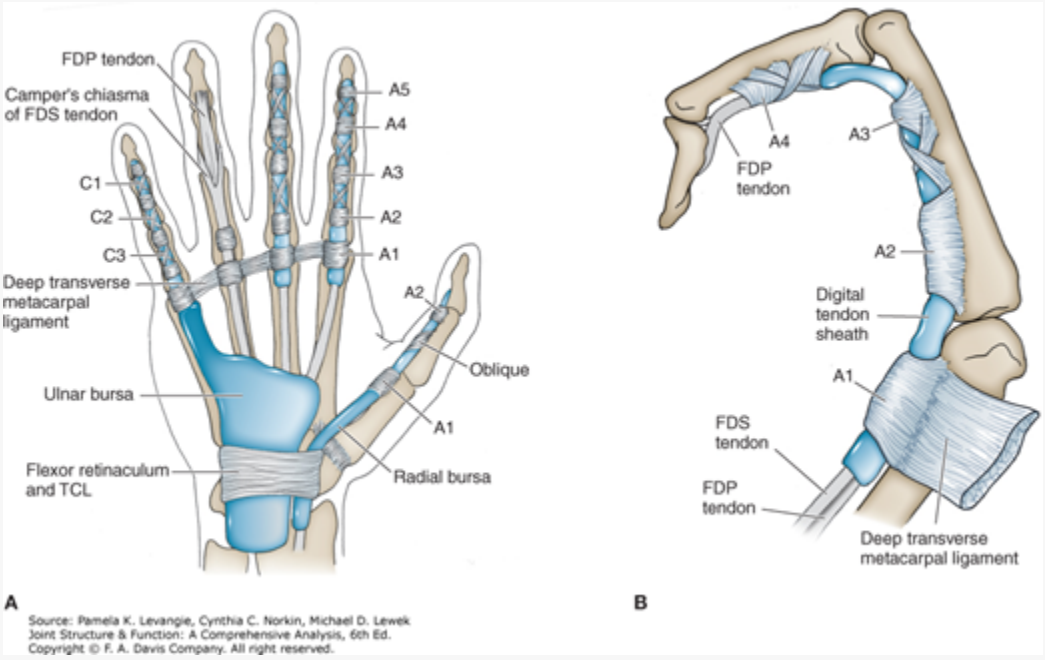
As the tendons of the flexor digitorum superficialis and flexor digitorum profundus muscles cross the wrist to enter the hand, they first pass beneath the proximal flexor retinaculum and through the carpal tunnel under the transverse carpal ligament (see Fig. 9–15). Friction between the tendons themselves and friction of the tendons on the overlying transverse carpal ligament are prevented by the radial and ulnar bursae that envelop the flexor tendons at this level (Fig. 9–33). All eight tendons of the flexor digitorum profundus and flexor digitorum supericialis muscles are invested together in the ulnar bursa (Fig. 9–33A). The bursa is compartmentalized to prevent friction of tendon on tendon. The flexor pollicis longus muscle that accompanies the flexor digitorum superficialis and flexor digitorum profundus muscles through the carpal tunnel is encased in its own radial bursa (see Figs. 9–15B and 9–33A). The radial and ulnar bursae contain a synovial-like fluid that minimizes frictional forces. The pattern of

bursae and tendon sheaths may vary among individuals. The most common representation shows the ulnar bursa to be continuous with the digital tendon sheath for the little finger (see Fig. 9–33A). However, Phillips and colleagues found continuity between the ulnar bursa and tendon sheath of the little finger in only 30% of 60 specimens.[130] The ulnar bursa is typically not continuous with the digital tendon sheaths for the index, middle, and ring fingers. Rather, for these fingers, the ulnar bursa ends just distal to the proximal palmar crease, and the digital tendon sheaths begin at the middle or distal palmar creases.[11] The radial bursa encases the flexor pollicis longus muscle and is continuous with its digital tendon sheath. The extent and communication of the digital tendon sheaths is functionally relevant because infection within a sheath will travel its full length, producing painful tenosynovitis. If a sheath is continuous with the ulnar or radial bursa, the infection may spread from the sheath into the palm (or vice versa).[32,87] The tendon sheaths for each finger terminate proximal to the insertion of the flexor digitorum profundus muscle, effectively ending at the distal aspect of the middle phalanx. Consequently, puncture wounds or injuries to the pad (distal phalanx) of the fingers that are a fairly common site of trauma are unlikely to introduce infection into the digital tendon sheaths.

FIGURE 9-33

A. The flexor mechanisms of the fingers and thumb include the fibro-osseous tunnels formed by the flexor retinaculum and transverse carpal ligament (TCL) at the wrist, the annular pulleys (A1 to A5), and the cruciate pulleys (C1 to C3). The tendons are protected within the tunnels by the radial and ulnar bursae and the digital tendon sheaths.

The pulleys and the tendon sheath have been removed from the ring finger to show how the deep flexor digitorum profundus (FDP) tendon emerges through Camper's chiasma in the flexor digitorum superficialis (FDS) tendon to pass on to the distal phalanx; after the split in the superficialis tendon at Camper's chiasma, the superficialis tendon rejoins and inserts on the base of the middle phalanx. **B.** The shape of the pulleys allows finger flexion without pinching of the pulleys, concomittantly more evenly distributing pressure on the tendon and sheath across the roof of the fibro-osseous tunnels.



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The flexor digitorum superficialis and flexor digitorum profundus tendons of each finger pass through a fibro-osseous tunnel that is composed of five transversely oriented **annular pulleys** (or **vaginal ligaments),** as well as three obliquely oriented **cruciate pulleys** (see Fig. 9–33A).[21,131] The first two annular pulleys lie closely together, with one

(designated the A1 pulley) at the head of the metacarpal and a second larger one (A2) along the volar midshaft of the proximal phalanx. The floor of the first (A1) pulley is formed by the flexor groove in the deep transverse metacarpal ligament, whereas all the other annular pulleys attach directly to bone. The third annular pulley (A3) lies at the distal most part of the proximal phalanx, and the fourth (A4) lies centrally on the middle phalanx. A fifth pulley (A5) may lie at the base of the distal phalanx. The base of each of the pulleys (on the bone) is longer than its more superficial roof. This trapezoidal shape prevents the pulleys from pinching each other at extremes of flexion, forming nearly one continuous tunnel in full grasp.[21] The shorter roof of the fibro-osseous tunnel also minimizes the pressure on the tendon when it is under tension, distributing pressure throughout the tunnel during finger flexion rather than just at the edges of the tunnel (see **Fig. 9–33B**).[131] The three cruciate (crisscrossing) pulleys also tether the long flexor tendons. One is located between the A2 and A3 pulleys and is designated as C1; the next cruciate pulley (C2) lies between the A3 and A4 pulleys; and the last cruciate pulley (C3) lies between the A4 and A5 pulleys. The A4, A5, and C3 structures contain only the flexor digitorum profundus tendon because the flexor digitorum superficialis muscle inserts on the middle phalanx proximal to these structures. The annular pulleys and cruciate ligaments vary among individuals in both number and extent.[131] More recently, an additional annular pulley found proximal to the A1 has also been described and has been named the **palmar aponeurosis (PA) pulley.**[132] The thumb has a distinct pulley system, including two annular and one oblique pulley (see Fig. 9–33A).[133]

Friction of the flexor digitorum superficialis and flexor digitorum profundus tendons on the annular pulleys and cruciate ligaments is minimized by the digital tendon sheaths

that envelop the tendons from the point at which the tendons pass into the most proximal annular pulley (PA or A1) to the point at which the tendon of the flexor digitorum profundus muscle passes through the most distal cruciate pulley (C3 or A5). The synovial-like fluid contained in each of the digital tendon sheaths permits gliding of the tendons beneath their ligamentous constraints and between each other. This is particularly important over the proximal phalanx, where the flexor digitorum superficialis tendon splits to either side of the flexor digitorum profundus tendon and rejoins *beneath* the flexor digitorum profundus tendon to insert on the middle phalanx. The flexor digitorum profundus tendon, consequently, must pass through Camper's chiasma (shown for the ring finger in Fig. 9–33A). Once the flexor digitorum profundus tendon is distal to the last annular pulley, the tendon sheath ends because lubrication of the tendon is no longer needed. Vascular supply to the gliding mechanism is critical to maintaining synovial fluid and tendon nutrition. Direct vascularization of each tendon occurs through vessels that reach the tendon via the **vincula tendinum.** The vincula are folds of the synovial membrane (usually four in number) that carry blood vessels to the body of the tendon and to the tendinous insertions of the flexor digitorum superficialis and flexor digitorum profundus muscles of each finger.[21,134] The tendons also receive some of their nutrition directly from the synovial fluid within the sheath and, through that mechanism, can withstand at least partial loss of direct vascularization.[134,135]

The function of the annular pulleys is to keep the flexor tendons close to the bone, allowing only a minimum amount of bowstringing and migration volarly away from the joint axes.[136,137] This sacrifices the increase in moment arm that might occur with substantial bowstringing of the tendons, but enhances tendon excursion and work

efficiency of the long flexors.[126,138] Any interruption in either the annular pulleys or the digital tendon sheaths can result in substantial impairment of flexor digitorum superficialis and flexor digitorum profundus muscle functioning or in structural deformity. **Trigger finger** is one example of the disability that can be created when repetitive trauma to a flexor tendon results in the formation of nodules on the tendon and thickening of an annular pulley. During active finger flexion, the nodule gets caught beneath the pulley, requiring passive extension to "unlock" the stuck flexed position.[10] Of the potential six annular pulleys (PA, A1 through A5), integrity of pulleys A2 and A4 is credited with being most critical to maintaining flexor digitorum superficialis/flexor digitorum profundus muscle efficiency, and they should be salvaged if at all possible during tendon reconstruction procedures.[133,138,139]



Foundational Concepts 9–3

Flexor Gliding Mechanism at the Metacarpophalangeal Joint

The flexor gliding mechanism at the metacarpophalangeal joint is particularly complex because of its multilayered structure. From *deep to superficial at each of the metacarpophalangeal joints of the fingers,* there are:

1. the fibrocartilaginous volar plate, which is in contact with the metacarpal head;
2. the longitudinal fibers of the metacarpophalangeal joint capsule that blends with the volar aspect of the plate;
3. the deep transverse metacarpal ligament (with fibers oriented perpendicularly to those of the longitudinal fibers of the capsule), which has grooves on its volar surface for the long flexor tendons of the fingers and forms the floor of a fibro-osseous tunnel;
4. the flexor digitorum profundus tendon, which lies in the groove of the transverse metacarpal ligament;

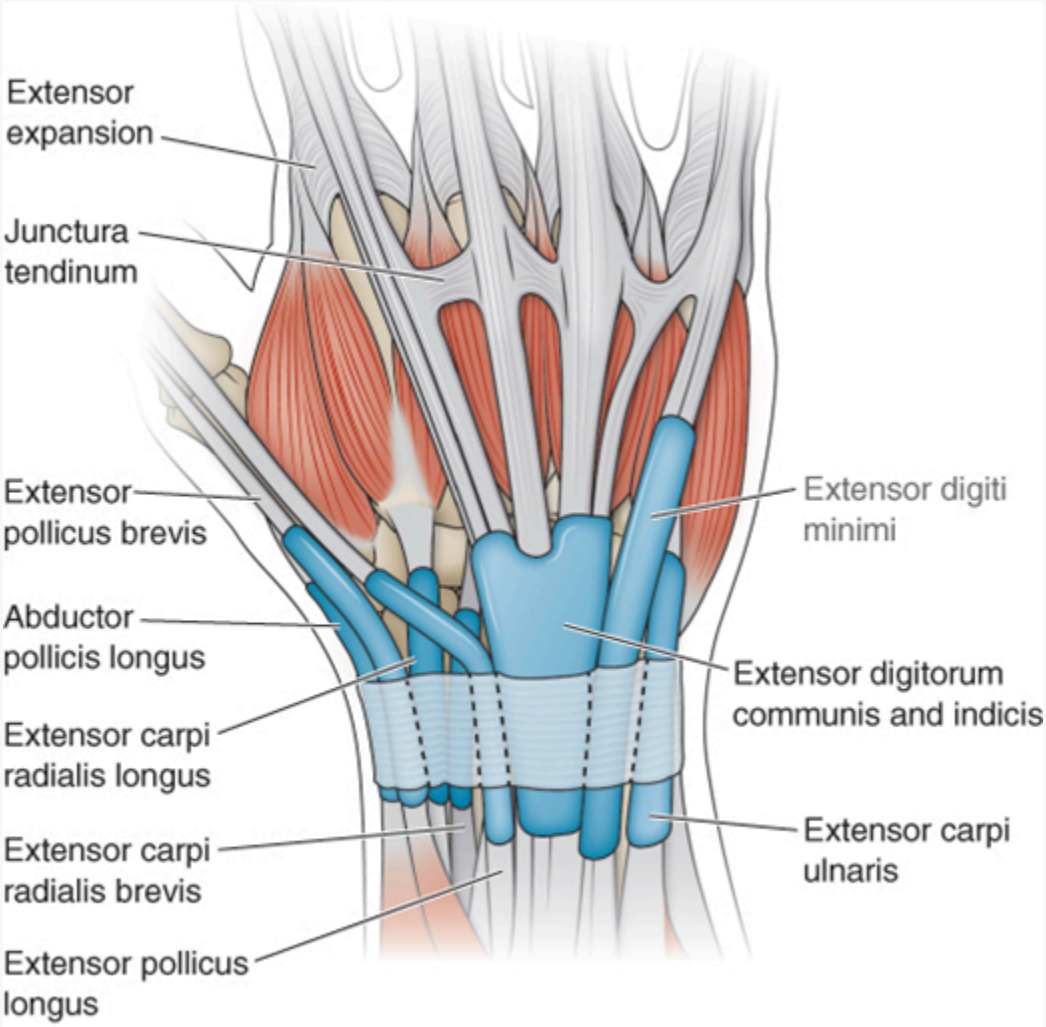
5. the flexor digitorum superficialis tendon that lies just superficial to the flexor digitorum profundus tendon;
6. the digital tendon sheath that envelops the flexor digitorum profundus and flexor digitorum superficialis tendons; and
7. the A1 annular pulley that forms the roof of the fibro-osseous tunnel and lies most superficially in this set of interconnected layers.

Extrinsic Finger Extensors

The extrinsic finger extensors are the extensor digitorum communis, the extensor indicis proprius, and the extensor digiti minimi muscles. Each of these muscles passes from the forearm to the hand beneath the extensor retinaculum at that wrist that maintains proximity of the tendons to the joints and improves excursion efficiency. Each of the six extensor tendons is contained within a compartment of the extensor retinaculum and is enveloped by an isolated bursa or tendon sheath that generally ends as soon as the tendons emerge distal to the extensor retinaculum (Fig. 9–34). At approximately the level of the metacarpophalangeal joint, the extensor digitorum communis tendon of each finger merges with a broad aponeurosis known interchangeably as the extensor expansion, the **dorsal hood,** or the **extensor hood.** The extensor indicis proprius and extensor digiti minimi tendons insert into the extensor digitorum communis tendons of the index and little fingers, respectively, at or just proximal to the extensor hood. Given the attachments of the extensor indicis proprius and extensor digiti minimi tendons to the extensor digitorum communis structure, the extensor indicis proprius and extensor digiti minimi muscles add force and independence of action to the index and ring finger extension rather than different actions.

FIGURE 9-34

Dorsal view of the hand, illustrating the six dorsal compartments of the extensor retinaculum at the wrist, the synovial sheaths, and the finger extensors (extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi muscles) that merge with the extensor expansion at the metacarpophalangeal joint. The juncturae tendinum of the extensor digitorum communis muscle lies just proximal to the metacarpophalangeal joints.



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The tendons of the extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi muscles show a good deal of variability on the dorsum of the hand. Most of the time, the index finger has one extensor digitorum communis tendon leading to the extensor hood and one extensor indicis proprius tendon inserting into the hood on the ulnar side of the extensor digitorum communis tendon.[140–142] At the little finger, the extensor digiti minimi tendon alone may merge with the extensor hood, with no extensor digitorum communis tendon to the little finger in as many as 30% of specimens.[143] The middle and ring fingers do not have their own auxiliary extensor muscles but frequently have two or even three extensor digitorum communis tendons leading to the hood.[142] The extensor digitorum communis tendons of one finger may also be connected to the tendon or tendons of an adjacent finger by **junctura tendinae** (see Fig. 9–34). These fibrous interconnections (frequently visible along with the extensor tendons on the dorsum of the hand) cause active extension of one finger to be accompanied by passive extension of the adjacent finger—with the patterns of interdependence varying with the connections.[89] In general, the extensor digitorum communis, extensor indicis proprius, extensor digiti minimi, and junctura tendinae connections result in the index finger having the most independent extension, with extension of the little, middle, and ring fingers in declining order of independence.[98]

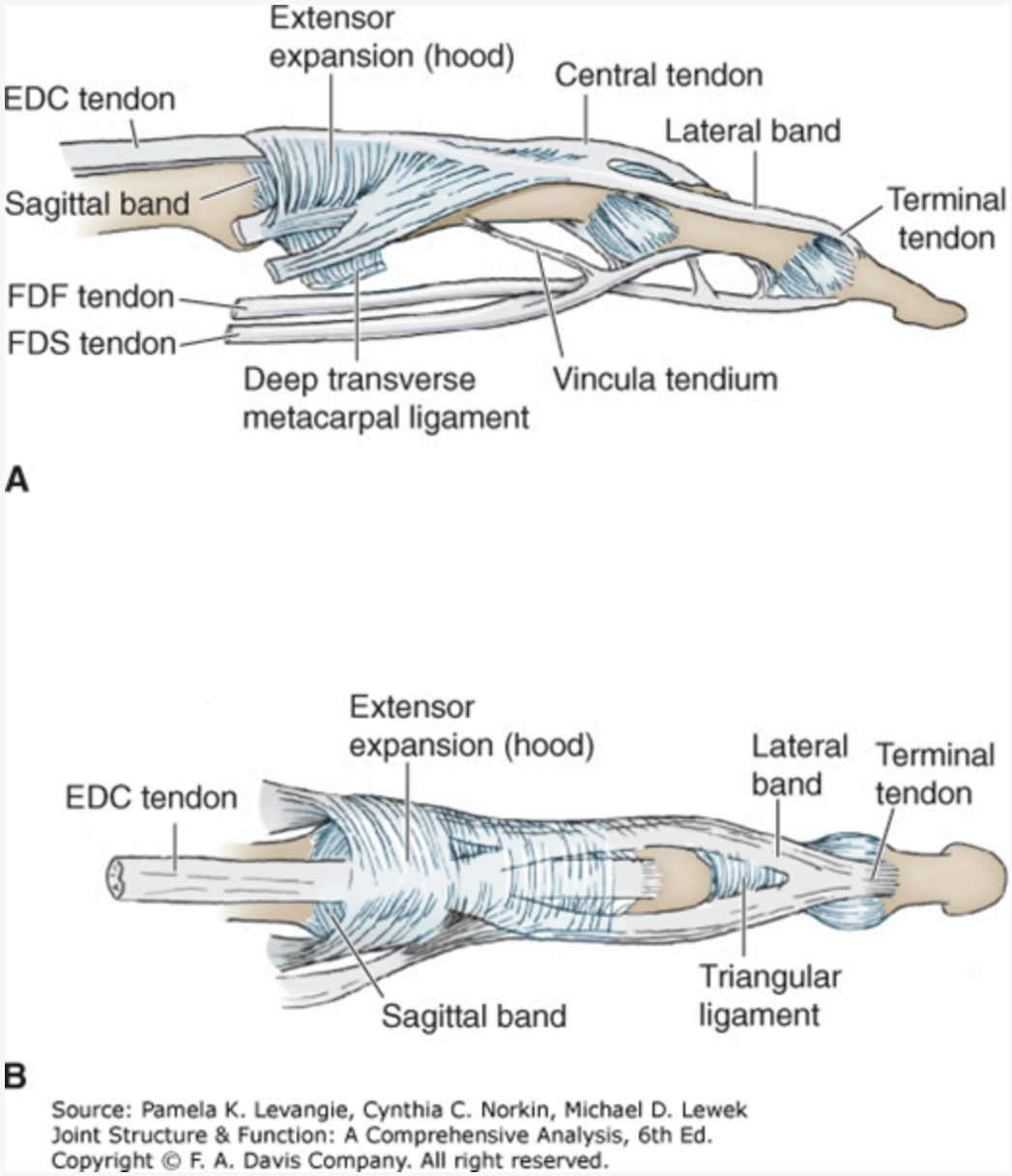
The extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi are the only muscles capable of extending the metacarpophalangeal joints of the fingers. These muscles extend the metacarpophalangeal joint via their connection to the extensor hood, as well as to the sagittal bands that interconnect the volar plates and the extensor digitorum communis tendon or extensor hood (as noted in the discussion of the volar plates).[89] Active tension on the extensor hood from one or more

of these muscles will extend the metacarpophalangeal joint even though there are no bony attachments to the proximal phalanx.[144] The extrinsic extensors are also wrist extensors by continued action. Because the extensor indicis proprius and extensor digiti minimi muscles share innervation, insertion, and function with the extensor digitorum communis muscle to which each attaches, discussion of the extensor digitorum communis muscle from this point on should be assumed to include contributions from the extensor indicis proprius or the extensor digiti minimi muscles. For the sake of brevity, all three muscles will not be named each time.

Distal to the extensor hood (and therefore after the extensor indicis proprius and extensor digiti minimi tendons have joined the extensor digitorum communis tendon), the extensor digitorum communis tendon at each finger splits into three bands: the **central tendon,** which inserts on the base of the middle phalanx, and two **lateral bands,** which rejoin to form the **terminal tendon** that inserts into the base of the distal phalanx (Fig. 9–35).[89] Although tension on the hood can produce metacarpophalangeal joint extension, the central and terminal tendons distal to the extensor expansion cannot be tightened sufficiently by activity in the extrinsic extensor muscles alone to produce full extension at either the proximal or distal interphalangeal joints. To produce full active interphalangeal extension, the extensor digitorum communis muscle requires the assistance of two intrinsic muscle groups that also have attachments to the extensor hood and the lateral bands. The extensor digitorum communis tendon and all its complicated active and passive interconnections at and distal to the metacarpophalangeal joint are known together as the **extensor mechanism.**

FIGURE 9-35

In the lateral **(A.)** and superior **(B.)** views, one can see the building blocks of the extensor mechanism. The elements include the extensor digitorum communis (EDC) tendon that merges with the extensor hood and sagittal bands and then continues distally to split into a central tendon inserting into the middle phalanx, and two lateral bands that merge into a terminal tendon that inserts into the distal phalanx. The two lateral bands are stabilized dorsally by the triangular ligament.



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Extensor Mechanism

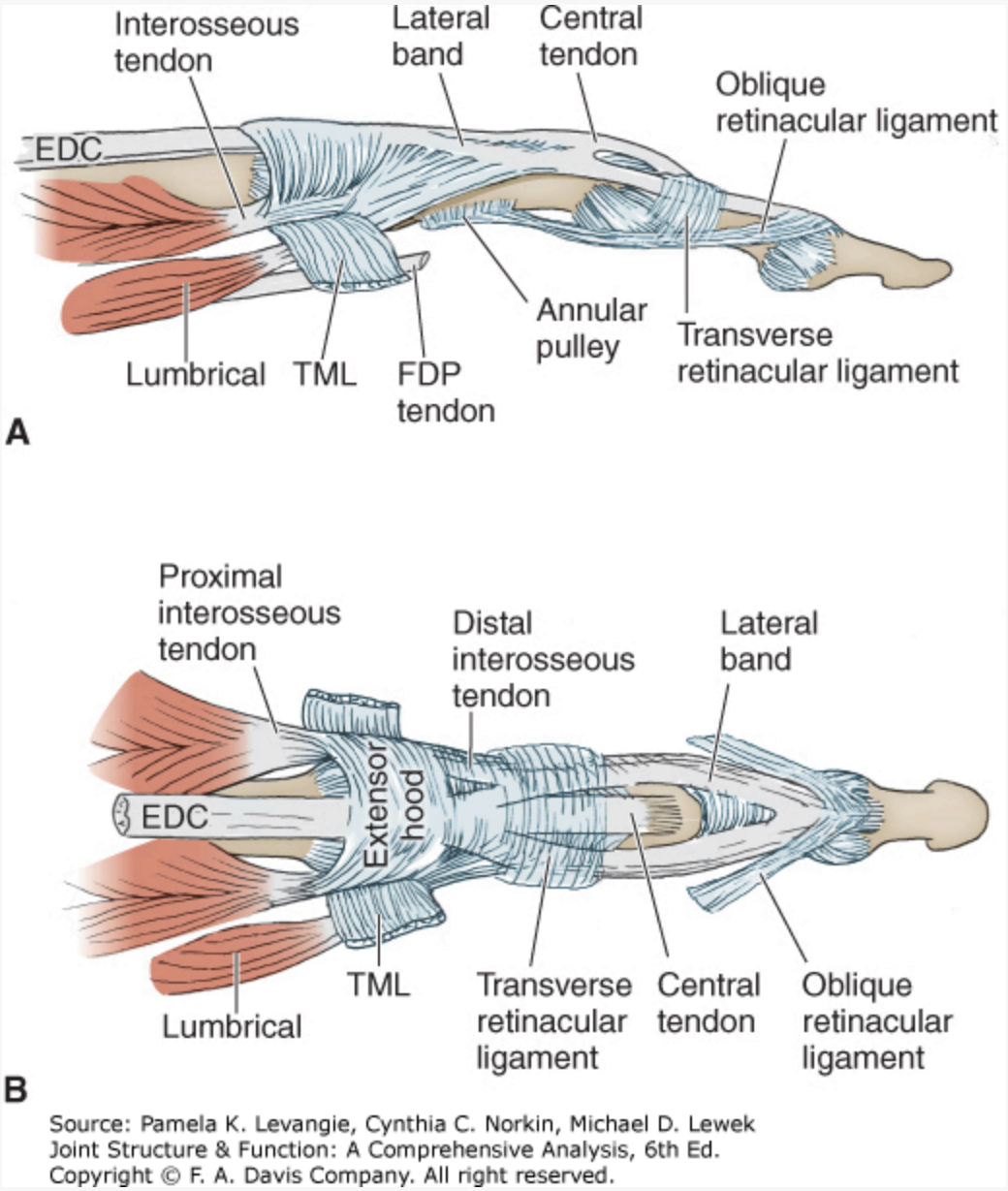
The foundation of the extensor mechanism is formed by the tendons of the extensor digitorum communis muscle (with extensor indicis proprius and extensor digiti minimi muscles), the extensor hood, the central tendon, and the lateral bands that merge into the terminal tendon. The first two components that we will add to the extensor mechanism are the passive components of the **triangular ligament** and the sagittal

bands (see Fig. 9–35). The lateral bands are interconnected dorsally by a triangular band of superficial fibers known as the triangular, or **dorsal retinacular,** ligament.[89] The triangular ligament helps stabilize the lateral bands on the dorsum of the finger. The sagittal bands connect the dorsal surface of the hood to the volar plates and deep transverse metacarpal ligament. The sagittal bands aid in stabilization not only of the volar plates but also of the hood at the metacarpophalangeal joint. The sagittal bands help to prevent bowstringing of the extensor mechanism during active metacarpophalangeal joint extension, as well as transmit forces that will help extend the proximal phalanx.[89] The sagittal bands are also responsible for centralizing the extensor digitorum communis tendon over the metacarpophalangeal joint dorsally, preventing tendon subluxation.[145]

The **dorsal interossei (DI), volar interossei (VI),** and **lumbrical** muscles are the active components of the extensor mechanism (Fig. 9–36). The dorsal interossei and volar interossei muscles arise proximally from the sides of the metacarpal joints. Distally, some muscle fibers go deep to insert directly into the proximal phalanx, whereas others join with and become part of the hood that wraps around the proximal phalanx. The interossei muscles may also contribute fibers to the central tendon and both lateral bands. The lumbrical muscles attach proximally to the flexor digitorum profundus tendons and distally to the lateral band. The lumbrical and interossei muscles together are often referred to as the **intrinsic muscles** of the fingers. Once we add the **oblique retinacular ligaments (ORLs),** the structure of the extensor mechanism for each finger will be complete.

FIGURE 9-36

In both the lateral **(A.)** and superior **(B.)** views, it can be seen that the interossei muscles pass dorsal to the transverse metacarpal ligament (TML) and may attach directly to the extensor hood or may have fibers that attach more distally to the central tendon and lateral bands. The lumbrical muscles attach to the flexor digitorum profundus (FDP) tendon volarly (in A.) and to the lateral bands. The oblique retinacular ligament (in A.) is attached to the annular pulley proximally and to the lateral bands distally, lying just deep to the transverse retinacular ligament. EDC, extensor digitorum communis.



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Final passive elements that contribute to the extensor mechanism are the oblique retinacular ligaments, which arise from both sides of the proximal phalanx and the sides of the annular and cruciate pulleys volarly. The slender oblique retinacular ligaments continue distally to insert into the lateral bands just distal to the proximal interphalangeal joint (see Fig. 9–36A).[146,147] The oblique retinacular ligaments lie volar

to the axis of the proximal interphalangeal joint and dorsal to the axis of the distal interphalangeal joint via its attachment to the lateral bands. Function of the extensor mechanism can now be presented by looking in more detail at the active and passive elements that compose it and by referencing the relation of relevant segments to each joint individually.

Extensor Mechanism Influence on Metacarpophalangeal Joint Function

The extensor digitorum communis tendon passes dorsal to the metacarpophalangeal joint axis. An active contraction of the muscle creates tension on the sagittal bands of the extensor mechanism, pulls the bands proximally over the metacarpophalangeal joint, and extends the proximal phalanx. An isolated contraction of the extensor digitorum communis muscle will result in metacarpophalangeal joint hyperextension with interphalangeal flexion.[148–150] The accompanying interphalangeal flexion is not produced by an active flexor but by passive tension in the flexor digitorum superficialis and flexor digitorum profundus muscles that are stretched over the extended metacarpophalangeal joint. This position of the fingers (metacarpophalangeal joint hyperextension with passive interphalangeal flexion) is known as **clawing**. Similar to what we saw at the proximal carpal row of the wrist complex, clawing is the classic zigzag pattern that occurs when a compressive force is exerted across several linked segments, one of which is an unstable "intercalated" segment. In the instance of clawing of the finger, the proximal phalanx hyperextends on the metacarpal below, while the middle and distal phalanx flex over it.

To simultaneously extend the proximal interphalangeal and distal interphalangeal joints, the extensor digitorum communis muscle requires active assistance. The other

active forces that are part of the extensor mechanism are the dorsal interossei, volar interossei, and lumbrical muscles. Each of these muscles passes volar to the metacarpophalangeal joint axis and, through their connections on the extensor expansion, dorsal to the interphalangeal joints. When the extensor digitorum communis, interossei, and lumbrical muscles all contract simultaneously, the metacarpophalangeal joint will extend along with the interphalangeal joints because the extensor torque produced by the extensor digitorum communis muscle at the metacarpophalangeal joint exceeds the metacarpophalangeal joint flexor torque of the intrinsic muscles. **Collapse** (*excessive* extension) of the proximal phalanx is prevented by active tension in the lumbrical or interossei muscles that pass volar to the metacarpophalangeal joint axis.[151,152] When the intrinsic muscles are weak or paralyzed (as in a low ulnar nerve injury), the extensor digitorum communis muscle is unopposed, and the fingers claw not only with active metacarpophalangeal joint extension but also at rest (**Fig. 9–37**). The clawing at rest demonstrates that the passive tension in the intact extensor digitorum communis muscle exceeds the passive tension in the remaining metacarpophalangeal joint flexors. The clawed position is also known as an **intrinsic minus position** because it is attributed to the absence of the finger intrinsic muscles.

FIGURE 9-37

In the ulnar nerve-deficient hand (claw hand) at rest, the metacarpophalangeal joints of the ring and little finger are hyperextended because of relatively unopposed passive tension in the intact extensor digitorum communis muscle resulting from the loss of

the interossei and lumbrical muscles; the interphalangeal joints of those fingers are flexed because of increased passive tension in the long flexors stretched over the extended metacarpophalangeal joints. The index and middle fingers are less affected because these fingers still have intact median nerve-innervated lumbrical and flexor digitorum profundus muscles.



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Extensor Mechanism Influence on Interphalangeal Joint Function

The proximal interphalangeal and distal interphalangeal joints are joined by active and passive forces in such a way that the distal interphalangeal extension and proximal interphalangeal extension are interdependent. When the proximal interphalangeal joint is actively extended, the distal interphalangeal joint will also extend. Similarly, active

distal interphalangeal extension will create proximal interphalangeal extension. The interdependence can be understood by examining structural relationships in the extensor mechanism.

Each proximal interphalangeal joint is crossed dorsally by the central tendon and lateral bands of the extensor mechanism (see Fig. 9–35). The extensor digitorum communis, interossei, and lumbrical muscles all have attachments to the hood, central tendon, or lateral bands at or proximal to the proximal interphalangeal joint (see Fig. 9–36). Consequently, the extensor digitorum communis, interossei, and lumbrical muscles are each capable of producing at least some tension in each of the following: the central tendon, the lateral bands, and (via the lateral bands) the terminal tendon, resulting in each contributing to some extensor force at the proximal interphalangeal and distal interphalangeal joints. An extensor digitorum communis muscle contraction alone will not produce effective interphalangeal extension. An active contraction of a dorsal interossei, volar interossei, or lumbrical muscle alone *is* capable of extending the proximal and distal interphalangeal joints completely because of their more direct attachments to the central tendon and lateral bands. However, if one or more of the intrinsic muscles (dorsal interossei, volar interossei, or lumbrical) contracts without a simultaneous contraction of the extensor digitorum communis muscle of that finger, the metacarpophalangeal joint will flex because each intrinsic muscles passes *volar* to the metacarpophalangeal joint axis. Although it may appear that the intrinsic muscles are independently extending the interphalangeal joints, passive tension in the extensor mechanism (stretched over the flexed metacarpophalangeal joint) may be assisting the active intrinsic muscles with interphalangeal extension.

Stack proposed that the interossei and lumbrical muscles would not be able to generate sufficient tension to cause independent interphalangeal extension if the extensor digitorum communis tendon was completely slack or severed.[153] Therefore, it appears that *two* sources of tension in the extensor expansion may be necessary to fully extend the interphalangeal joints. Source 1 is normally an active contraction of one or more of the intrinsic finger muscles. Source 2 may be either an *active contraction* of the extensor digitorum communis muscle (with active metacarpophalangeal joint extension) or *passive stretch* of the extensor digitorum communis muscle created by metacarpophalangeal joint flexion resulting from an active contraction of the intrinsic muscles.



Expanded Concepts 9–5

Interphalangeal Joint Extension in the Absence of Intrinsic Muscles

When the finger intrinsic musculature is paralyzed, as in an ulnar nerve injury, the ulnar nerve innervated interossei and lumbrical muscles of the ring and little fingers cannot provide the active assistance that the extensor digitorum communis muscle needs to fully extend the interphalangeal joints. The extensor digitorum communis muscle may be able to extend the interphalangeal joints independently, but *only* if the metacarpophalangeal joint is maintained in at least some flexion by some external force. If some passive tension in the extensor digitorum communis muscle can be maintained by stretch over the flexed metacarpophalangeal joint (source 1), additional tension to the extensor expansion can then be provided by an active contraction of the extensor digitorum communis muscle (source 2). These two sources (simultaneous active and passive tension in the extensor digitorum communis muscle) may be sufficient to produce full or nearly full proximal interphalangeal and distal interphalangeal joint extension in the absence of intact intrinsic muscles.[149] An external orthosis or surgical fixation of the metacarpophalangeal joints in a semiflexed position (Fig. 9–38A) is necessary to maintain some metacarpophalangeal joint flexion to stretch the extensor digitorum communis muscle while providing resistance to further metacarpophalangeal joint

extension that the active extensor digitorum communis muscle would otherwise cause.[154] This can also be thought of as providing the means for the extensor digitorum communis muscle to strongly contract without the concomitant loss of tension that would happen if permitted to complete the metacarpophalangeal joint extension ROM. The arrangement of the orthosis shown here restricts metacarpophalangeal joint extension when the hand is actively opened, without restricting the ability of the intact flexor digitorum superficialis muscle (and the intact flexor digitorum profundus muscle, depending on the level of ulnar nerve injury) to actively flex the proximal joints of the ring and little fingers (Fig. 9–38B).

FIGURE 9-38

A. In the ulnar nerve-deficient hand, the extensor digitorum communis muscle can indepdendently extend the interphalangeal joints of the ring and little fingers in the absence of the interossei and lumbrical muscles if (and only if) full metacarpophalangeal joint extension is prevented by an orthosis to maintain some passive stretch on the extensor tendon during an active extensor digitorum communis muscle contraction. **B.** The orthosis is shaped so that the intact long finger flexor or flexors can still actively flex the fingers with minimal interference by the orthosis. EDC, extensor digitorum communis.



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Some of the linkage between proximal interphalangeal and distal interphalangeal joint extension may be attributed to passive tension in the oblique retinacular ligaments. The oblique retinacular ligaments pass just volar to the proximal interphalangeal joint axis and attach distally to the lateral bands (see Fig. 9–36A). When the proximal and distal interphalangeal joints are both flexed, the oblique retinacular ligament is slack at the proximal interphalangeal joint while under some tension from the stretch of the lateral bands (to which they attach) and terminal tendon at the distal interphalangeal joint. If the proximal interphalangeal joint begins to extend (whether actively or passively), the oblique retinacular ligament will get increasingly tensed proximally and will,

consequently, pull on the taut lateral bands, causing the distal interphalangeal joint to begin to extend as well. The lengths of the oblique retinacular ligaments are such, however, that the contribution of proximal interphalangeal extension to producing distal interphalangeal extension via the oblique retinacular ligaments may be significant only during the first half of the distal interphalangeal joint's return from flexion (90° to 45° flexion) when the lateral bands and attached oblique retinacular ligaments are most stretched.[146,152] Overall, the complex structure of the extensor expansion and its contributing active and passive elements result in a relative linking between proximal interphalangeal and distal interphalangeal extension.[10,11,148,150]

Just as proximal and distal interphalangeal joint extension are typically coupled, flexion of the proximal and distal interphalangeal joints are similarly linked by a complex combination of active and passive forces. When distal interphalangeal joint flexion is initiated by a contraction of the flexor digitorum profundus muscle, there is a simultaneous flexor torque applied at the proximal interphalangeal joint that the tendon also crosses, so simultaneous distal and proximal interphalangeal joint flexion is not surprising. However, the active force of the flexor digitorum profundus muscle on the distal phalanx might not be sufficient to produce simultaneous proximal interphalangeal flexion if extensor restraining forces at the proximal interphalangeal joint were not released at the same time.

When distal interphalangeal flexion is initiated by the flexor digitorum profundus muscle, the terminal tendon and its lateral bands are stretched over the dorsal aspect of the distal interphalangeal joint. The stretch in the lateral bands pulls the extensor hood (from which the lateral bands arise) distally. The distal migration in the extensor

hood causes the central tendon of the extensor expansion to relax, thus releasing its extensor influence at the proximal interphalangeal joint and, thereby, facilitating proximal interphalangeal flexion. The simultaneous flexor torque and release of extensor torque still might not be adequate for the flexor digitorum profundus muscle to simultaneously flex the proximal interphalangeal joint if the lateral bands remained taut on the dorsal aspect of the proximal interphalangeal joint. The bands, however, are permitted to separate somewhat by the elasticity of the interconnecting triangular ligament and by passive tension in the **transverse retinacular ligament** (see Fig. 9–36). Although the example used here was initiated by an active contraction of the flexor digitorum profundus muscle, the same set of mechanisms will tie passive distal interphalangeal flexion (produced by an external flexor force) to passive proximal interphalangeal flexion.



Expanded Concepts 9–6

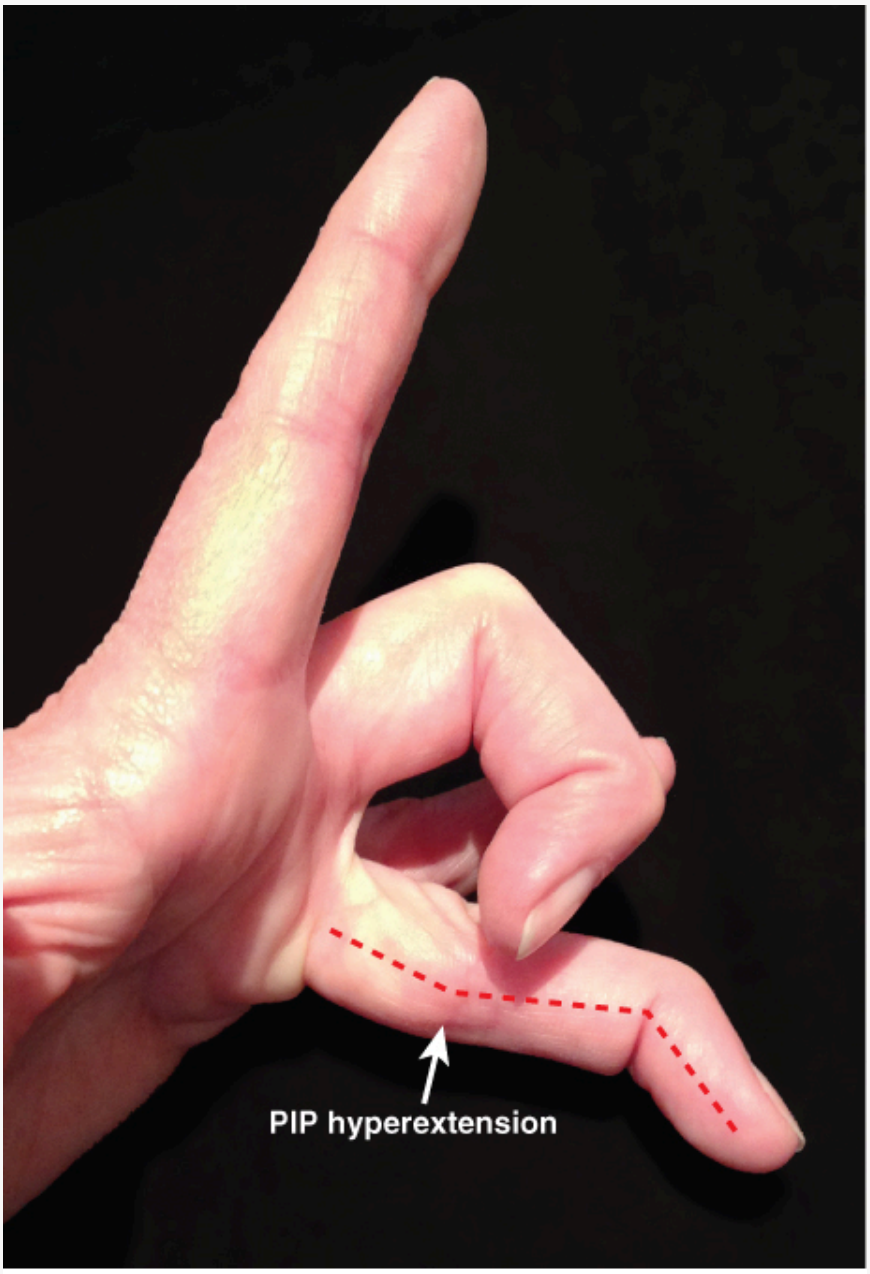
Finger Tricks: Distal Interphalangeal Joint Flexion with Proximal Interphalangeal Joint Extension

The normal coupling of proximal interphalangeal joint flexion with distal interphalangeal joint flexion can be overridden by some individuals; that is, some people can actively flex a distal interphalangeal joint (using the flexor digitorum profundus muscle) while maintaining the proximal interphalangeal joint in extension (Fig. 9–39). This “trick” is due to the influence of the oblique retinacular ligaments and requires some proximal hyperextension of the finger. When the proximal interphalangeal joint can be sufficiently hyperextended, the oblique retinacular ligaments that ordinarily lie just volar to the proximal interphalangeal joint axis will migrate dorsal to the proximal interphalangeal joint axis. At that point, tension in the oblique retinacular ligaments produced by active distal interphalangeal joint flexion (stretch of the terminal tendon and lateral bands to which the oblique retinacular ligaments attach) will accentuate proximal interphalangeal joint extension because the oblique retinacular ligaments function as passive proximal interphalangeal joint

extensors. The trick of active distal interphalangeal joint flexion and proximal interphalangeal joint extension serves no functional purpose and can be accomplished only in fingers in which proximal interphalangeal joint hyperextension is available. The “trick” does, however, highlight the necessity of releasing extensor tension at the proximal interphalangeal joint before the flexor digitorum profundus muscle can effectively flex that joint.

FIGURE 9-39

Some individuals can actively flex the distal interphalangeal joint in the presence of proximal interphalangeal (PIP) joint extension. This generally requires the individual to have at least some proximal interphalangeal joint hyperextension that causes the oblique retinacular ligaments to migrate dorsal to the proximal interphalangeal joint axis, making it a joint extensor rather than flexor when under tension.



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The functional coupling of proximal/distal interphalangeal joint action can be demonstrated by one other proximal/distal interphalangeal joint relation.[57] When the proximal interphalangeal joint is fully flexed actively by the flexor digitorum superficialis muscle or passively by an external force, the distal interphalangeal joint

cannot be actively extended. When the proximal interphalangeal joint is flexing, the dorsally located central tendon is becoming stretched. The increasing tension in the central tendon pulls the extensor hood (from which the central tendon arises) distally. This distal migration of the hood releases some of the tension in the lateral bands. The tension in the lateral bands is further released as the lateral bands separate slightly at the flexing proximal interphalangeal joint. Releasing tension in the lateral bands releases tension in the terminal tendon on the distal phalanx. As 90° of proximal interphalangeal flexion is reached, loss of tension in the terminal tendon is sufficient to eliminate any extensor force at the distal interphalangeal joint, including any potential contribution from the oblique retinacular ligaments because these ligaments have also been released by proximal interphalangeal flexion.[155] Although the distal interphalangeal joint can be actively flexed by the flexor digitorum profundus muscle when the proximal phalanx is already fully flexed, the distal phalanx cannot be actively re-extended as long as the proximal interphalangeal joint remains flexed.



Foundational Concepts 9–4

Summary of Coupled Actions of the Proximal and Distal Interphalangeal Joints

- Active extension of the proximal interphalangeal joint will normally be accompanied by extension of the distal interphalangeal joint.
- Active or passive flexion of the distal interphalangeal joint will normally initiate flexion of the proximal interphalangeal joint.
- Full flexion of the proximal interphalangeal joint (actively or passively) will prevent the distal interphalangeal joint from being actively extended.

Intrinsic Finger Musculature

Dorsal and Volar Interossei Muscles

The dorsal interossei and volar interossei muscles, as already noted, arise from between the metacarpals and are an important part of the extensor mechanism. There are four dorsal interossei muscles (one to each finger) and three to four volar interossei muscles. Many (but not all) anatomy texts describe the thumb as having a first volar interossei muscle. Mardel and Underwood suggested that the discrepancy may be in whether the controversial muscle is considered a separate volar interossei muscle or as part of the **flexor pollicis brevis (FPB)**.[156] Because the dorsal interossei and volar interossei muscles are alike in location and in some of their actions, these two muscle groups are often characterized by their ability to produce metacarpophalangeal joint abduction and adduction, respectively. Additional detail on the variable attachments of these muscles, as well as studies revealing multiple muscle heads, has increased our understanding of their contribution to hand function.[157] We will now look at how the attachments of the interossei muscles affect their role as metacarpophalangeal joint flexors or stabilizers, and as interphalangeal extensors.

The interossei muscle fibers join the extensor expansions in two locations. Some fibers attach *proximally* to the proximal phalanx and to the extensor hood; some fibers attach more *distally* to the lateral bands and central tendon of the extensor mechanism (see Fig. 9–36). Although individual variations in muscle attachments exist, studies have found some consistency in the point of attachment of the different interossei muscles.[147,153,158] The first dorsal interossei muscle has the most consistent attachment of its group, inserting entirely into the bony base of the proximal phalanx and the extensor hood. The dorsal interossei muscles of the middle and ring fingers (with the

middle finger having a dorsal interossei muscle on each side) each have both proximal and distal attachments (to the proximal phalanx/hood and to the lateral bands/central tendon). The little finger does not have a dorsal interossei muscle. The **abductor digiti minimi (ADM)** muscle is, in effect, a dorsal interossei muscle and typically has only a proximal attachment (proximal phalanx/hood).[98] The three volar interossei muscles of the fingers consistently appear to have distal attachments only (attachments to the lateral bands/central tendon). Conceptually, then, we can establish a frame of reference in which we can summarize the dorsal interossei and volar interossei attachments as follows:

- The first dorsal interossei muscle (index finger) has only a proximal attachment;
- The second, third, and fourth dorsal interossei muscles (second and third fingers) have both proximal and distal attachments
- The **fifth dorsal interossei** muscle (the abductor digiti minimi on the fourth finger) has only a proximal attachment
- The three volar interossei muscles of the first, third and fourth fingers have only distal attachments Note that the second finger does not have a volar interosseous muscle because it has a dorsal interosseous muscle on both sides.

Given the particular proximal or distal attachment patterns of the interossei and abductor digiti minimi muscles, these muscles can be characterized not only as abductors or adductors of their respective metacarpophalangeal joints but also as proximal or distal interossei according to the pattern of attachment. Proximal interossei

will have their predominant effect at the metacarpophalangeal joint alone, whereas the distal interossei will produce their predominant action at the interphalangeal joints, with a lesser effect at the metacarpophalangeal joint by continued action.

All of the dorsal interossei and volar interossei muscles (regardless of their designation as proximal or distal) pass *dorsal* to the transverse metacarpal ligament but just volar to the axis for metacarpophalangeal joint flexion/extension. All the interossei muscles, therefore, are potential flexors of the metacarpophalangeal joint. The ability of the interossei muscles to contribute to flexion at the metacarpophalangeal joint, however, will vary somewhat with metacarpophalangeal joint position.

ROLE OF THE INTEROSSEI MUSCLES AT THE MCP JOINT IN MCP JOINT EXTENSION

When the metacarpophalangeal joint is in extension, the moment arm (and rotary component) of all the interossei muscles for metacarpophalangeal joint flexion is so small that little flexion torque is produced by the interossei given that the action lines of the interossei muscles pass almost directly through the coronal (flexion/extension) axis. In spite of their poor flexor torque production with the metacarpophalangeal joint extended, the interossei muscles can be effective stabilizers (joint compressors) and appear to be important in helping to prevent clawing (metacarpophalangeal joint hyperextension) of the fingers by the intact extensors.[98,148]

There is typically no EMG activity recorded in the interossei muscles when the hand is at rest, when there is isolated extensor digitorum communis muscle activity, or when there is combined extensor digitorum communis/flexor digitorum profundus muscle activity. However, when these activities are performed in a hand that has long-standing ulnar nerve paralysis (therefore, no interossei muscles), an exaggerated

metacarpophalangeal joint extension or hyperextension (clawing) results. In a low ulnar nerve injury, the index and middle fingers retain the flexor digitorum superficialis and flexor digitorum profundus muscles as well as a lumbrical muscle each; therefore, the loss of the interossei muscles in the index and ring fingers is reflected by a resting metacarpophalangeal joint posture of neutral flexion/extension, rather than slight flexion, as is usually observed. The ring and little fingers will assume a metacarpophalangeal joint hyperextended and interphalangeal flexed posture at rest (clawing), even in the presence of intact flexor digitorum superficialis and flexor digitorum profundus muscles, because the ulnar nerve innervated interossei, abductor digiti minimi, and lumbricals are all missing from the ring and little fingers (see Fig. 9–37).[10,11,86,148] Clawing at rest is not evident in the ulnar nerve-deficient hand immediately after injury, but increases as the passive viscoelastic tension in the denervated interossei muscles is lost through atrophy and stretch and through stretching of the volar plates. In the intact resting hand, the interossei muscles that are typically inactive at rest still appear to play a role in balancing the passive tension of the metacarpophalangeal extensors by providing *passive* viscoelastic flexor tension at the metacarpophalangeal joints.



Expanded Concepts 9–7

Wartenberg's Sign

In addition to the clawing evident in the ring and little fingers in the ulnar nerve-deficient hand (see Fig. 9–37), the little finger may also assume an abducted metacarpophalangeal joint position with loss of the intrinsic muscles. Abduction of the little finger (Wartenberg's sign) may be the result of the unbalanced pull of the extensor digiti minimi muscle among those individuals having a direct connection of the extensor digiti minimi muscle to the abductor tubercle of the proximal phalanx—the only one of the extensor tendons that has an insertion directly onto the

proximal phalanx in a substantial number of people.[140]

When the metacarpophalangeal joint is extended, the interossei (and abductor digiti minimi) muscles lie at a relatively large distance from the A-P axis for metacarpophalangeal joint abduction/adduction. Consequently, in the metacarpophalangeal joint extended position, the interossei (and abductor digiti minimi) muscles are effective abductors or adductors of the metacarpophalangeal joint without the loss of tension that would occur if the muscles were simultaneously producing metacarpophalangeal joint flexion. The interossei muscles that insert proximally (on the proximal phalanx/hood) are more effective as metacarpophalangeal joint abductors/adductors, whereas the interossei muscles with more distal insertions (lateral bands/central tendon) are less effective at metacarpophalangeal abduction/adduction because they produce those motions by continued action. In our conceptual framework, the dorsal interossei muscles (the metacarpophalangeal joint abductors) all have proximal insertions, and the volar interossei muscles (the metacarpophalangeal joint adductors) have only distal insertions. Therefore, it makes sense that metacarpophalangeal joint abduction is stronger than metacarpophalangeal joint adduction. As importantly, the dorsal interossei muscles have twice the muscle mass of the volar interossei muscles. In a progressive ulnar nerve paralysis, the relatively ineffective metacarpophalangeal joint adduction component of the smaller volar interossei muscles is the first to show weakness.

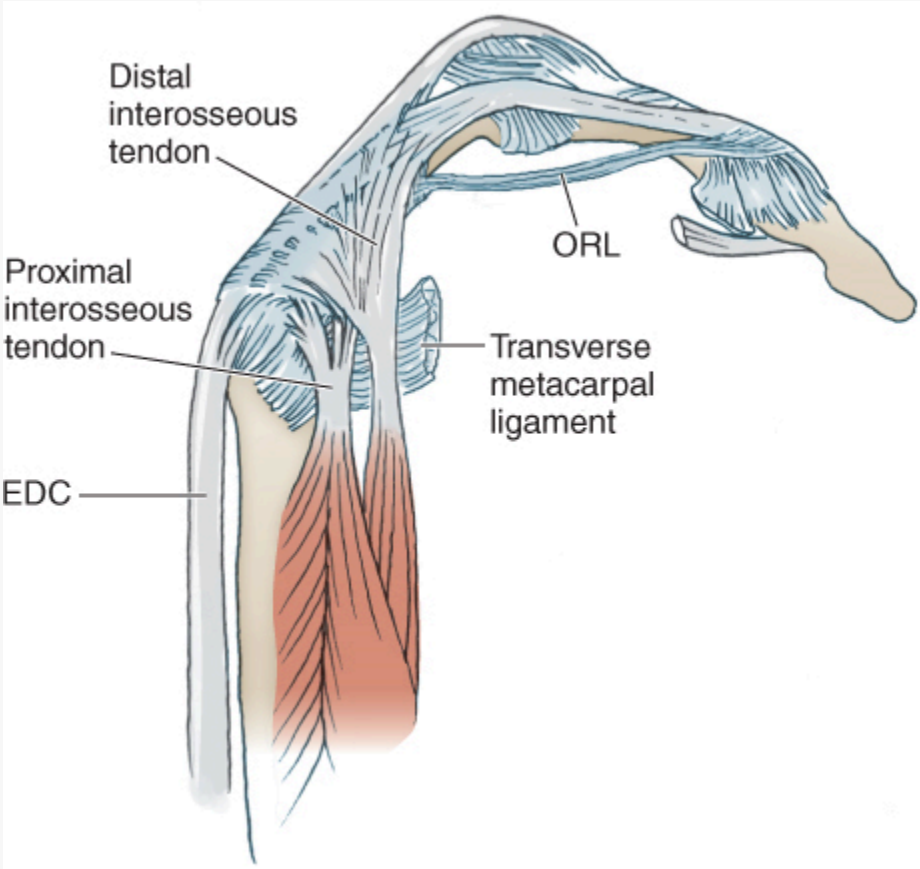
ROLE OF THE INTEROSSEI MUSCLES AT THE MCP JOINT IN MCP JOINT FLEXION

As the metacarpophalangeal joint flexes from the extended position, the tendons and action lines of the interossei muscles migrate volarly away from the

metacarpophalangeal joint's coronal flexion/extension axis, increasing the moment arm of the interossei for metacarpophalangeal flexion. In fact, in full metacarpophalangeal joint flexion, the action lines of the interossei muscles are nearly perpendicular to the moving proximal phalanx (Fig. 9–40). Consequently, the ability of the interossei muscles to create a metacarpophalangeal flexion torque increases as the metacarpophalangeal joint moves toward full flexion. As the metacarpophalangeal joint approaches full flexion, the deep transverse metacarpal ligament limits any further volar migration of the interossei tendons. Although the deep transverse metacarpal ligament limits further increases in the moment arm of the interossei muscles, the ligament also prevents the loss of active tension that would occur with bowstringing of the interossei tendons and simultaneously serves as an anatomical pulley. With increased metacarpophalangeal joint flexion, the collateral ligaments of the metacarpophalangeal joint also become increasingly taut, limiting side-to-side (abduction/adduction) movement of the proximal phalanx. The increasing tension in the collateral ligaments helps prevent the loss of metacarpophalangeal joint flexor force that would occur if the interossei muscles produced concommitant metacarpophalangeal joint abduction/adduction. In full metacarpophalangeal joint flexion, metacarpophalangeal joint abduction and adduction are completely restricted by tight collateral ligaments, by the shape of the condyles of the metacarpal head, and by active insufficiency of the fully shortened interossei muscles. The net effect of these combined mechanisms is that the ability of the interossei muscles to produce a metacarpophalangeal joint flexion torque (in the metacarpophalangeal flexed position) makes them powerful metacarpophalangeal joint flexor muscles that contribute to grip when a strong pinch or grip is required.[4,125,159]

FIGURE 9-40

When the metacarpophalangeal joint is flexed, the interossei muscles migrate volarly away from the metacarpophalangeal joint axis for flexion/extension, resulting in a relatively large moment arm and a line of pull that is nearly perpendicular to the proximal phalanx. The volar migration of the interossei muscles is limited by the deep transverse metacarpal ligament which prevents loss of tension and serves as an anatomical pulley for the interossei. EDC, extensor digitorum communis; ORL, oblique retinacular ligament.



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ROLE OF THE INTEROSSEI MUSCLES AT THE IP JOINT IN IP JOINT EXTENSION

The ability of the interossei muscles to produce interphalangeal joint extension is influenced by their attachments. To create sufficient tension in the extensor mechanism to contribute effectively to interphalangeal extension, the muscles must attach to the central tendon or lateral bands. All the interossei muscles have distal attachments except the hand's two "outside" abductors on the first and fourth fingers (first dorsal interossei and abductor digiti minimi muscles).

When the metacarpophalangeal joint is extended, the action lines of the distal interossei are ineffective in producing metacarpophalangeal joint flexion because of the poor moment arm, but are capable of extending the interphalangeal joints because the distal interossei attach directly to the central tendon and lateral bands. The interphalangeal extension produced by the distal interossei is stronger than their metacarpophalangeal joint abduction/adduction action because the abduction/adduction is produced by continued action. We have already noted that when the metacarpophalangeal joint flexes, the tendons of the interossei muscles migrate volarly at the metacarpophalangeal joint but are restricted in their volar excursion by the deep transverse metacarpal ligament. The deep transverse metacarpal ligament prevents the interosseous tendons from becoming slack through volar migration and has a pulley effect on the distal tendons. The anatomical pulley effect of the deep transverse metacarpal ligament may enhance the function of the distal interossei muscles as interphalangeal joint extensors because interphalangeal extension appears to be stronger in metacarpophalangeal joint flexion than in metacarpophalangeal joint extension.

The index and little fingers each have only one interosseous muscle with a distal insertion (second and fourth volar interossei muscles, respectively). The middle and ring fingers each have two distal tendons (second and third dorsal interossei muscles for the middle finger, and fourth volar interossei and fourth dorsal interossei muscles for the ring finger). The index and little fingers, therefore, are weaker in interphalangeal extension than are the middle and ring fingers because they have fewer distal interossei attachments.[158]

In approaching or holding the position of combined metacarpophalangeal flexion and interphalangeal extension the proximal components are effective metacarpophalangeal joint flexors, and the distal components are effective as both metacarpophalangeal joint flexors and interphalangeal extensors. The combination of active metacarpophalangeal flexion and interphalangeal extension produces the most consistent activity of the interossei muscles, a position that takes advantage of optimal biomechanics for both the proximal and the distal interossei muscles.[148,160]

Lumbrical Muscles

The lumbrical muscles are the only muscles in the body that attach at both ends to tendons of other muscles. Each lumbrical muscle arises from a flexor digitorum profundus tendon in the palm, passes volar to the deep transverse metacarpal ligament, and attaches to the lateral band of the extensor mechanism on the radial side (see Fig. 9–36).[161] Like the interossei muscles, the lumbrical muscles cross the metacarpophalangeal joint volarly and the interphalangeal joints dorsally. Differences in function in the two intrinsic muscle groups (lumbrical and interossei) can be attributed to: (i) the more distal insertion of the lumbrical muscles on the lateral band

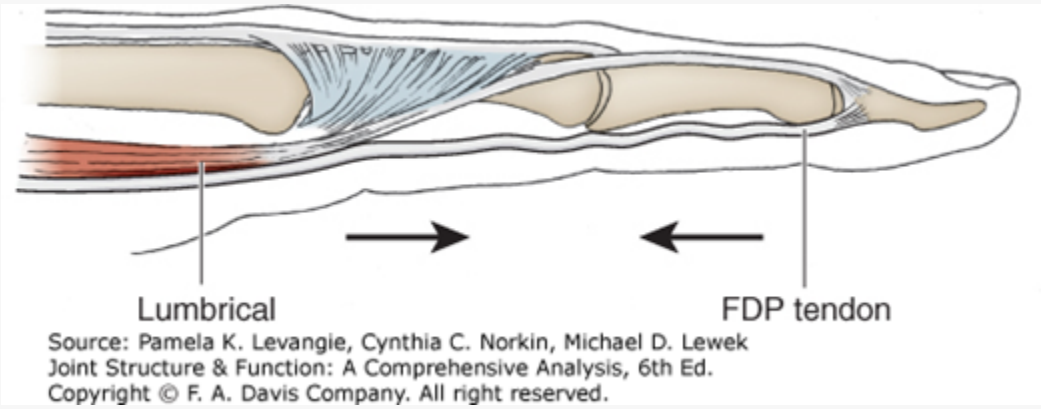
as compared with the distal interossei, (ii) the lumbrical's flexor digitorum profundus tendon origin, and (iii) the lumbrical's great contractile range.

The lumbrical muscles insert more distally on the lateral bands of the extensor mechanism than the distal interossei muscles, making the lumbricals consistently effective interphalangeal extensors regardless of metacarpophalangeal joint position. Studies have found the lumbrical muscles to be more frequently active as interphalangeal extensors in the metacarpophalangeal joint extended position than are the interossei muscles.[148,162] The deep transverse metacarpal ligament prevents the volarly located lumbrical muscle from migrating dorsally and losing tension as the metacarpophalangeal joint extends. When a lumbrical muscle contracts, it pulls not only on its distal attachment (the lateral band) but also on its proximal attachment (the flexor digitorum profundus tendon). Because the proximal attachment of the lumbrical muscle is on a somewhat movable tendon, shortening of the lumbrical muscle not only increases tension in the lateral bands to extend the interphalangeal joints but also pulls the flexor digitorum profundus tendon distally in the palm. The distal migration of the flexor digitorum profundus tendon releases much of the passive flexor tension in the inactive flexor digitorum muscle that otherwise would be present at the metacarpophalangeal and interphalangeal joints (Fig. 9–41). Ranney and Wells confirmed this, finding that the lumbrical muscles did not begin to extend the interphalangeal joints until the tension within the lumbrical muscle equaled the tension in the flexor digitorum profundus tendon (produced by the lumbrical muscle's distal pull on the flexor digitorum profundus tendon).[152] Given these circumstances, the lumbrical muscles might be considered to be both agonists and synergists for interphalangeal extension. Tension in the lumbrical muscles on the lateral bands

produces interphalangeal extension, while the lumbrical muscle simultaneously releases antagonistic tension in the flexor digitorum profundus tendon.[163] The distal insertion of the interossei muscles can extend the interphalangeal joints. However, they are less effective as interphalangeal extensors in the absence of the lumbrical muscles because the interossei muscles do not have the same ability to release the passive resistance of the flexor digitorum profundus tendon to interphalangeal extension.

FIGURE 9-41

The lumbrical muscle attaches to the flexor digitorum profundus (FDP) tendon proximally and to the lateral band of the extensor expansion distally. A contraction of the lumbrical muscle will create tension in the lateral band, leading to proximal/distal interphalangeal joint extension, concomitantly pulling the tendon of the relaxed flexor digitorum profundus distally and releasing the passive flexor tension that could impede interphalangeal extension.



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The complexity of the interconnections of the intrinsic muscles with the extensor mechanism can be highlighted not only by the interrelationships of the interossei

muscles but also by the lumbrical muscle's interdependence with the flexor digitorum profundus and extensor muscle expansion to produce interphalangeal extension. Although active interphalangeal extension is facilitated by the active lumbrical muscle's effective release of passive flexor digitorum profundus tendon tension, the lumbrical muscle is also dependent on flexor digitorum profundus tendon tension; that is, *some* tension in the flexor digitorum profundus tendon is critical to lumbrical function. If passive tension were not present in the tendon of the inactive flexor digitorum profundus muscle (e.g., if the flexor digitorum profundus tendon were cut), an active lumbrical contraction would pull the flexor digitorum profundus tendon so far distally that the lumbrical would become actively insufficient and ineffective as an interphalangeal extensor. Similarly, active or passive tension in the extensor digitorum communis tendon and extensor expansion are necessary as one source of tension before the second source of tension, the active lumbrical muscle, can be effective in fully extending both interphalangeal joints.[152] The lumbrical muscles may also assist the flexor digitorum profundus muscle indirectly with hand closure. When the flexor digitorum profundus muscle contracts, the flexor digitorum profundus tendon moves proximally, carrying its associated (presumably passive) lumbrical muscle along with it. This creates passive pull of the lumbrical muscle on the lateral band during hand closure, which may assist the flexor digitorum profundus muscle in flexing the metacarpophalangeal joint *before* the interphalangeal joints, thus avoiding the problem of catching the fingertips in the palm during grasp as occurs in the intrinsic minus hand.[4]

The lumbrical muscles' role as a metacarpophalangeal joint flexor is relatively minimal. The lumbrical muscles actually have a greater moment arm for metacarpophalangeal

joint flexion than do the interossei muscles because the lumbrical muscles lie volar to the interossei muscles. Functionally, however, metacarpophalangeal flexion is weaker in the lumbrical muscles than in the interossei muscles.[4,148,158,162,164] This relative weakness may be attributed to the small cross-section of the lumbrical muscles in comparison with the interossei muscles. However, it may also have to do with the moving attachment of the lumbrical muscle on the flexor digitorum profundus tendon. A contraction of a lumbrical muscle causes the associated flexor digitorum profundus tendon to migrate distally and carries the lumbrical muscle along with it. The distal migration of the flexor digitorum profundus tendon and lumbrical muscle has the effect both of releasing passive tension in the inactive flexor digitorum profundus tendon that might otherwise contribute to metacarpophalangeal joint flexion and simultaneously minimizing the active force of the lumbrical muscle at the metacarpophalangeal joint. Although the active lumbrical muscles may lose tension at the metacarpophalangeal joint in metacarpophalangeal flexion, metacarpophalangeal flexion does not appear to weaken the lumbricals' effectiveness as interphalangeal extensors. The unusually large contractile range of the lumbrical muscles seems to prevent the lumbrical muscles from becoming actively insufficient when shortening both over the metacarpophalangeal joints and at the interphalangeal joints.



Foundational Concepts 9–5

Intrinsic Muscles Summary

The complex functions of the interossei muscles are summarized in the Anatomy Overview at the beginning of the chapter. The function of the lumbrical muscles is simpler than that of the interossei muscles. The key points are that:

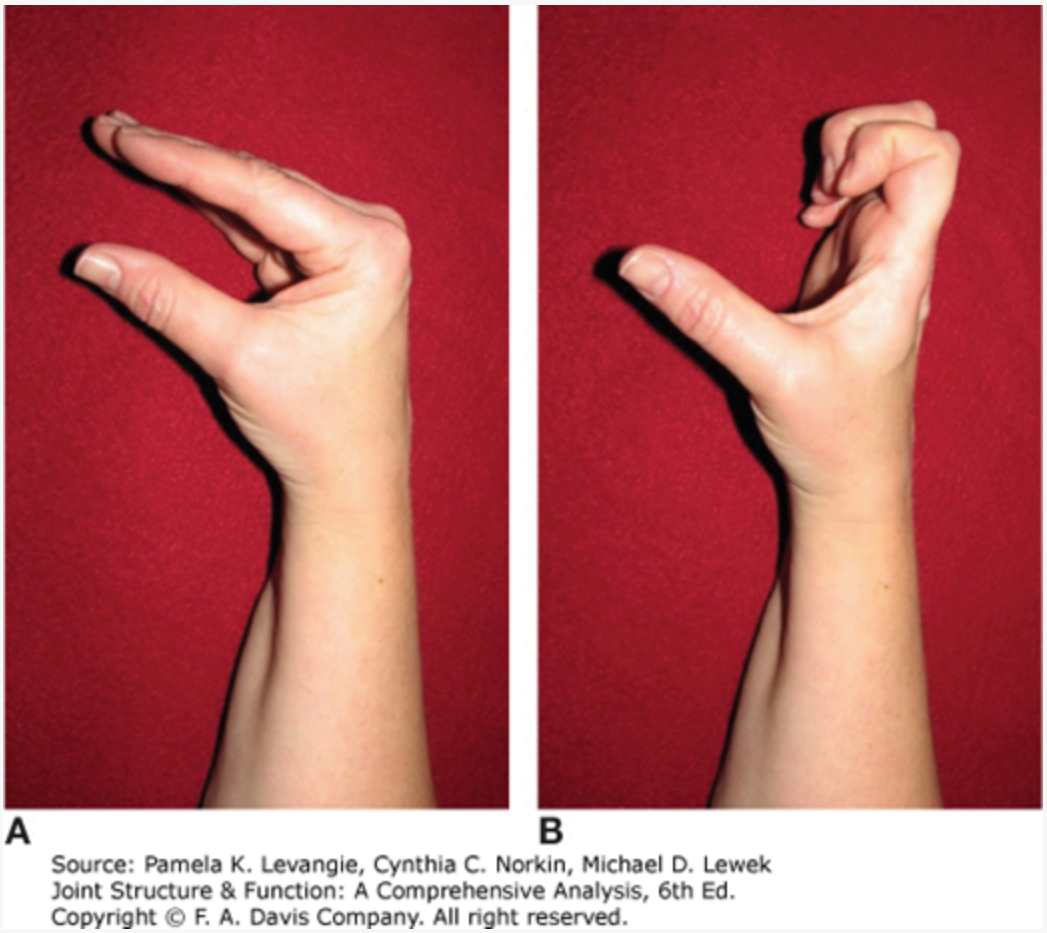
1. The lumbrical muscles are strong extensors of the interphalangeal joints,

regardless of metacarpophalangeal joint position.

2. The lumbrical muscles are relatively weak metacarpophalangeal joint flexors, regardless of metacarpophalangeal joint position.
3. The ability of the lumbrical muscles to extend the interphalangeal joints appears to depend only on intact tension in the extensor mechanism and in the flexor digitorum profundus tendons.
4. When the lumbrical and interossei muscles contract together without any extrinsic finger muscle activity, these muscles produce flexion and interphalangeal extension, the so-called **intrinsic plus** position of the hand (Fig. 9–42A).
5. When the extrinsic finger flexors and extensors are active without any concomitant activity of the intrinsic muscles, the hand assumes an intrinsic minus position (Fig. 9–42B).

FIGURE 9-42

A. Activity of the lumbrical and interossei muscles without any extrinsic finger flexor or extensor activity produces the “intrinsic plus” position of the hand (metacarpophalangeal flexion and interphalangeal extension). **B.** Activity of the extrinsic finger flexors and extensors without any activity of intrinsic finger muscles produces the “intrinsic minus” position of the hand (metacarpophalangeal extension and interphalangeal flexion).



Structure of the Thumb

Carpometacarpal Joint of the Thumb

The carpometacarpal (or **trapeziometacarpal [TM]**) joint of the thumb is the articulation between the trapezium and the base of the first metacarpal. Unlike the carpometacarpal joints of the fingers, the first carpometacarpal joint is a saddle joint with two degrees of freedom: flexion/extension and abduction/adduction.[165] The joint also permits some axial rotation, which occurs concurrently with the other motions. The net effect at this joint is a circumduction motion commonly termed **opposition.** Opposition permits the tip of the thumb to oppose the tips of the fingers.

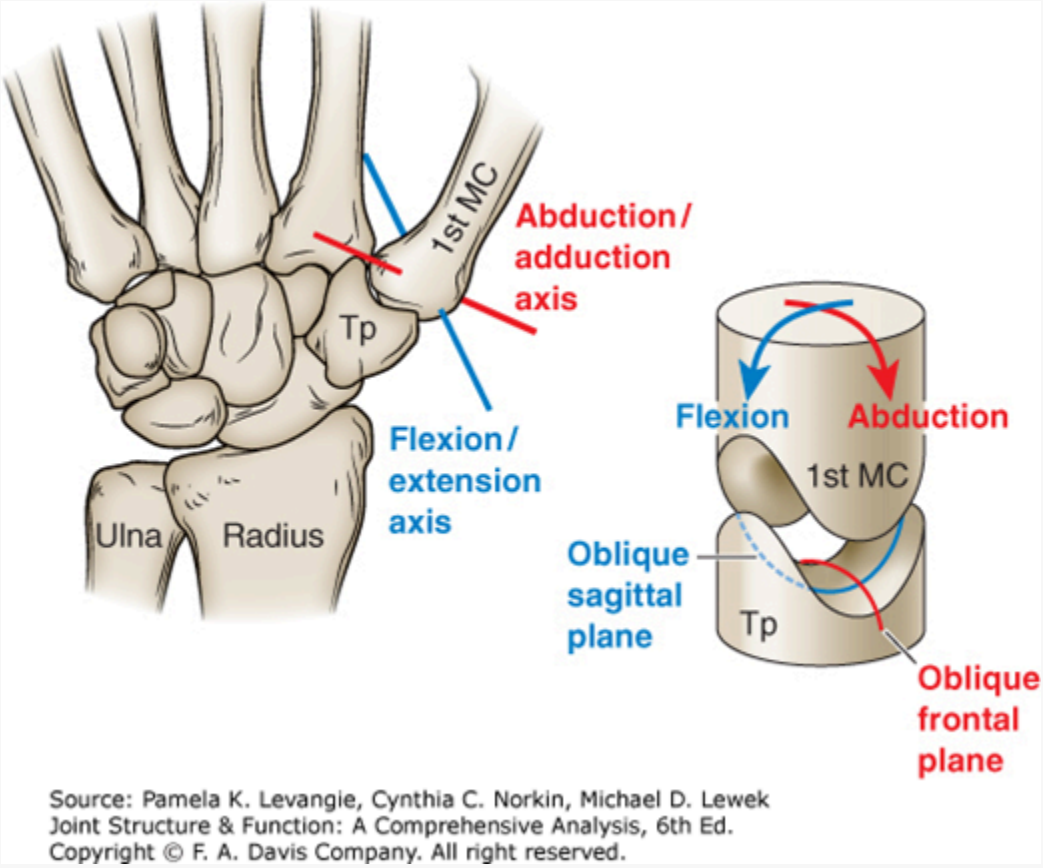
FIRST CARPOMETACARPAL JOINT STRUCTURE

Zancolli and associates proposed that the first carpometacarpal joint surfaces consist not only of the traditionally described saddle-shaped surfaces but also of a spherical portion located near the anterior radial tubercle of the trapezium.[166] The saddle-shaped portion of the trapezium is concave in the sagittal plane (abduction/adduction) and convex in the frontal plane (flexion/extension; Fig. 9–43). The spherical portion is convex in all directions. The base of the first metacarpal has a reciprocal shape to that of the trapezium. Flexion/extension and abduction/adduction are proposed to occur on the saddle-shaped surfaces, whereas the axial rotation of the metacarpal that accompanies opposition is proposed to occur on the spherical surfaces.[166] Flexion/extension of the joint occurs around a somewhat oblique A-P axis, whereas abduction/adduction occurs around an oblique coronal axis. This is a reversal of what is found at most other joints, with flexion/extension usually occurring around a coronal axis and abduction/adduction around an A-P axis. The change in the carpometacarpal joint motions occurs because the orientation of the trapezium effectively rotates the volar surface of the thumb medially. As a consequence, flexion/extension occurs nearly parallel to the palm, with abduction/adduction occurring nearly perpendicular to the palm. Cooney and associates measured the first carpometacarpal joint ROM as an average of 53° of flexion/extension, 42° of abduction/adduction, and 17° of rotation.[167]

FIGURE 9-43

Flexion/extension of the saddle-shaped first carpometacarpal joint occurs in an oblique frontal plane as movement of the convex proximal metacarpal (MC) on the concavity of the trapezium (Tp).

Flexion/extension occurs in an oblique sagittal plane as movement of the concave proximal metacarpal on the convexity of the trapezium. Atypically compared with other joint motions, flexion/extension takes place around an (oblique) A-P axis while abduction/adduction takes place around an (oblique) coronal axis.



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The capsule of the carpometacarpal joint is relatively lax but is reinforced by radial, ulnar, volar, and dorsal ligaments. There is also an **intermetacarpal ligament** that helps tether the bases of the first and second metacarpals, preventing extremes of radial and dorsal displacement of the base of the first metacarpal joint.[166,168] The **dorsoradial** and **anterior oblique ligaments** are reported to be key stabilizers of the carpometacarpal

joint.[169,170] Although some investigators hold that the axial rotation seen in the metacarpal during opposition is a function of incongruence and joint laxity,[11,171] Zancolli and associates theorized that it is a result of the congruence of the spherical surfaces and resultant tensions encountered in the supporting ligaments.[166] It seems, however, that some incongruence must exist at the joint. Osteoarthritic changes with aging are common at the first carpometacarpal joint and may be attributable to the cartilage thinning in high-load areas imposed on this joint by pinch and grasp across incongruent surfaces.[172] Ateshian and colleagues found gender differences in the fit of the trapezium with the metacarpal, with the trapezium of women showing more incongruence than that of men in a group of older individuals.[173] This matches an increased incidence of osteoarthritis of the first carpometacarpal joint among older women, but it does not address whether the incongruence of the trapezium is a cause or effect of degenerative changes. The first carpometacarpal joint is close-packed both in extremes of abduction and adduction, with maximal motion available in neutral position.[107]

FIRST CARPOMETACARPAL JOINT FUNCTION

It is the unique range and direction of motion at the first carpometacarpal joint that produces opposition of the thumb. Opposition is, sequentially, abduction, flexion, and adduction of the first metacarpal, with simultaneous rotation.[174] These movements change the orientation of the metacarpal, bringing the thumb out of the palm and positioning the thumb for contact with the fingers. The functional significance of the carpometacarpal joint of the thumb and of the movement of opposition can be appreciated when one realizes that use of the thumb against a finger occurs in almost all forms of **prehension** (grasp and dexterity activities). When the first carpometacarpal

joint is fused in extension and adduction, opposition cannot occur. The importance of opposition is such that fusion of the first carpometacarpal joint may be followed over time by adaptation of the **trapezioscaphoid joint** into a more saddle-shaped configuration to restore some of the lost opposition.[111] This amazing shift in joint function is an excellent example of the body's ability to replace essential functions whenever possible.

Patient Application 9–4

Megan Fiore is a 78-year-old woman referred to a hand surgeon by her primary care physician after she reported progressive onset of thumb pain. Her symptoms of aching and tenderness were exacerbated by activities such as turning a key, opening a jar, and writing. Manual longitudinal compression of the first metacarpal joint into the trapezium produced pain and crepitation (positive Grind test), indicative of carpometacarpal osteoarthritis. Osteoarthritic changes and subluxation of the metacarpal were evident on radiograph (**Fig. 9–44**). A custom thumb orthosis was fabricated, and the patient was instructed in activity modifications along with general joint protection principles, including avoiding forceful, repetitive, and sustained pinching, along with utilizing pens and kitchen utensils with larger handles.

FIGURE 9-44

Degenerative changes between the trapezium (Tp) and first metacarpal (M1) (first carpometacarpal joint) cause painful opposition.



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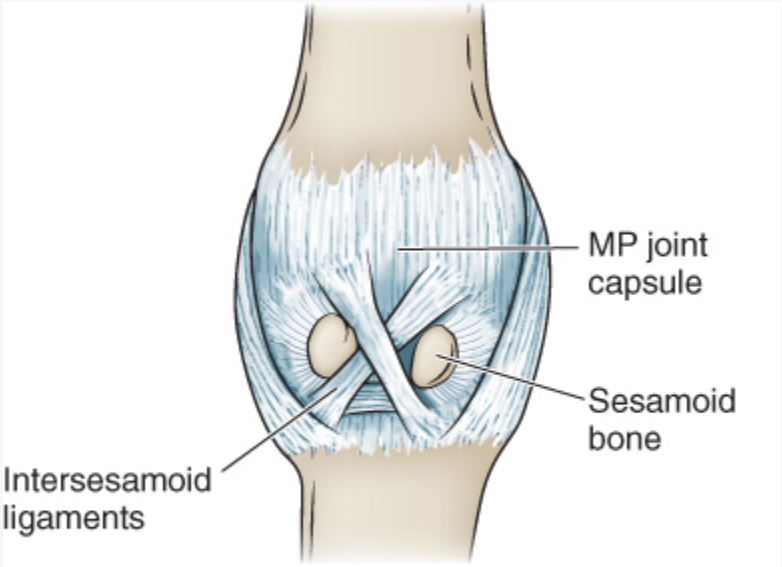
Metacarpophalangeal and Interphalangeal Joints of the Thumb

The metacarpophalangeal joint of the thumb is the articulation between the head of the first metacarpal and the base of its proximal phalanx. It is considered to be a condyloid joint with two degrees of freedom: flexion/extension and abduction/adduction.[98] There is an insignificant amount of passive rotation.[111] The metacarpal

head is not covered with cartilage dorsally or laterally, and it more closely resembles the head of the proximal phalanx, without the central groove of the proximal phalanx. The joint capsule, the reinforcing volar plate, and the collateral ligaments are similar to those of the other metacarpophalangeal joints. The main functional contribution of the first metacarpophalangeal joint is to provide additional flexion range to the thumb during opposition and to allow the thumb to grasp and contour to objects. Despite the structural similarities between the metacarpophalangeal joints, the first metacarpophalangeal joint is far more restricted in motion than those of the fingers. Although the available range varies significantly among individuals, the first metacarpophalangeal joint rarely has more than half the flexion available at the fingers and little if any hyperextension. Abduction/adduction and rotation are extremely limited. This limitation to motion is probably attributable to the major structural difference between the metacarpophalangeal joints of the thumb and fingers; the first metacarpophalangeal joint is reinforced extracapsularly on its volar surface by two sesamoid bones (Fig. 9–45). These are maintained in position by fibers from the collateral ligaments and by an **intersesamoid ligament.** Goldberg and Nathan proposed that the sesamoid bones are the result of friction and pressure on the tendons in which the sesamoid bones are embedded.[175] They support this by noting that the sesamoid bones of the first metacarpophalangeal joint do not appear until around 12 years of age and that sesamoid bones in some investigations have also been found in as many as 70% of fifth metacarpophalangeal joints and 50% of second metacarpophalangeal joints.

FIGURE 9-45

The metacarpophalangeal joint of the thumb has two sesamoid bones secured to the volar aspect of the joint capsule by intersesamoid ligaments.



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The interphalangeal joint of the thumb is the articulation between the head of the proximal phalanx and the base of the distal phalanx. It is structurally and functionally identical to the interphalangeal joints of the fingers, so it does not require a separate description.

Thumb Musculature

The muscles of the thumb have been compared with guy wires supporting a flagpole that is not set in concrete; there must be a continuous effective pull in every direction by the muscles of the thumb to maintain stability. The metacarpal and the proximal and distal phalanges form an articulated shaft that sits on the trapezium. Because the

stability comes from the muscles more than it does from articular constraints (at least at the carpometacarpal joint), the majority of muscles that attach to the thumb tend to be active during most thumb motions. There is also substantial individual variability in motor strategies among normal subjects.[176] Consequently, exploration of muscle function in the thumb (and, to a somewhat lesser extent, function throughout the hand) is not really an issue of when a muscle is active but of when the preponderance of muscle activity might be expected between differing tasks. Because of individual variability, the role of the extrinsic and intrinsic thumb muscles will be presented as generalizations (conceptual frameworks), as will the final section of this chapter, on hand prehension.

Extrinsic Thumb Muscles

There are four extrinsic thumb muscles: the flexor pollicis longus (FPL), extensor pollicis brevis (EPB), extensor pollicis longus (EPL), and abductor pollicis longus (APL) muscles. The flexor pollicis longus muscle is located volarly (see Fig. 9–15A) and is the correlate of the flexor digitorum profundus muscles of the fingers. The flexor pollicis longus tendon at the wrist is invested by the radial bursa and its typically continuous digital tendon sheath (see Fig. 9–33A). The flexor pollicis longus muscle is unique in that it functions independently of other muscles and is the only muscle responsible for flexion of the thumb interphalangeal joint.[6] The flexor pollicis longus tendon sits between the sesamoid bones at the head of the metacarpal and appears to derive some protection from those bones.

Three of the thumb extrinsic muscles are located dorsoradially. The extensor pollicis brevis and abductor pollicis longus muscles run a common course from the dorsal

forearm, traversing through the first dorsal compartment and crossing the wrist on its radial aspect (see Fig. 9–34) to their insertion. The abductor pollicis longus muscle inserts on the base of the metacarpal joint, whereas the extensor pollicis brevis muscle inserts on the base of the proximal phalanx. Both muscles abduct the carpometacarpal joint. The extensor pollicis brevis muscle also extends the metacarpophalangeal joint. The abductor pollicis longus and extensor pollicis brevis muscles also radially deviate the wrist slightly.

The extensor pollicis longus muscle originates in the forearm by the abductor pollicis longus and extensor pollicis brevis muscle, but crosses the wrist closer to the dorsal midline before using the **dorsal radial (Lister's) tubercle** as an anatomical pulley to turn toward the thumb (see Fig. 9–34); the extensor pollicis longus muscle inserts on the base of the distal phalanx. At the level of the proximal phalanx, the extensor pollicis longus tendon is joined by expansions from the **abductor pollicis brevis (APB)** muscle, the first volar interossei muscle, and the **adductor pollicis (AdP)** muscle.[21] There is no further elaboration of the extensor expansion at the metacarpophalangeal joint of the thumb (compared with what we see in the fingers), but there is the same balance of metacarpophalangeal joint abductors and adductors that contribute to resting tension in the metacarpophalangeal joint and to stabilization of the long extensor tendon. The intrinsic abductor pollicis brevis and adductor pollicis muscles that attach to the extensor pollicis longus tendon can extend the thumb interphalangeal joint to neutral but cannot complete the range into hyperextension in individuals who have that range available. The extensor pollicis longus is the only muscle that can complete the full range of hyperextension at the interphalangeal joint, as well as applying an extensor force at the metacarpophalangeal joint along with the extensor pollicis brevis muscle.[89]

The extensor pollicis longus muscle can also extend and adduct the carpometacarpal joint of the thumb. Maximal glide of the extensor pollicis longus tendon is achieved with the wrist in an extended position.[177] In contrast to the fingers, there is a separate extensor tendon for each joint of the thumb. The abductor pollicis longus muscle attaches to the base of the metacarpal, the extensor pollicis brevis muscle to the base of the proximal phalanx, and the extensor pollicis longus muscle to the base of the distal phalanx.

Because the extrinsic thumb muscles span multiple joints, they must coordinate together to influence the positioning and functioning of the thumb as a whole.[178] As is true for other extrinsic hand muscles, wrist positioning is an essential factor in providing an optimal length–tension relationship for the extrinsic muscles of the thumb.[4] The flexor pollicis longus muscle is less effective as an interphalangeal joint flexor when the wrist is in flexion. The extensor pollicis longus muscle cannot complete interphalangeal extension when the wrist, carpometacarpal, and metacarpophalangeal joints are simultaneously extended. The abductor pollicis longus and extensor pollicis brevis muscles require the synergy of an ulnar deviator of the wrist to prevent the muscles from creating wrist radial deviation that would affect their ability to generate tension over the joints of the thumb.

Intrinsic Thumb Muscles

There are four **thenar** (intrinsic thumb) muscles that originate primarily from the carpal bones and the flexor retinaculum (or transverse carpal ligament).[179] The **opponens pollicis (OP)** is the only intrinsic thumb muscle to have its distal attachment on the first metacarpal. Its action line is nearly perpendicular to the long axis of the metacarpal

joint and is applied to the lateral side of the bone. The opponens pollicis muscle, therefore, is very effective in positioning the metacarpal in an abducted, flexed, and rotated posture. The abductor pollicis brevis, flexor pollicis brevis, and adductor pollicis muscles all insert on the thumb's proximal phalanx. The flexor pollicis brevis muscle has two heads of insertion. Its larger lateral head attaches distally with the abductor pollicis brevis muscle and also applies some abductor force. The flexor pollicis brevis muscle crosses the sesamoid bones at the metacarpophalangeal joint, increasing the moment arm of the flexor pollicis brevis muscle for metacarpophalangeal joint flexion. The medial head of the flexor pollicis brevis muscle attaches distally with the adductor pollicis muscle and assists in thumb adduction.

Although not generally considered a thenar muscle, the first dorsal interossei muscle may make a contribution to thumb function, along with its contribution to metacarpophalangeal flexion and interphalangeal extension of the index finger. The first dorsal interossei muscle is a bipennate muscle arising from both the first and second metacarpals and from the intercarpal ligament that joins the metacarpal bases. Brand and Hollister proposed that the first dorsal interossei muscle is a carpometacarpal joint distractor rather than a joint compressor as is typical of most muscles, because it pulls the first metacarpal distally toward the first dorsal interossei muscle's insertion on the base of the index proximal phalanx (Fig. 9–46).[4] These investigators also argued that the thumb attachment of the first dorsal interossei muscle has little or no ability to move the thumb but is important in offsetting the compressive and dorsoradially directed forces that the flexor/adductor muscles create across the carpometacarpal joint in lateral pinch and power grip. When flexor/adductor forces were created in laboratory specimens without tension in the first dorsal

interossei muscle, the carpometacarpal joint subluxed.[4] Belanger and Noel suggested that the first dorsal interossei muscle can assist with thumb adduction.[180]

FIGURE 9-46

Brand and Hollister proposed that the first dorsal interossei muscle is a distractor of the first carpometacarpal joint, which helps offset the strong compressive forces that occur across the first carpometacarpal joint.[4]



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The thenar muscles are active in most grasping activities, regardless of the precise position of the thumb as it participates. The opponens pollicis muscle works together most frequently with the abductor pollicis brevis and the flexor pollicis brevis muscles, although the intensity of the relation varies. When the thumb is *gently* brought into contact with any of the fingers, activity of the opponens pollicis muscle predominates

in the thumb, and abductor pollicis brevis activity exceeds that of the flexor pollicis brevis muscle. When opposition to the index finger or middle finger is performed *firmly,* activity of the flexor pollicis brevis muscle exceeds that of the opponens pollicis muscle. As opposition moves to the ring and little fingers, the proportion of opponens pollicus activity increases, equaling activity of the flexor pollicis brevis muscle with firm opposition to the little finger.[70] The change in balance of muscle activity with firm opposition and with increasingly ulnar opposition can be accounted for by the increased need for thumb abduction and metacarpal rotation. Increased pressure in opposition additionally appears to bring in activity of the adductor pollicis muscle. The adductor pollicis muscle stabilizes the thumb against the opposed finger. In *firm* opposition to the index and middle fingers, adductor pollicis activity exceeds the very minimal activity of the abductor pollicis brevis muscle. With more ulnarly located opposition, the increased need for abduction of the thumb results in simultaneous activity of the thumb's intrinsic abductor and adductor.[70]

Activity of the extrinsic thumb musculature in grasp appears to be partially a function of helping to position the metacarpophalangeal and interphalangeal joints. The main function of the extrinsic muscles is in release: returning the thumb to extension (out of the palm) from its position in the palm. Although release of an object is essentially an extrinsic function, some opponens pollicis and abductor brevis activity have been identified.[70] This muscular activity may assist in maintaining the thumb in abduction and in maintaining the first metacarpal rotation that facilitates the next move of the thumb back into opposition.

The joint structure and musculature of the wrist complex, the fingers, and the thumb

have each been examined in this chapter. Some instances of specific muscle activity have been presented to clarify the potential function of the muscle. A summary of wrist and hand function, however, can best be presented through the assessment of purposeful hand activity. Because the entire upper limb is geared toward execution of movement of the hand, it is appropriate to complete the description of the upper limb by looking at an overview of the wrist and hand in prehension activities.

Prehension

Prehension activities of the hand involve the grasping or taking hold of an object between any two surfaces in the hand; the thumb participates in most but not all prehension tasks. There are numerous ways that objects of varying sizes and shapes may be grasped, with strategies also varying among individuals. Consequently, the nomenclature related to these functional patterns also varies.[181] In spite of nomenclature differences, a broad classification system for grasp has evolved that will permit general observations about the coordinated muscular function necessary to produce or maintain common forms of grasp.

Prehension can be categorized as either **power grip** (full hand prehension) or **precision handling** (finger–thumb prehension; Fig. 9–47).[182] Each of these two categories has subgroups that further define the grasp. Power grip is generally a forceful act resulting in flexion at all finger joints. When the thumb is used, it acts as a stabilizer to the object held between the fingers and, most commonly, the palm. Precision handling, in contrast, is the skillful placement of an object between fingers or between finger and thumb.[183] The palm is not involved. Landsmeer suggested that power grip and

precision handling can be differentiated on the basis of the dynamic and static phases.[184] Power grip is the result of a sequence of (1) opening the hand, (2) positioning the fingers, (3) bringing the fingers to the object, and (4) maintaining a static phase that actually constitutes the grip. Although precision handling shares the first three steps of the sequence, it does not contain a static phase at all. In power grip, the object is grasped so that the object can be moved through space by the more proximal joints; in precision handling, the fingers and thumb grasp the object for the purpose of manipulating it between the fingers or within the hand.

FIGURE 9-47

Prehension generally consists of either **A.** power grip, in which the object makes full contact with the palm and is moved through space, or **B.** precision handling, in which the thumb and fingers dynamically manipulate the object.



A



B

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In assessment of the muscular function during each type of grasp, synergy of the hand muscles results in almost constant activity of all intrinsic and extrinsic muscles.[4,185,186] The task becomes more one of identifying when muscles are *not* working or when the balance of activity between muscles might shift. It should also be emphasized that the

muscular activity documented by EMG studies is specific to the activity as performed in a given study. Even in studies using similar forms of prehension, variables such as size of object, firmness of grip, timing, and instructions to the subject can cause substantial changes in reported muscle activity. However, as indications of general muscular activity patterns, the studies are useful in the development of a conceptual framework within which hand function can be understood.

Power Grip

The fingers in power grip usually function in concert to clamp on and hold an object in the palm. The fingers assume a position of sustained flexion that varies in degree with the size, shape, and weight of the object. The palm is likely to contour to the object as the palmar arches form around it. The thumb may serve as an additional surface to the finger–palm vise by adducting against the object, or it may be removed from the object. When involved, the thumb generally is adducted to clamp the object to the palm. This is in contrast to precision handling in which the thumb is more likely to be abducted.[187] Four varieties of power grip studied by Long and associates exemplify the similarities and differences seen in power grip.[124] These are the cylindrical grip (Fig. 9–48), the spherical grip, hook grip, and lateral prehension (Fig. 9–49).

FIGURE 9-48

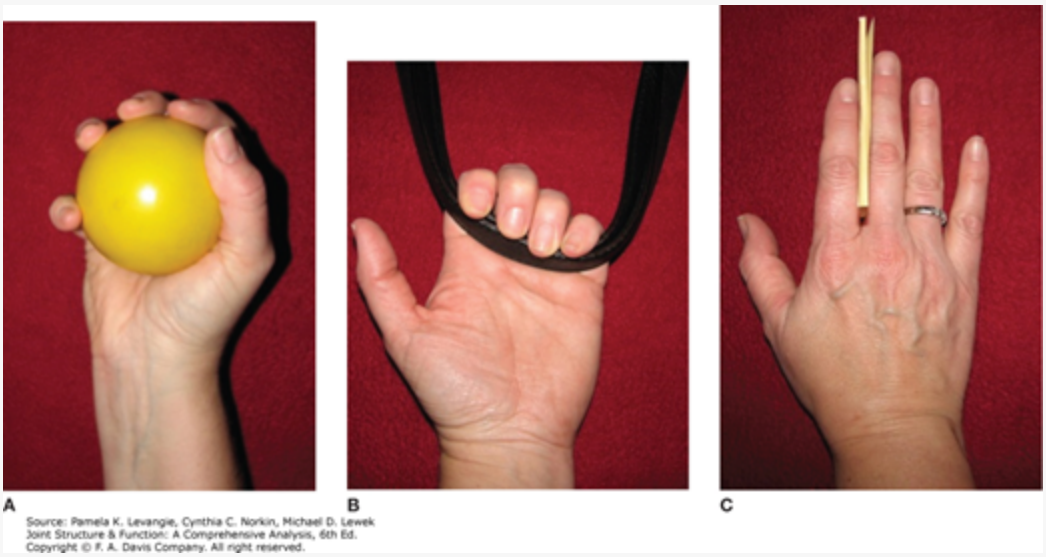
In cylindrical grip, the MP joints may ulnarly deviate using the interossei muscles to help orient the fingers toward the thumb. Ulnar deviation of the wrist may help this orientation.



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FIGURE 9-49

Other forms of power grip, besides cylindrical grip: **A.** Spherical grip, **B.** Hook grip. **C.** Lateral prehension.



Cylindrical Grip

Cylindrical grip (Fig. 9–48) predominantly uses the extrinsic finger flexors to position the fingers around and maintain grasp on an object. The function in the fingers is performed largely by the flexor digitorum profundus muscle, especially in the dynamic closing action of the fingers. In the static phase, the flexor digitorum superficialis muscles assist when the intensity of the grip requires greater force. Although power grip traditionally has been thought of as exclusively an extrinsic muscle activity, studies have indicated considerable interosseous (intrinsic) muscle activity. In strong grip, the magnitude of torque production of the interossei muscles for metacarpal flexion was found to nearly equal that of the extrinsic flexors.[4,164,183] Because both the metacarpophalangeal and the interphalangeal joints are being flexed during cylindrical grip, the metacarpophalangeal joint flexion task most likely falls to the proximal (dorsal) interossei muscles because their attachments to the proximal phalanx and hood do not have a significant (and antagonistic) interphalangeal extension influence. The interossei muscles may also ulnarly deviate the metacarpophalangeal joint (Fig.

9–48) to direct the distal phalanges of the fingers toward the thumb. The combination of metacarpophalangeal joint flexion and ulnar deviation (adduction for the index finger and abduction for the middle, ring, and little fingers) points the fingers toward the thumb but also tends to produce ulnar subluxation forces on the metacarpophalangeal joints and on the tendons of the long flexors at the metacarpophalangeal joint. The subluxing forces are ordinarily counteracted by the radial collateral ligaments, by the annular pulleys that anchor the flexor long tendons in place, and by the sagittal bands that connect the volar structures to the extensor mechanism. Active or passive tension in the extensor digitorum communis muscle can further stabilize the restraining mechanisms, as well as increase joint compression and enhance overall joint stability during power grip.[183] Although the location of the lumbrical muscles indicates a possible contribution to metacarpophalangeal joint flexion in power grip, their lack of EMG activity, regardless of strength grip, is consistent with their predominant role as interphalangeal extensors.[124]

Thumb position in cylindrical grip is the most variable of the digits. The thumb usually comes around the object, then flexes and adducts to close the vise (see Fig. 9–48). The flexor pollicis longus and thenar muscles are all active. The activity of the thenar muscles will vary with the width of the web space, with the carpometacarpal rotation required, and with increased pressure or resistance. A distinguishing characteristic of power grip over precision handling is, in general, the greater magnitude of activity in the adductor pollicis muscle during power grip. The extensor pollicis longus muscle may be variably active as a metacarpophalangeal joint stabilizer or adductor.

Muscles of the **hypothenar eminence** usually are active in cylindrical grip. The abductor

digiti minimi functions as a proximal interosseous muscle to flex and abduct (ulnarly deviate) the fifth metacarpophalangeal joint. The opponens digiti minimi and the **flexor digiti minimi** muscles are more variable but frequently are active in direct proportion to the amount of abduction and rotation of the first metacarpal. In fact, increased activity of the opponens pollicis muscle automatically results in increased activity of the opponens digiti minimi and flexor digiti minimi muscles.

Cylindrical grip is typically performed with the wrist in neutral flexion/extension and slight ulnar deviation (see Fig. 9–48). Ulnar deviation also puts the thumb in line with the long axis of the forearm; this alignment better positions the object in the hand to be turned by pronation/supination of the forearm as, for example, in turning a door knob.[187] Ulnar deviation of the wrist is the position that optimizes force of the long finger flexors. The least finger flexion force is generated when the wrist is in flexion.[2] The heavier an object is, the more likely it is that the wrist will ulnarly deviate. In addition, a strong contraction of the flexor carpi ulnaris muscle at the wrist will increase tension on the transverse carpal ligament. This provides a more stable base for the active hypothenar muscles that originate from that ligament. It is interesting to note that regardless of wrist position, the percentage of total interphalangeal flexor force allocated to each finger is relatively constant. The ring and little fingers can generate only 70% of the flexor force of the index and middle fingers.[2] The ring and little fingers seem to serve as weaker but more mobile assists to the more stable and stronger index and middle fingers. The contribution of the ring and little finger to grip can be improved if full flexion of the joints in those fingers (and concomitant loss of[7] tension) is prevented by an object that is wider ulnarly than radially (the pistol-grip shape; see Fig. 9–32A).

Spherical Grip

Spherical grip (Fig. 9–49A) is similar in most respects to cylindrical grip. The extrinsic finger and thumb flexors and the thenar muscles follow similar patterns of activity and variability. The main distinction can be made by the greater spread of the fingers to encompass the object. This evokes more interosseous activity than is seen in other forms of power grip.[124] The metacarpophalangeal joints do not deviate in the same direction (e.g., ulnarly) but tend to abduct. The phalanges are no longer parallel to each other, as they commonly are in cylindrical grip. The metacarpophalangeal joint abductors must be joined by the adductors to stabilize the joints that are in the loose-packed position of semiflexion at the metacarpophalangeal joints. Although flexor activity predominates in the digits as it does in all forms of power grip, the finger extensors do have a role. The extensors not only provide a balancing force for the flexors but also are essential for smooth and controlled opening of the hand and release of the object. Opening the hand during object approach and release is primarily an extensor function, calling in activity of the lumbrical, extensor digitorum communis, and thumb extrinsic muscles.

Hook Grip

Hook grip (Fig. 9–49B) is actually a specialized form of prehension. It is included in power grip because it has more characteristics of power grip than of precision handling. It is a function primarily of the fingers. It may include the palm but never includes the thumb. It can be sustained for prolonged periods of time as anyone can attest who has carried a briefcase or books at his or her side or hung onto a commuter strap on a bus or train. The major muscular activity is provided by the flexor digitorum profundus and flexor digitorum superficialis muscles. The load may be sustained completely by one

muscle or the other, or by both muscles in concert. This depends on the position of the load in relation to the phalanges. If the load is carried more distally so that distal interphalangeal flexion is mandatory, the flexor digitorum profundus muscle must participate. If the load is carried more in the middle of the fingers, the flexor digitorum superficialis muscle may be sufficient. Some interosseous muscle activity has been demonstrated on EMG, but its purpose is not fully understood. It may help prevent clawing in the metacarpophalangeal joints, although the activity is not evident in every finger.[124] In hook grip, the thumb is held in moderate to full extension by thumb extrinsic muscles.

Lateral Prehension

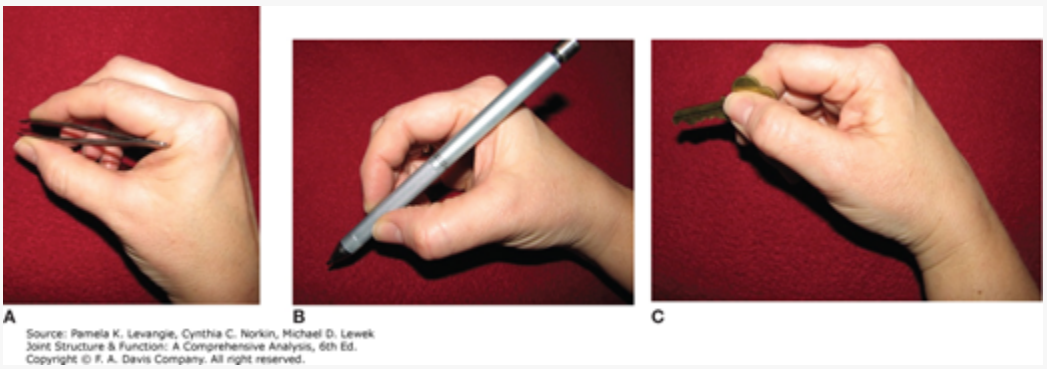
Lateral prehension (Fig. 9–49C) is a unique form of grasp. Contact occurs between two adjacent fingers. The metacarpophalangeal and interphalangeal joints are usually maintained in extension while the other metacarpophalangeal joints simultaneously abduct and adduct. This is the only form of prehension in which the finger extensor musculature predominates in the maintenance of the posture. The extensor digitorum communis and the lumbrical muscles are active to extend the metacarpophalangeal and interphalangeal joints, and metacarpophalangeal joint abduction and adduction are performed by the interossei muscles. Lateral prehension is included here as a form of power grip because lateral prehension involves the static holding of an object that is then moved by the more proximal joints of the upper extremity. Although not a "powerful" grip, neither is lateral prehension used to manipulate objects in the hand. It is generally typified by holding a cigarette.

Precision Handling

The positions and muscular requirements of precision handling are somewhat more variable than those of power grip, require much finer motor control, and are more dependent on intact sensation. The thumb serves as one *jaw* of what has been termed a **two-jaw chuck;** the thumb is generally abducted and rotated from the palm. The second and opposing *jaw* is formed by the distal tip, the pad, or the side of a finger. When two fingers are opposed to the thumb simultaneously, it is called a **three-jaw chuck.** The three varieties of precision handling are **pad-to-pad prehension, tip-to-tip prehension,** and **pad-to-side prehension** (Fig. 9–50)**.** Each tends to be a dynamic function with relatively little static holding.

FIGURE 9-50

Three varieties of precision handling: **A.** Pad-to-pad prehension. **B.** Tip-to-tip prehension. **C.** Pad-to-side prehension. (KIA video – Gripping Tasks Precision, Gripping with Rheumatoid Arthritis)



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Pad-to-Pad Prehension

Pad-to-pad prehension involves opposition of the pad, or pulp, of the thumb to the pad, or pulp, of the finger (Fig. 9–50A)**.** The pad of the distal phalanx of each digit has the

greatest concentration of tactile corpuscles found in the body. Of all forms of precision handling, 80% are considered to fall into the category of pad-to-pad.[1] The finger used in two-jaw chuck is usually the index; in three-jaw chuck, the middle finger is added. The metacarpophalangeal and proximal interphalangeal joints of the fingers are partially flexed, with the degree of flexion being dependent on the size of the object being held. The distal interphalangeal joint may be fully extended or in slight flexion. When the distal interphalangeal joint is extended, the flexor digitorum superficialis muscle can perform the metacarpophalangeal and proximal interphalangeal flexion alone without the assistance of the flexor digitorum profundus muscle. Extension of the distal interphalangeal joint in this instance is caused by flexion of the middle phalanx (flexor digitorum superficialis muscle) against the upward force of the object or thumb on the distal phalanx in what is effectively a closed chain. When partial flexion of the distal phalanx is required by the pad-to-pad task, the flexor digitorum profundus muscle must be active. Interosseous activity is often present both to supplement metacarpophalangeal joint flexor force and to provide the metacarpophalangeal joint abduction or adduction required in object manipulation. In dynamic manipulation, the volar interossei and dorsal interossei muscles tend to work reciprocally, rather than in the synergistic co-contraction pattern observed during power grip. With increased force of pad-to-pad pinch, the interossei muscles may again co-contract.[124]

The thumb in pad-to-pad prehension is held in carpometacarpal flexion, abduction, and rotation (opposition). The first metacarpophalangeal and interphalangeal joints may be partially flexed or fully extended (see Fig. 9–50A). The thenar muscle control is provided by the opponens pollicis, flexor pollicis brevis, and abductor pollicis brevis muscles, each of which is innervated by the median nerve. The adductor pollicis activity (ulnar

nerve innervated) increases with increased pressure of pinch. In ulnar nerve paralysis, loss of adductor pollicis function (as well as loss of function of the first dorsal interossei and first volar interossei muscles) makes the thumb less stable and affects the precision of the grasp activity.

Fine adjustments in the flexion angle of the distal interphalangeal joint of the finger and the interphalangeal joint of the thumb control the points of contact on the pads of the digits. If the finger's distal interphalangeal and thumb's interphalangeal joint are in extension, contact occurs on the more proximal portion of the pad of the finger (see Fig. 9–50A). As flexion of the finger's distal interphalangeal and thumb interphalangeal joints increase, the contact moves distally toward the nails. Flexion of the distal phalanx, when required, is provided by the flexor digitorum profundus muscle for the finger and by the flexor pollicis longus muscle for the thumb. Distal interphalangeal flexion in the finger is accompanied by a proportional flexion in the proximal interphalangeal joint.

As is found in power grip, the extensor musculature is used for opening the hand to grasp, for release, and for stabilization when necessary. In the thumb, the extensor pollicis longus muscle may be used to maintain the interphalangeal joint in extension when contact is light and on the proximal portion of the pad of the finger. Synergistic wrist activity must also occur to balance the forces created by the flexor digitorum superficialis and flexor digitorum profundus muscles. The wrist is more typically held in neutral radial/ulnar deviation and slight extension.[185]

Tip-to-Tip Prehension

Although the muscular activity found in tip-to-tip prehension (see **Fig. 9–50B**) is nearly

identical to that of pad-to-pad prehension, there are some key differences.[124] In tip-to-tip prehension, the interphalangeal joints of the finger and thumb must have the range and available muscle force to create nearly full joint flexion. The metacarpophalangeal joint of the finger opposing the thumb must also be ulnarly deviated (with fingertip pointed radially) to present the tip of the finger to the thumb. In the first finger, the ulnar deviation occurs as metacarpophalangeal joint adduction. If opposition occurs with one of the other fingers, the ulnar deviation is produced by metacarpophalangeal abduction. If the flexion range for the distal phalanx in either the opposing finger or the thumb is not available, or if the active force for interphalangeal flexion and metacarpophalangeal joint ulnar deviation cannot be provided, tip-to-tip prehension cannot be performed effectively. As the most precise form of grasp, it is also the most easily disrupted. Tip-to-tip prehension has all the same muscular requirements as pad-to-pad prehension in both fingers and thumb. In addition, however, activity of the flexor digitorum profundus, flexor pollicis longus, and interossei muscles is a necessity in tip-to-tip prehension, whereas they are not a necessity in pad-to-pad prehension.

Pad-to-Side Prehension

Pad-to-side prehension is also known as **key grip** (or **lateral pinch**) because a key is held between the pad of the thumb and side of the index finger (see Fig. 9–50C). Pad-to-side prehension differs from the other forms of precision handling only in that the thumb is more adducted and less rotated. The activity level of the flexor pollicis brevis muscle increases and that of the opponens pollicis muscle decreases in comparison with tip-to-tip prehension. Activity of the adductor pollicis muscle also increases over that seen in either tip-to-tip or pad-to-pad prehension.[14] Slight flexion of the distal phalanx of the thumb is required. If the pad-to-side prehension is being used for something like

turning a key, the wrist will again assume neutral flexion/extension and drop into slight ulnar deviation to put the key in line with the forearm so that pronation or supination can be used to turn the key.

Pad-to-side prehension is the least precise of the forms of precision handling; it can actually be performed by a person with paralysis of all hand muscles. If the hand muscles are paralyzed as they would be in a person with a spinal cord injury above the C7 level, active wrist extensors (assuming they are present) can create pad-to-side prehension. Wrist extension provided by the intact and active extensor carpi ulnaris, extensor carpi radialis longus, and extensor carpi radialis brevis muscles contributes to metacarpophalangeal and interphalangeal joint flexion of the fingers and thumb by generating passive tension in the extrinsic finger flexor tendons (flexor digitorum superficialis and flexor digitorum profundus) as the tendons are stretched over the extending wrist. The grip may be released by relaxing the wrist extensor muscles and allowing gravity to flex the wrist. As the wrist flexes, the tendons of the flexor digitorum superficialis and flexor digitorum profundus muscles become slack, and the tendons of the extensor digitorum communis (with the related extensor indicis proprius and extensor digiti minimi tendons) and extensor pollicis longus tendons become stretched. The passive tension in the long finger extensors in a dropped (flexed) wrist is adequate to partially extend both metacarpophalangeal and interphalangeal joints. The phenomenon of using active wrist extension to passively close the fingers and passive wrist flexion to passively open the fingers is known as **tenodesis** (KIA Video – Tenodesis). The same tenodesis action can achieve a cylindrical grip if the proper balance of tension exists in the extrinsic flexors. The flexors must be loose enough to permit the partially flexed fingers of the *open* hand to surround the object in wrist flexion, while

still being tight enough to hold onto the object when the wrist is extended. Active control of at least one wrist extensor muscle is the minimal requirement for functional use of tenodesis in a person without any active control of finger or thumb musculature. Tenodesis was described in Chapter 3 (see Fig. 3–25) when we first discussed passive insufficiency. As was noted then, tenodesis can and does also occur in the fully intact hand, although the presence of balancing muscles permits us to override it.

Functional Position of the Wrist and Hand

There can be little doubt that the hand cannot function either as a manipulator or as a sensory organ unless an object can enter the palmar surface and unless moderate finger flexion and thumb opposition are available to allow sustained contact. Application of either an active muscular or passive tendinous flexor force to the digits requires the wrist to be stabilized in moderate extension and ulnar deviation. Delineation of the so-called functional position of the wrist and hand takes into account these needs and is the position from which optimal hand function is most likely to occur. It is *not necessarily* the position in which a hand should be immobilized. Position for hand immobilization will be dependent upon the injury or disability.

The functional position is:

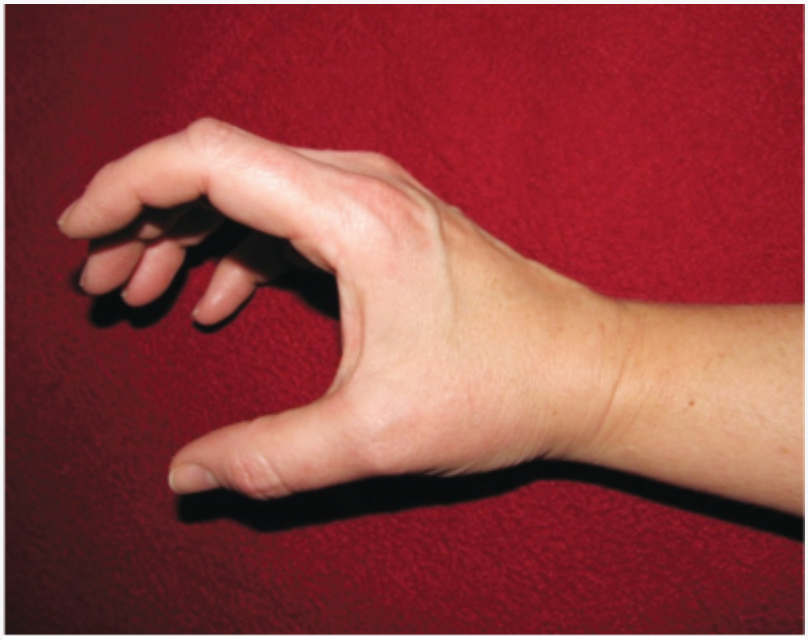
1. Wrist in slight extension (20°)
2. Wrist in slight ulnar deviation (10°)
3. Fingers moderately flexed at the metacarpophalangeal joints (45°)

4. Fingers moderately flexed at the proximal interphalangeal joints (30°)

5. Fingers slightly flexed at the distal interphalangeal joints (Fig. 9–51)[1]

FIGURE 9-51

Functional position of the hand: wrist extension and ulnar deviation with moderate flexion of the metacarpophalangeal and interphalangeal joints of the finger and thumb. (***KIA***/Passive Insuffiency)



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
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The wrist position optimizes the power of the finger flexors so that hand closure can be accomplished with the least possible effort. It is also the position in which all wrist muscles are under equal tension. With similar considerations for the position of the joints of the digits, the functional position provides the best opportunity for the

disabled hand to interact with the brain that controls it.

Summary

- Despite the many articulations that make up the hand and wrist complex, the bony and ligamentous components of these joints have less potential for problems than the musculotendinous structures that cross and act on other joints.
- The motor control of and sensory feedback from the wrist and hand alone occupy more space topographically on the primary motor and sensory cortices of the brain than does the entire lower extremity.
- As we proceed to examine the joints of the lower extremity, an analogy to the corresponding joints of the upper extremity can and should be made. However, the primary weightbearing function of the lower extremities does not require the complexity and delicate balance of muscular control that can so profoundly affect functional performance in the hand.

Study Questions

1. Name the bones of the wrist complex; describe the articulations that occur between these bones and the functional joints that are formed.
2. Describe the components and role of the triangular fibrocartilage complex (TFCC) in wrist function.
3. What is the total ROM normally available at the wrist complex? How are the

motions distributed between the radiocarpal and midcarpal joints of the complex?

4. Describe the sequence of carpal motion occurring from full wrist flexion to full extension and radial to ulnar deviation, emphasizing the role of the scaphoid.

5. What effect does release of the scapholunate stabilizers or of the lunotriquetral stabilizers have on bony positions?

6. Identify the muscles that can extend the wrist; include the joints crossed, actions produced, and activity levels of each.

7. Describe the transverse carpal ligament, its attachments, and its role in wrist and hand function.

8. What is the function of the carpometacarpal joints of the fingers? How do the variations in ROM among the four carpometacarpal joints of the fingers contribute to function?

9. What role does the transverse metacarpal ligament play at the carpometacarpal joint? What role at the metacarpophalangeal joint?

10. Describe the locations and functions of the volar (palmar) fibrocartilage plates.

11. What metacarpophalangeal joint position is most prone to injury and why?

12. Compare the joint structure of the metacarpophalangeal joints with that of the interphalangeal joints of the fingers. Identify both similarities and differences.

13. Describe the mechanisms, joint motions, and muscles that are necessary for the fingers to gently close into the palm without friction or loss of the length-tension relationship.

14. How does the “pistol-grip” design of most tools (larger ulnarly) relate to the metacarpophalangeal joint ROM and muscular function of the four fingers?

15. When is the flexor digitorum superficialis muscle active as the primary finger flexor? When does it back up the flexor digitorum profundus muscle?

16. What muscles are active in gentle closure of the normal hand? What role, if any, do the intrinsic muscles play in this activity?

17. What wrist position is assumed when a person needs to optimize finger flexion strength? Which wrist position is least effective for grasp?

18. What are annular pulleys and cruciate ligaments in the digits? Where are they found, and what functions do they serve?

19. Identify the bursae of the hand. What are their functions and how are they most typically related to the digital tendon sheaths?

20. Describe the active and passive elements that make up the extensor mechanism. What are the specific functions of each?

21. What role do the extensor digitorum communis, extensor indicis proprius, and extensor digiti minimi muscles play in active extension of the proximal interphalangeal and distal interphalangeal joints of the hand?

22. How do the proximal and distal attachments of the interossei muscles affect function at the metacarpophalangeal and interphalangeal joints?

23. Describe the attachments of the lumbrical muscles to the extensor mechanism. How do these muscles contribute to interphalangeal extension? What is their role at the metacarpophalangeal joint?

24. Why is active distal interphalangeal joint flexion normally accompanied by proximal interphalangeal joint flexion at the same time?

25. Explain why the distal interphalangeal joint cannot be actively extended if the proximal interphalangeal joint is fully flexed.

26. Why will an isolated contraction of the extensor digitorum communis muscle produce flexion of the proximal interphalangeal and distal interphalangeal joints? What is this finger position called?

27. How are the extrinsic flexors and extensors stabilized at the metacarpophalangeal joints?

28. Why is finger extension weaker in the index and little fingers?

29. Why does metacarpophalangeal joint adduction weaken more quickly than abduction in a progressive ulnar nerve problem?

30. Which are stronger flexors of the metacarpophalangeal joint, the lumbrical or the interossei muscles?

31. Compare and contrast the metacarpophalangeal joint structure of the thumb with the metacarpophalangeal joint structure of the fingers.

32. What does the motion of thumb opposition require in terms of joint function and musculature?

33. What are the primary muscles of extension (release) in the wrist and hand?

34. In general, what is the difference between power grip and precision handling at the wrist, in the fingers, and in the thumb? What do these two forms of prehension have in common? Name 2 functional activities specific to each.

35. Cylindrical grip is generally referred to as an extrinsic hand function. Why is this true?

36. What requirement does spherical grip have that differentiates it from cylindrical grip?

37. Which form of prehension requires only intrinsic musculature?

38. Which forms of prehension do not require the thumb?

39. What roles do interossei muscles play in precision handling?

40. What requirements does tip-to-tip prehension have that are not necessary for pad-to-pad prehension?

41. What is the finest (most precise) form of prehension that can be accomplished by someone without intact hand musculature, assuming availability of an active

wrist extensor?

42. What is the functional position of the wrist and hand? Why is this the optimal resting position when there is no specific hand problem?

43. Why is an ulnar nerve injury called “claw hand”? What deficiency causes the clawing, and in which fingers does it occur?

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Chapter 10: The Hip Complex

RobRoy L. Martin; Benjamin Kivlan

Anatomy Overview

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MUSCLE ACTIONS OF THE HIP (FEMOROACETABULAR JOINT)		
Muscle actions are considered with the hip beginning from anatomical position. Muscle actions can change if activated from a different position.		
TABLE KEY: Primary muscle Secondary muscles Dependent on hip position		
Sagittal Plane	Flexion	Extension
	Iliopsoas Rectus femoris Tensor fascia latae Sartorius Pectineus Adductor longus Adductor magnus Gracilis Gluteus minimus (anterior fibers)	Gluteus maximus Semitendinosus Semimembranosus Biceps femoris, long head Gluteus medius (posterior fibers) Adductor magnus (posterior fibers) Piriformis
Frontal	Abduction	Adduction

Plane		
	Gluteus medius Gluteus minimus Tensor fascia latae Piriformis Sartorius Rectus femoris	Adductor magnus Pectineus Adductor longus Adductor brevis Gracilis Obturator externus
Transverse Plane	Medial rotation	Lateral rotation
	Tensor fascia latae Gluteus minimus Gluteus medius (anterior fibers) Pectineus Adductor longus and brevis Adductor magnus (posterior fibers) Semimembranosus Semitendinosus	Gluteus maximus Piriformis Quadratus femoris Obturator internus and externus Gemellus superior and inferior Sartorius Gluteus medius (posterior fibers) Biceps femoris, long head

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Muscle	Proximal Attachment	Distal Attachment
Iliopsoas	*Psoas major* and *minor:* transverse processes and lateral aspect of T12–L5 vertebral bodies and discs *Iliacus:* Iliac crest, iliac fossa, sacral ala and anterior SI ligaments	Lesser trochanter
Rectus femoris	AIIS	Quadriceps tendon to patella and ultimately tibial tuberosity
Tensor fascia latae	ASIS	Iliotibial (IT) band to lateral condyle of tibia
Sartorius	ASIS	Superior aspect of medial tibia
Pectineus	Superior ramus of pubis	Pectineal line (femur), just inferior to lesser trochanter
Adductor longus	Body of pubis, inferior to pubic crest	Middle third of linea aspera (femur)
Adductor brevis	Body and inferior ramus of pubis	Pectineal line and proximal part of linea aspera (femur)
Adductor magnus	Inferior ramus of pubis to ischial	Gluteal tuberosity to

	tuberosity	adductor tubercle of femur via linea aspera (femur)
Gracilis	Body and inferior ramus of pubis	Superior aspect of medial tibia
Gluteus maximus	Posterior ilium, sacrum and coccyx	Iliotibial (IT) band to lateral condyle of tibia
Gluteus medius	Lateral surface of ilium between anterior and posterior gluteal lines	Greater trochanter (femur)
Gluteus minimus	Lateral surface of ilium between anterior and inferior gluteal lines	Greater trochanter (femur)
Semimembranosus	Ischial tuberosity	Posterior aspect of medial tibial condyle
Semitendinosus	Ischial tuberosity	Superior aspect of medial tibia
Biceps femoris, long head	Ischial tuberosity	Lateral aspect of fibular head
Piriformis	Anterior surface of sacrum	Greater trochanter (femur)
Obturator externus	Edge of obturator foramen	Trochanteric fossa (femur)
Obturator internus	Pelvic edge of obturator foramen	Greater trochanter to trochanteric fossa (femur)
Gemellus superior and inferior	*Superior:* ischial spine *Inferior:* ischial tuberosity	Greater trochanter to trochanteric fossa (femur)

Quadratus femoris	Ischial tuberosity	Inter-trochanteric crest (femur)

Introduction

The hip joint, also called the **coxofemoral** or **femoroacetabular joint,** is the articulation of the acetabulum of the pelvis and the head of the femur (Fig. 10–1). These two segments form a diarthrodial ball-and-socket joint with three degrees of freedom: flexion/extension in the sagittal plane, abduction/adduction in the frontal plane, and medial/lateral rotation in the transverse plane. Although the hip joint and the shoulder complex have a number of common features, each has undergone such extensive functional and structural adaptations to support their respective roles that comparisons between these joints are more for general interest than functional relevance. For example, the role of the shoulder complex is to provide a stable base on which a wide range of mobility for the hand can be superimposed. Shoulder complex structure gives precedence to this open-chain function. Instead, the primary function of the hip joint is to support the weight of the head, arms, and trunk (HAT) in static erect posture and dynamic postures such as ambulation, running, and stair climbing. The hip joint, like the other joints of the lower extremity, is structured primarily to serve its weightbearing function. Although we examine hip joint structure and function as if the joint were designed to move the foot through space in an open chain, hip joint structure is more influenced by the demands placed on the joint when the limb is bearing weight. As we shall see later in this chapter, weightbearing function of the hip joint and its

related weightbearing responses are basic to understanding the hip joint and the interactions that occur between the hip joint and the other joints of the spine and lower extremities.

FIGURE 10-1

The hip joint is the connection between the leg and the trunk and is important during many functional activities.



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Patient Application 10–1

Gabriella Martinez is a 32-year-old woman currently teaching kindergarten, a position she has held for the past 10 years. Gabriella has been bothered with recurrent left hip pain that has been intermittent throughout her young adult life. She recalls several

instances of hip pain that interrupted her participation in gymnastics and dance as a teenager. Although Gabriella no longer participates in dance or gymnastics, she has remained physically active until recently by running as well as taking Pilates and yoga classes.

Over the past few months, she has experienced increasing problems, predominantly on her left side, that interfere with climbing the stairs in her two-story home, performing activities with her kindergarten class, and caring for her 3-year-old daughter. In addition to pain complaints, she also reports feeling as though her hip may "pop out of the socket" when she is doing twisting poses in yoga and has an occasional painful "click" in the hip. It has become too painful for her to run and she has suspended participation in her exercise classes. She remembers being told by a physician when she was in her teens that she would probably have problems with her hips when she was older because her hips were "shallow."

Gabriella localizes her primary pain to her left groin, although the lateral hip area can also be painful. On clinical examination, Gabriella is observed to walk with reduced hip extension on the left. Pain occurs during mid to late stance phase and swing phase on the left. She also has a slight left lateral lean during left stance. During examination, you find that Gabriella has pain with passive hip flexion with medial rotation on the left. In the supine position, there is an increase in medial rotation and decrease in lateral rotation at the left hip.

Structure and Function of The Hip Joint

Structure

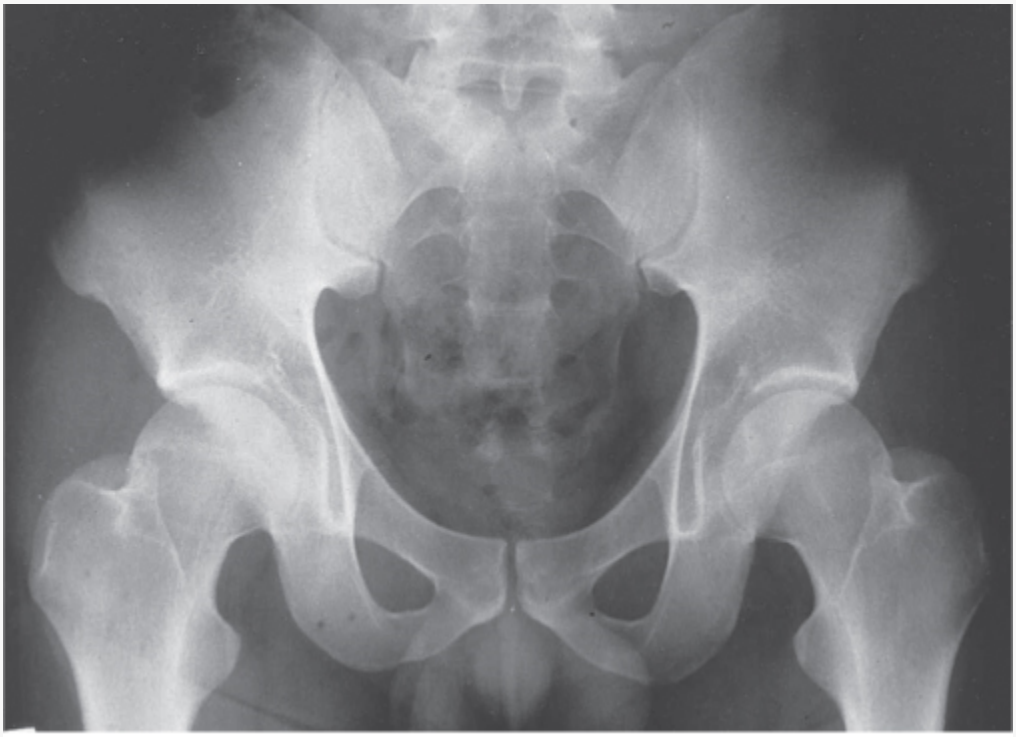
Proximal Articular Surface

The cuplike concave socket of the hip joint is called the **acetabulum** and is located on the lateral aspect of the **innominate** or **os coxa** (pelvic bone; Fig. 10–2). Three bones form the pelvis: the ilium, the ischium, and the pubis. Each of the three bones contributes to the structure of the acetabulum (Fig. 10–3). The pubis forms one-fifth of the acetabulum, the ischium forms two fifths, and the ilium forms the remainder (Fig. 10–3A). Until full ossification of the pelvis occurs between 20 and 25 years of age, the

separate segments of the acetabulum may remain visible on radiograph (Fig. 10–3B).[1]

FIGURE 10-2

The hip joint is formed by the head of the femur and the acetabulum of the innominate bone (one half) of the pelvis.

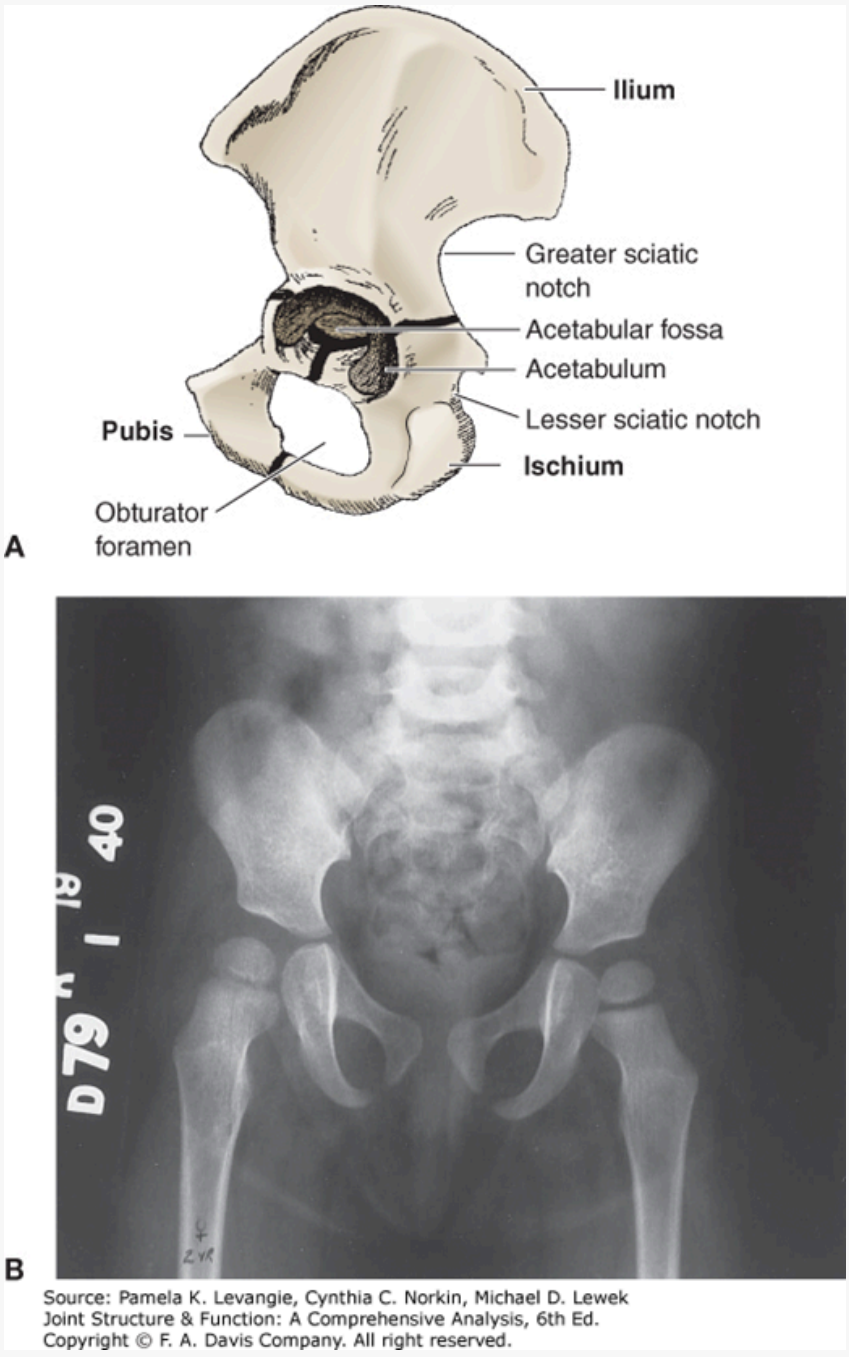


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FIGURE 10-3

A. The acetabulum is formed by the union of the three bones of the pelvis, with only the upper horseshoe-shaped area being articular. **B.** In this radiograph of a 2-year-old without impairments, the cartilaginous rather than bony union of the acetabulum is clearly

evident.



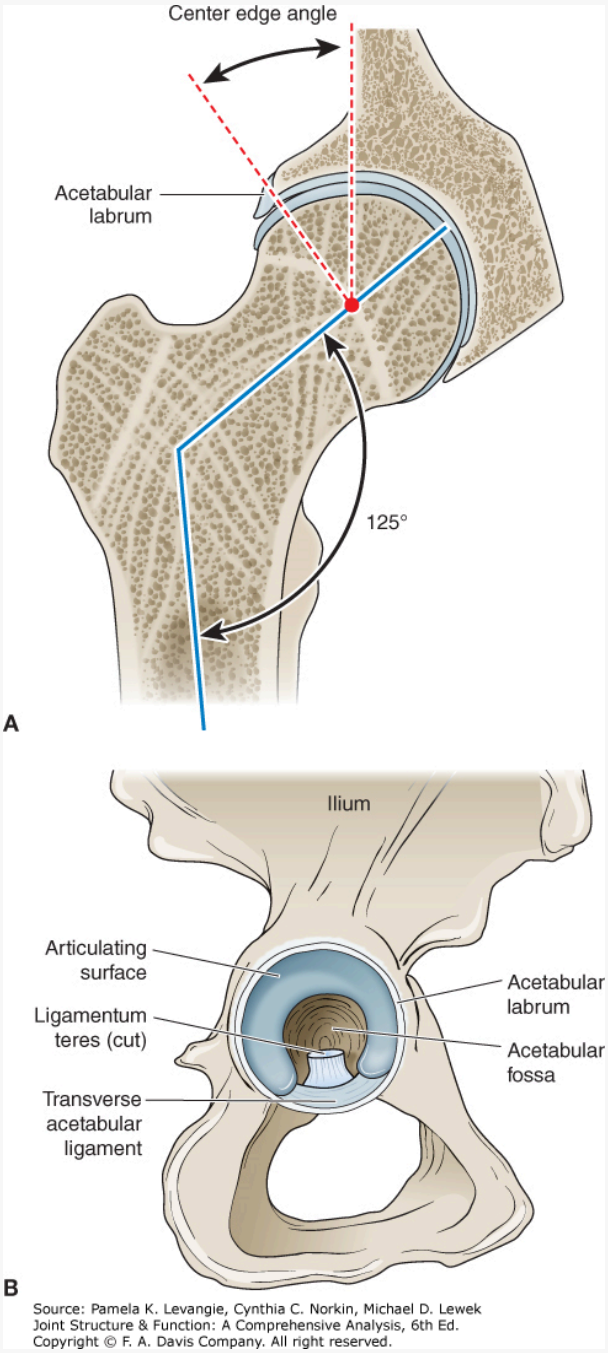
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The **lunate surface** (the periphery of the acetabulum) is covered with hyaline cartilage. This horseshoe-shaped area of cartilage articulates with the head of the femur (see Fig. 10–3A) and allows for contact stress to be uniformly distributed.[2] The inferior aspect of

the lunate surface (the base of the horseshoe) is interrupted by a deep notch called the **acetabular notch,** and is spanned by a fibrous band, the **transverse acetabular ligament,** which connects the two inferior ends of the lunate surface. As the transverse acetabular ligament spans the acetabular notch, it creates a fibro-osseous tunnel that contains fibroelastic fat covered with synovial membrane through which blood vessels pass into the central and deepest portion of the acetabulum, called the **acetabular fossa.** Unlike the lunate surface, the acetabular fossa does not articulate with the femoral head (Fig. 10–4).

FIGURE 10-4

A. The center edge angle of the acetabulum is formed between a vertical line through the center of the femoral head and a line connecting the center of the femoral head and the bony edge of the acetabulum. **B.** The acetabular labrum deepens the acetabulum.



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ACETABULUM

Although appearing to be spherical, only the upper margin of the acetabulum has a true circular contour.[3] The opening of the acetabulum faces inferiorly by 50° and is rotated anteriorly (anteverted) 20°.[4–7] Women will generally have a few degrees greater

inclination and anteversion compared with males.[4] The articular surface area of the acetabulum tends to be smaller in women than it is in men.[6] Normal functioning of the hip joint requires optimal femoral head coverage by the acetabulum. Femoral head coverage is largely determined by acetabular depth. *Acetabular dysplasia, coxa profunda, acetabular protrusio, anteversion,* and *retroversion* are terms used to describe acetabular abnormalities that can potentially lead to pathology, including excessive cartilage wear and osteoarthritis.[6]

Acetabular dysplasia is an abnormally shallow acetabulum that results in a lack of femoral head coverage.[7] It may contribute to instability of the hip joint or lead to disproportionate loading of the superior acetabular rim. **Coxa profunda** and **acetabular protrusio** are terms used to describe conditions in which the acetabulum excessively covers the femoral head. Acetabular over coverage can lead to mechanical restrictions of range of motion (ROM) and impingement between the femoral head–neck junction and the acetabulum.[7] Acetabular depth can be measured as the **center edge angle** (see Fig. 10–4).[8] The center edge angle is formed by a line connecting the lateral rim of the acetabulum and the center of the femoral head and a vertical line from the center of the femoral head. Center edge angles are classified as follows: definite dysplasia (less than 16°), possible dysplasia (16° to 25°), and normal (greater than 25°).[8] Lateral center edge angles greater than 40° may indicate excessive acetabular over coverage.[7]

Abnormalities in acetabular depth, inclination, and version (abnormal positioning in the transverse plane) affect femoral head coverage. **Anteversion** of the acetabulum exists when the acetabulum is positioned anteriorly in the transverse plane; **retroversion** exists when the acetabulum is positioned posteriorly in the transverse

plane. An acetabulum that is positioned with less inclination or more anteversion can lead to instability. Likewise, an acetabulum that is positioned with more inclination or retroversion can lead to over coverage and impingement between the acetabulum femoral head–neck junction.[7]

ACETABULAR LABRUM

The acetabular labrum is a ring of wedge-shaped fibrocartilage attached to the outer periphery of the acetabulum (see labrum cross-section in Fig. 10–4). The labrum is attached to the periphery of the acetabulum by a zone of calcified cartilage.[10] The labrum of the hip is analogous to the meniscus of the knee and the labrum of the glenohumeral joint. The acetabular labrum deepens the socket and increases the concavity of the acetabulum through its triangular shape, grasping the head of the femur to maintain contact with the acetabulum. The labrum also acts as a seal to maintain negative intra-articular pressure and enhance stability.[9] Nerve endings within the labrum may provide proprioceptive feedback to further enhance joint stability.[11] However, the nerve endings may also be a source of pain.[11] When the labrum is compromised, friction stresses increase, which may lead to deterioration of the articular cartilage of the hip joint that defines osteoarthritis. Although the normal hip is intrinsically stable because of the deeply recessed acetabulum, an abnormally shallow acetabulum will increase stress on the surrounding capsule and labrum.

The transverse acetabular ligament is considered to be a continuation of the acetabular labrum. Histologically, it is predominantly ligamentous, but contains cartilaginous cells believed to withstand compressive loads.[12] However, experimental data do not support the transverse acetabular ligament as a load-bearing structure.[13] The ligament is

believed to serve primarily as a tension band between the anteroinferior and posteroinferior aspects of the acetabulum (the “feet” of the horseshoe-shaped articular surface) that protects the blood vessels traveling beneath it to reach the head of the femur.[13]

Patient Application 10–2

Gabriella’s left groin pain is provoked by passive hip flexion and medial rotation.[14] This clinical finding may be indicative of damage to the anterosuperior labrum and possibly a site of osteoarthritic changes.[15,16] Due to her age, history of hip pain, and clinical findings, her physician ordered a magnetic resonance arthrogram (MRA) to identify the pathoanatomical source of her pain. After reviewing her MRA, Gabriella’s physician confirmed a superior anterior labral tear and early stage osteoarthritis. Acetabular labral tears are increasingly recognized as a source of hip pain and as a starting point for degenerative changes at the acetabular rim.[11,15,17–20] Damage to the labrum was evident in 96% of postmortem and cadaveric hip joints in persons 61 to 98 years of age, with 74% showing damage in the anterosuperior quadrant.[11] Isolated labral tears are uncommon and usually occur in conjunction with bony abnormalities of the acetabulum or femoral neck junction.[21] Although a labral tear is not always symptomatic, a torn labrum can potentially cause anterior groin pain, clicking, locking, catching, instability, giving way, or joint stiffness.[14,22,23]

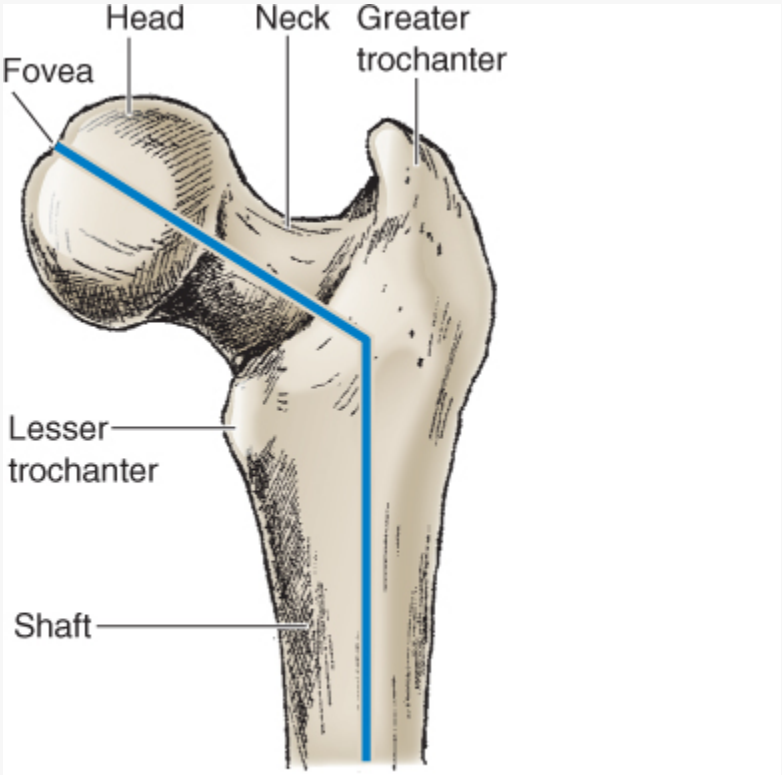
Distal Articular Surface

The head of the femur is a rounded hyaline cartilage covered surface.[24] The articular area of the femoral head forms approximately two-thirds of a sphere and is more circular than the acetabulum.[3,25] Femoral shape and dimensions can be variable even in bones of the same overall size.[25] The radius of curvature of the femoral head is smaller in women than in men in comparison with the dimensions of the pelvis.[25,26] Just inferior to the most medial point on the femoral head is a small, roughened pit called the **fovea** or **fovea capitis** (Fig. 10–5). The fovea is not covered with articular

cartilage and is the point at which the **ligamentum teres** (the ligament of the head of the femur) is attached.

FIGURE 10-5

Posterior view of the proximal portion of the right femur shows the relationship between the head, neck, trochanters, and femoral shaft.



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The femoral head is attached to the femoral neck, which in turn is attached to the shaft of the femur between the greater and lesser trochanters. The femoral neck is, in general, only about 5 cm long and may vary slightly based on gender and ethnicity.[14,27] The femoral neck is angulated so that the femoral head faces medially, superiorly, and anteriorly with respect to the femoral shaft and distal femoral condyles.[15]

ANGULATION OF THE FEMUR

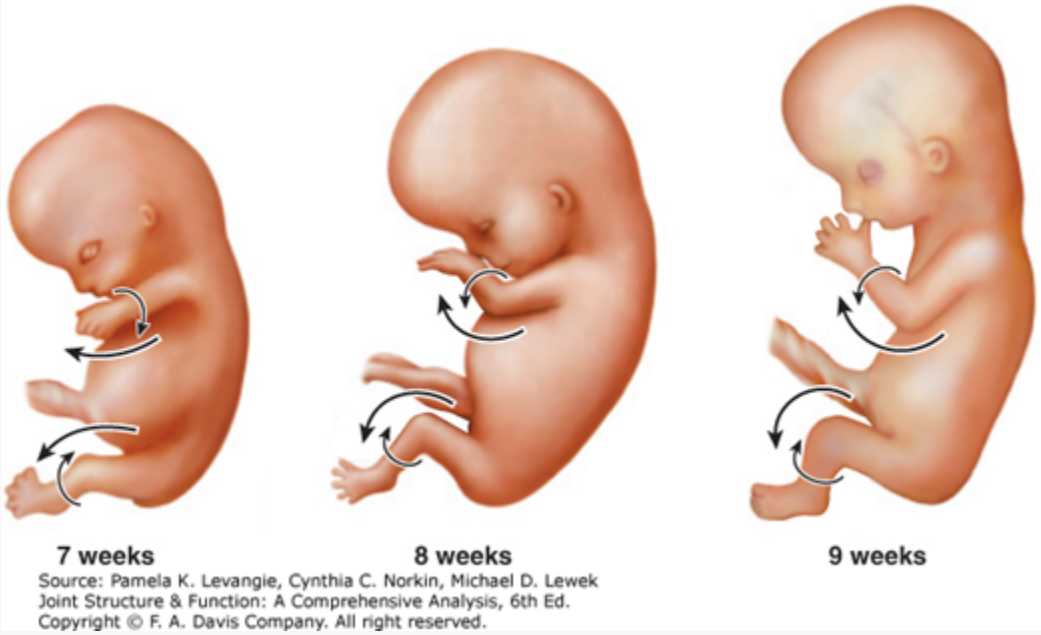
There are two angulations made by the head and neck of the femur in relation to the shaft. The **angle of inclination** occurs in the frontal plane between an axis through the femoral head and neck and the longitudinal axis of the femoral shaft. The **angle of torsion** occurs in the transverse plane between an axis through the femoral head and neck and an axis through the distal femoral condyles. The origin and variability of these angulations can be understood in the context of the embryonic development of the lower limb.

In the early stages of fetal development, both upper extremity and lower extremity limb buds project laterally from the body as if in full abduction (Fig. 10–6). During the seventh and eighth weeks of gestational age and before full definition of the joints, adduction of the buds begins. At the end of the eighth week, the "fetal position" has been achieved, but the upper and lower limbs are no longer positioned similarly. Although the upper limb buds have undergone torsion somewhat laterally (so that the ventral surface of the limb bud faces anteriorly), the lower limb buds have undergone torsion medially, so that the ventral surface faces posteriorly.[14] The result for the lower limb is critical to understanding function. The knee flexes in the opposite direction from the elbow, and the extensor (dorsal) surface of the lower limb is located anteriorly rather than posteriorly. Although the head and neck of the femur retain the original position of the limb bud, the femoral shaft is inclined medially and undergoes medial torsion with regard to the head and neck. The magnitude of medial inclination and torsion of the distal femur (with regard to the head and neck) is dependent on embryonic growth and, presumably, fetal positioning during the remaining months of uterine life. The development of the angulations of the femur appears to continue after

birth and through the early years of development. Both normal and abnormal angles of inclination and torsion of the femur are *properties of the femur alone.* However, abnormalities in the angulations of the femur can substantially alter hip joint stability, weightbearing biomechanics of the hip joint, and muscle biomechanics. Likewise, such abnormalities in femoral angulation can influence compensatory changes in the remainder of the lower extremity and spine.

FIGURE 10-6

During fetal development the upper and lower extremities rotate in opposite directions.



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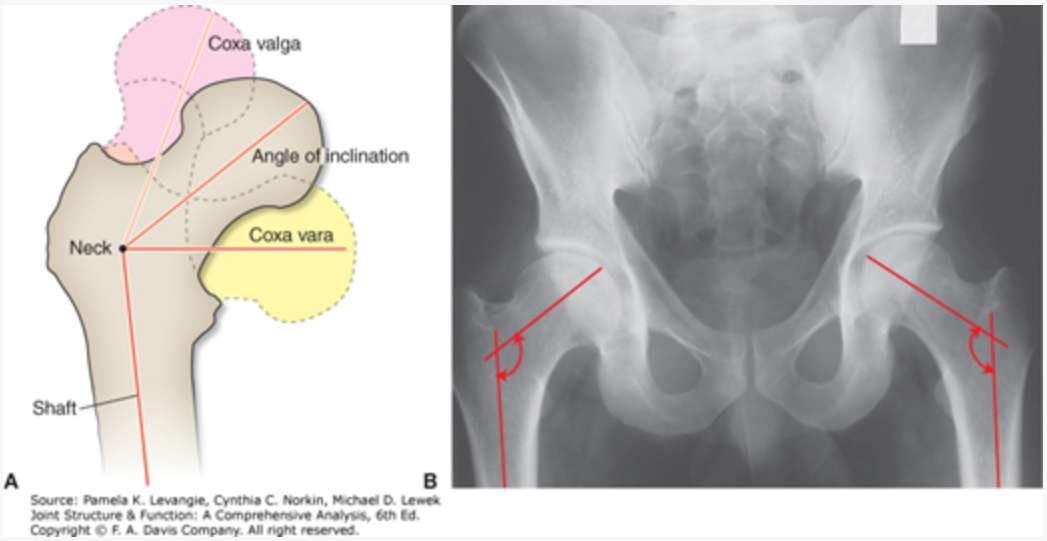
ANGLE OF INCLINATION OF THE FEMUR

The angle of inclination of the femur is approximately 125° (referencing the medial angle formed by the axes of the head-neck and the shaft; Fig. 10–7).[16,17] This value can have a normal range from 110° to 144° in the unimpaired adult (Fig. 10–7A).[18–20,25,26] As

with the angle of inclination of the humerus, there are variations not only among individuals but also from side to side. In women, the angle of inclination is somewhat smaller than it is in men, owing to the greater width of the female pelvis.[14] With a normal angle of inclination, the greater trochanter lies at the level of the center of the femoral head.[21] The angle of inclination of the femur changes across the life span; it approximates 150° at birth (see Fig. 10–3B) and gradually declines to about 125° at skeletal maturity.[16,17] This angle can continue to decline again in the elderly.[19] A pathologically greater medial angulation between the neck and shaft is called **coxa valga,** and a pathologically smaller angle is called **coxa vara** (Fig. 10–8).

FIGURE 10-7

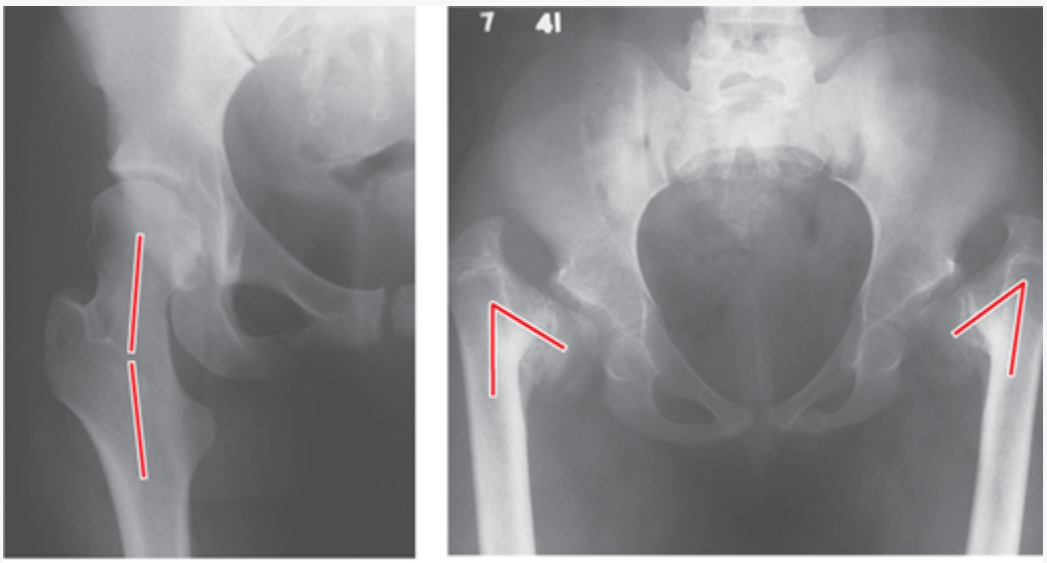
The axis of the femoral head and neck forms an angle with the axis of the femoral shaft called the *angle of inclination.* **A.** Greater medial angles are called *coxa valga,* whereas a smaller medial angle is called *coxa vara.* **B.** In this adult subject without impairments, the angles are slightly less than 130°, with a couple of degrees of variation from side to side.



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FIGURE 10-8

Abnormal angles of inclination found in two young adults with developmental hip dysplasia. **A.** A pathological increase in the angle of inclination is called *coxa valga.* **B.** A pathological decrease in the angle is called *coxa vara.*



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Both coxa vara and coxa valga can lead to abnormal lower extremity biomechanics, altered muscle function, and gait abnormalities that contribute to pathologies such as labral pathology, hip and knee osteoarthritis, and slipped capital femoral epiphysis.[22,23,28,29]

In coxa valga (see Fig. 10–8A), the angle of inclination in the femur is greater than the normal adult angle of 125°. The increased angle brings the vertical weightbearing line closer to the shaft of the femur, diminishing the shear, or bending, force across the femoral neck. The reduction in force across the femoral neck is reflected in a reduction in density of the lateral trabecular system.[30] However, the decreased distance between the femoral head and the greater trochanter also decreases the length of the moment arm of the hip abductor muscles. The decreased muscular moment arm results in an increased demand for muscular force generation to maintain sufficient abduction torque to counterbalance the gravitational adduction moment acting around the supporting hip joint during single-limb support. Either the additional muscular force requirement will increase the total joint reaction force within the hip joint or the abductor muscles will be unable to meet the increased demand and will be functionally weakened. Although the strength of the abductors may be otherwise normal, the reduction in biomechanical effectiveness may produce the compensations typical of primary abductor muscle weakness. Coxa valga also decreases the amount of femoral articular surface in contact with the dome of the acetabulum. As the femoral head points more superiorly, there is a decreasing amount of coverage from the acetabulum superiorly. Consequently, coxa valga decreases the stability of the hip and predisposes the hip to dislocation.[7,31] This may further lead to femoroacetabular impingement and the progression of labral tearing, loss of joint stability, and eventual arthrosis.[32]

Coxa vara (see Fig. 10–8B) is considered to give the advantage of improved hip joint stability (if angle reduction is not too extreme). The apparent improvement in congruence occurs because the decreased angle between the neck and shaft of the femur will place the femoral head deeper into the acetabulum, decreasing the amount of articular surface exposed superiorly and increasing coverage from the acetabulum. A varus femur, if not caused by trauma, may also increase the length of the moment arm of the hip abductor muscles by increasing the distance between the femoral head and the greater trochanter.[33] The increased moment arm decreases the amount of force that must be generated by the abductor muscles in single-limb support and reduces the joint reaction force. However, coxa vara has the disadvantage of increasing the bending moment along the femoral head and neck. This increase in bending force can be seen by the increased density of trabeculae laterally in the femur, caused by the increase in tensile stresses.[34] The increased shear force along the femoral neck will also increase the predisposition toward a femoral neck fracture.[35]

Coxa vara may increase the likelihood in the adolescent that the femoral head will slide on the cartilaginous epiphysis of the head of the femur. In childhood, the epiphysis is fairly horizontal.[7] Consequently, the superimposed weight merely compresses the head into the epiphyseal plate. In adolescence, growth of the bone results in a more oblique orientation of the epiphyseal plate.[36] The epiphyseal obliquity makes the plate more vulnerable to shear forces at a time when the plate is already weakened by the rapid growth that occurs during this period of life.[28] Weightbearing forces may slide the femoral head inferiorly, resulting in a **slipped capital femoral epiphysis.** The surgical interventions to correct coxa vara deformities in children may come with inherent complications.[37–40] Despite correction of the deformity, a common complication and

resultant failure of the procedure is an unstable femoral epiphysis. Optimal results require addressing the stability of the proximal femoral epiphysis and exemplifies the vulnerability of the growth plate of the femur in patients with a preexisting coxa vara deformity. As is true for a hip fracture, the altered biomechanics and at-risk blood supply necessitate restoration of normal alignment before secondary degenerative changes can occur.



Foundational Concepts 10–1

Angle of Inclination of the Femur

- Normal angle of inclination of the femur in an adult is approximately 125°.
- A pathological increase in the angle of inclination is called coxa valga and often creates a “longer” limb, and may be associated with joint instability, particularly when the hip is in adduction. Individuals who present with coxa valga are often observed to adopt positions of hip abduction and are commonly accompanied by genu vara.
- A pathological decrease in the angle of inclination is called coxa vara and often creates a “shorter” limb length. Coxa vara is associated with greater hip stability, but the low angle of the femoral neck makes it susceptible to femoral neck fractures or a higher incidence of a slipped capital femoral epiphysis. Cases of coxa vara are often accompanied by a position of increased hip adduction and genu valgum.



Expanded Concepts 10–1

Slipped Capital Femoral Epiphysis

Slipped capital femoral epiphysis (SCFE) is the most common adolescent hip disorder occurring when the femoral head displaces posteriorly on the femoral neck at the level of the growth plate (physis). A decrease in femoral neck-shaft angle (coxa vara) and high body mass index increase the shear force across the physis.[28,38] The risk can be reduced with weight loss to lower body mass index.[39] Slipped capital femoral epiphysis can cause widening of the femoral head–neck junction and lead to internal impingement with the acetabulum.[38]

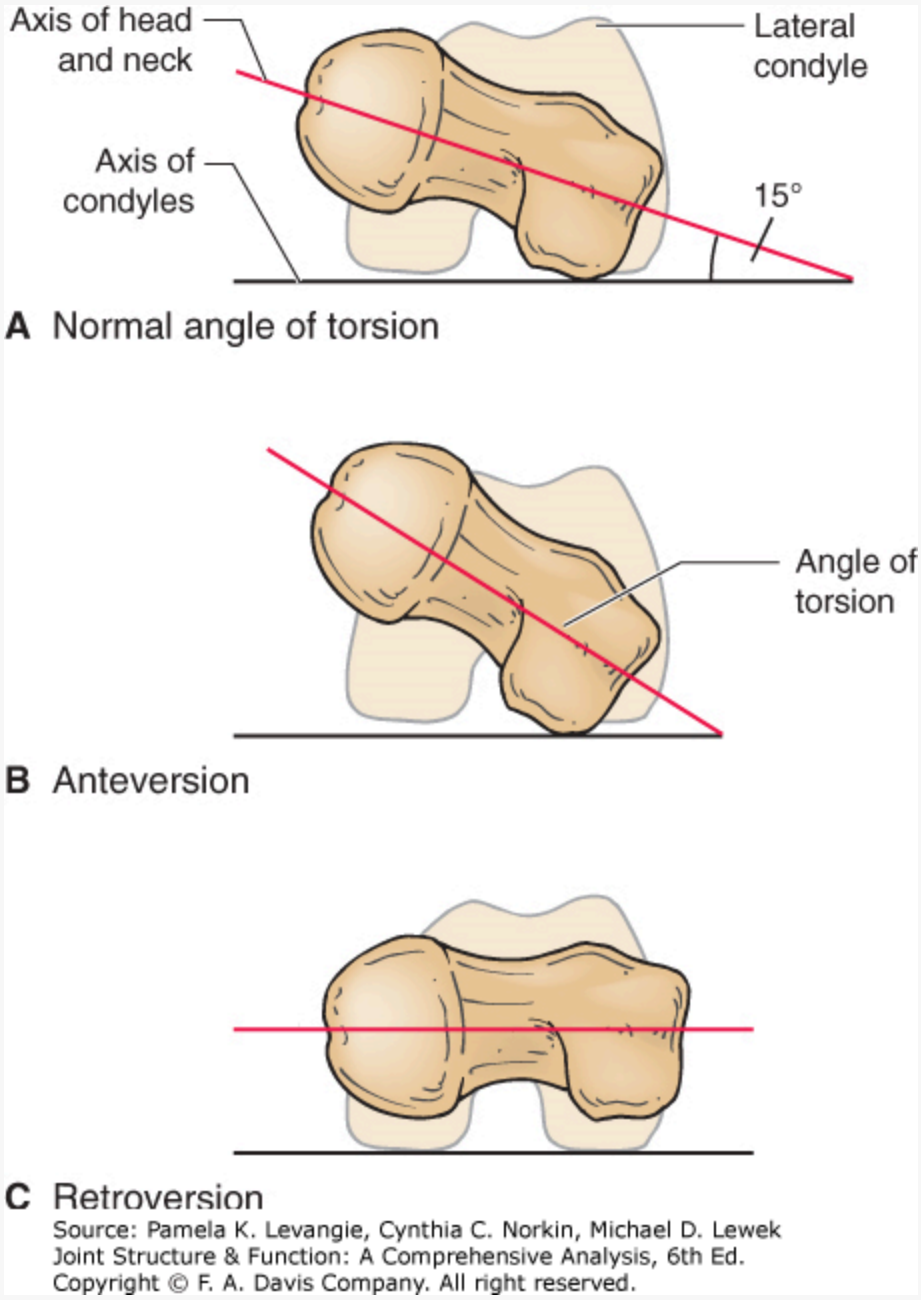
ANGLE OF TORSION OF THE FEMUR

The angle of torsion of the femur can best be viewed by looking down the length of the femur from top to bottom. An axis through the femoral head and neck in the transverse plane will lie at an angle to an axis through the femoral condyles, with the head and neck torsioned anteriorly (laterally rotated) with regard to an angle through the femoral condyles (**Fig. 10–9**). This angulation reflects the medial rotational migration of the lower limb bud that occurred during fetal development (see Fig. 10–6). The apparent contradiction between medial torsion of the embryonic limb bud and lateral torsion of the femoral head and neck simply reflects a shift in reference. In medial torsion of the limb bud, the proximal end is fixed and the distal end migrates medially. When torsion of the femur is assessed in a child or adult, the reference is an axis through the femoral condyles (the knee joint axis) that is generally presumed to lie in the frontal plane (**Fig. 10–9A**). If the axis through the femoral condyles lies in the frontal plane (as it functionally should), then the head and neck of the femur are torsioned anteriorly (laterally rotated), relatively speaking, on the femoral condyles (**Fig. 10–9B**). The angle of anterior torsion decreases with age. In the newborn, femoral neck anteversion averages 30° to 40°.[40] This angle decreases approximately 1.5° per year until skeletal maturity and is usually symmetrical in the right and left sides.[34,40,41] In the adult, the normal angle of torsion is considered to be 10° to 20°: 15° for males and 18° for females.[40,42,43]

FIGURE 10-9

Angles of torsion in a right femur. A line parallel to the posterior femoral condyles and a line through the head and neck of the femur

normally make an angle with each other that averages 10° to 20° in an adult without impairments. The femoral head and neck are in torsion anteriorly (medially) with respect to the femoral condyles. **A.** An angle of torsion within normal limits. **B.** A pathological increase in the angle of torsion is called anteversion. **C.** A pathological decrease in the normal angle of torsion is called retroversion.



Femoral anteversion is considered to exist when the angle of anterior torsion is greater than 15° to 20°. A reversal of anterior torsion, known as **femoral retroversion,** occurs when angles are less than 15° to 20° (Fig. 10–9C). There may not be one angulation at which pathological femoral torsion may be diagnosed, given the substantial normal

variability.[44] Variations in the degree of anteversion or retroversion may also be dependent on the method used to assess it, as computed tomography (CT) scan, radiograph, and ultrasound have each been used to measure the angle of femoral torsion.[44] Clinical measures of anteversion have correlated with imaging measurements. For example, femoral anteversion is associated with increased medial rotation ROM and concurrent decreased lateral rotation so that the total excursion of hip rotation motion remains the same.[45]

Although some structural deviations such as femoral anteversion and coxa valga are commonly found together, each may occur independently of the other. Each structural deviation warrants careful consideration as to the impact on hip joint function *and* function of the joints both proximal and distal to the hip joint. As shall be evident when the knee and foot are discussed in subsequent chapters, femoral anteversion is often implicated in dysfunction at both the knee and foot. The other pathological angulations of the femur (retroversion, coxa vara, and coxa valga) similarly affect the hip joint and other joints proximally and distally.

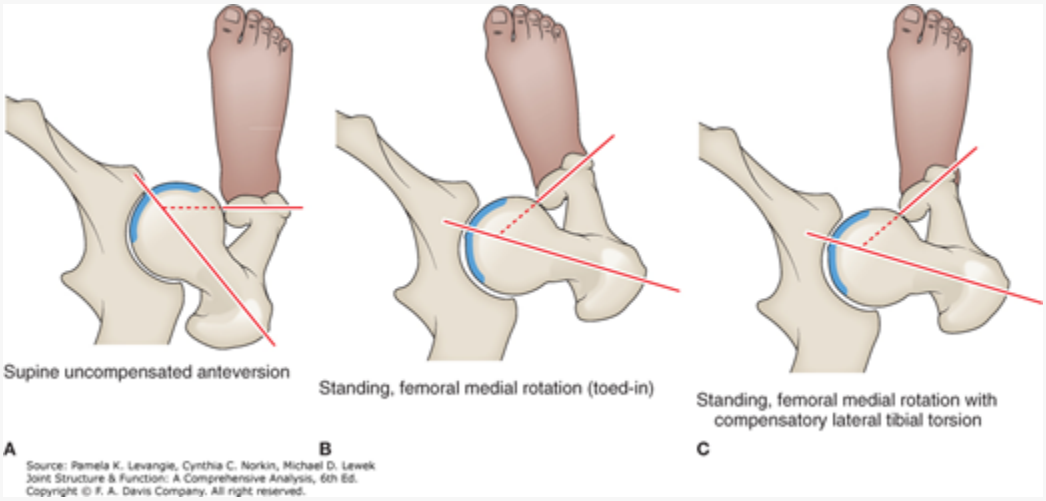
Variations in the angle of torsion also affect hip biomechanics and function (Fig. 10–10). Anteversion of the femoral head reduces hip joint stability because the femoral articular surface is more exposed anteriorly (Fig. 10–10A). The line of pull for the hip abductors may fall more posterior to the joint, reducing the moment arm for abduction.[46] As is true for coxa valga, the resulting need for additional abductor muscle force may predispose the joint to arthrosis or may produce more energy-consuming gait deviations. The effect of femoral anteversion may also be seen at the knee joint. When the femoral head is anteverted, femoroacetabular congruency is diminished. To

improve this congruency, pressure from the anterior capsuloligamentous structures and the anterior musculature may rotate the entire femur medially (Fig. 10–10B). Although this medial rotation of the femur improves the congruence in the acetabulum, the knee joint axis through the femoral condyles is now turned medially. Medial rotation of the femoral condyles alters the plane of knee flexion/extension and results, at least initially, in a toe-in gait.[47] However, the toe-in position of the foot may appear to diminish over time because it is not uncommon to see a compensatory lateral tibial torsion develop. Although the foot placement looks better, the underlying hip problem generally remains (with some developmental reduction). The abnormal position of the knee joint axis is commonly labeled **medial femoral torsion.** Medial femoral torsion and femoral anteversion are the same abnormal condition of the femur. The label designates whether the exaggerated twist in the femur is altering the mechanics at the hip joint (femoral anteversion) or at the knee joint (medial femoral torsion). Excessive femoral anteversion may be noted with increased knee adduction, hip flexion and medial rotation, and increased anterior pelvic tilt during gait.[48] As shall be seen in the next two chapters, an anteverted femur will also affect the biomechanics of the patellofemoral joint at the knee and the subtalar joint in the foot.

FIGURE 10-10

A. With the femoral condyles aligned in the frontal plane (in supine), the anteverted femoral head is exposed anteriorly. Lateral rotation will be limited, but medial rotation is relatively excessive. **B.** In standing, the anteverted femur tends to medially rotate within the acetabulum for improved joint congruency. The result is medial

rotation of the femoral condyles in relation to the plane of progression. The torsional deformity of the femur, when assessed in standing, is referred to as medial femoral torsion. **C.** If there is an accompanying lateral tibial torsion, the expected toe-in may be minimized or reversed.



Femoral retroversion is the opposite of anteversion and may create problems opposite those of femoral anteversion. Studies have shown patients with symptomatic labral tears have significantly reduced femoral anteversion compared with healthy controls.[49,50] This has led some to believe that femoral anteversion serves as a protective measure against impingement and subsequent labral pathology. However, recent research has shown larger labral tears are associated with greater femoral anteversion.[51] Thus, excessive femoral anteversion or retroversion compared with "normal" femoral torsion may be a factor in progressive hip joint disease.

Expanded Concepts 10–2
Femoral Rotation and Effect on Gait

Individuals with excessive femoral anteversion will often medially rotate the femur to center the femoral head in the acetabulum. By maintaining this medially rotated hip position, the medial rotators may become shortened, ultimately limiting the amount of lateral rotation available for passive ROM. Likewise, it is possible that this position leads to stretch weakness of the hip lateral rotators. Individuals who maintain a medially rotated position at the hip because of the presence of excessive anteversion can also present with "toeing-in" type gait. In a similar fashion, individuals with decreased femoral anteversion, or relative femoral retroversion, will often present with "toeing-out" type deviation as the femur laterally rotates to center the femoral head in the acetabulum.

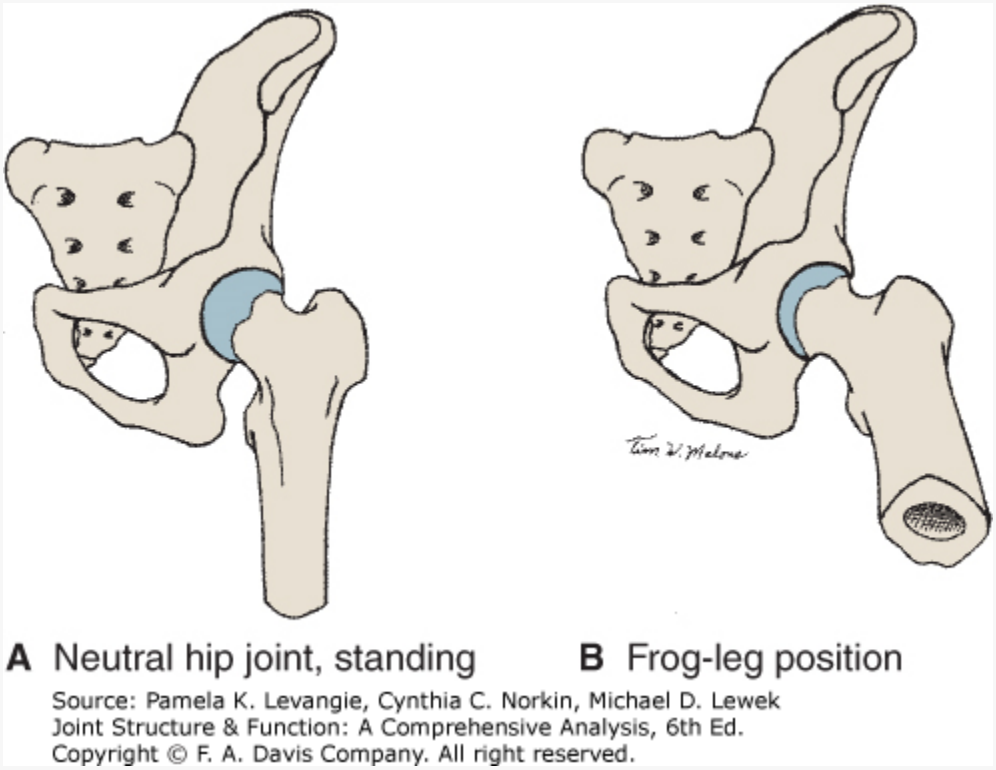
Articular Congruence

The shape of the hip joint helps to decrease the peak contact stress.[2] The hip joint is considered to be a highly congruent joint. However, there is substantially more articular surface on the head of the femur than there is on the acetabulum (Fig. 10–11). In the neutral or standing position, the articular surface of the femoral head remains exposed anteriorly and somewhat superiorly (Fig. 10–11A). The acetabulum does not fully cover the head superiorly, and the anterior torsion of the femoral neck exposes a substantial amount of the femoral head's anterior articular surface. Articular contact between the femur and the acetabulum can be increased in the normal non-weightbearing hip joint by a combination of flexion, abduction, and slight lateral rotation (Fig. 10–11B).[52] This position (also known as the frog-leg position) corresponds to that assumed by the hip joint in a quadruped position and is the true physiological position of the hip joint.[52]

FIGURE 10-11

In the neutral hip joint' articular cartilage from the head of the femur is exposed anteriorly and' to a lesser extent' superiorly. Maximum

articular contact of the head of the femur with the acetabulum is obtained when the femur is flexed' abducted' and slightly rotated laterally.



A Neutral hip joint, standing **B** Frog-leg position

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Congruency of the hip joint is greater in weightbearing and is less when in non-weightbearing positions.[13] In weight-bearing, the elastic deformation of the acetabulum increases contact with the femoral head, with primary contact at the anterior, superior, and posterior articular surfaces of the acetabulum.[13] An additional contribution to articular congruence and coaptation (joining together) of joint surfaces may be made by the non-articular and non-weightbearing acetabular fossa. The acetabular fossa may be important in setting up a partial vacuum in the joint so that atmospheric pressure contributes to stability by helping maintain contact between the femoral head and the acetabulum. Atmospheric pressure in hip flexion activities has

shown to have a greater role in stabilization of the hip joint than capsuloligamentous structures.[53] Intra-articular pressure keeps the femoral head and acetabulum together even after the joint capsule has been opened. The pressure within the joint must be broken before the hip can be dislocated.[52] The labrum enhances joint stability by both deepening the acetabulum and by acting as a seal to maintain negative intra-articular pressure.[54] When a labral tear is present, the labrum's ability to act as a buttress to prevent excessive movement and to act as a seal to maintain negative intra-articular pressure may be compromised, placing increased stress on the surrounding joint capsule.[54,55]



Patient Application 10–3

Gabriella's history may be consistent with her history of hip dysplasia. Current radiographs and magnetic resonance imaging (MRI) showed a valgus, anteverted femur and a shallow acetabulum of her left hip. Gabriella's structural deviations of femoral anteversion and coxa valga and a shallow acetabulum (decreased center edge angle) can result in increased articular exposure of the femoral head, less congruence, and reduced stability of the hip joint in the neutral weightbearing position.

If Gabriella's hip dysplasia had been diagnosed in infancy, frog-leg positioning might have been maintained using a Frejka pillow or Pavlik harness (Fig. 10–12).[56] These devices may decrease the deformities by increasing the contact between the femoral head and acetabulum. The position of combined flexion, abduction, and rotation is commonly used for immobilization of the hip joint when the goal is to improve articular contact and joint congruence in developmental dysplasia of the hip joint.[56] Whether this was done or not, Gabriella's deformities of the femur and acetabulum persisted. Considering the reduction in center edge angle alone, the stress distribution within the joint is now concentrated in a smaller weightbearing area throughout the gait cycle, compared with a normal center edge angle.[57] This increase in stress is likely to lead to degenerative changes over time. The center edge angle in addition to age and the presence of a labral tear has been associated with

osteoarthritis.[58] It appears that Gabriella's history of hip dysplasia may predispose her to progressive intra-articular joint pathology and subsequent osteoarthritis of the hip.

FIGURE 10-12

An infant can easily be maintained at the hip joint position of flexion, abduction, and external rotation (frog-leg position) by using a positioning device.



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Hip Joint Capsule and Ligaments

HIP JOINT CAPSULE

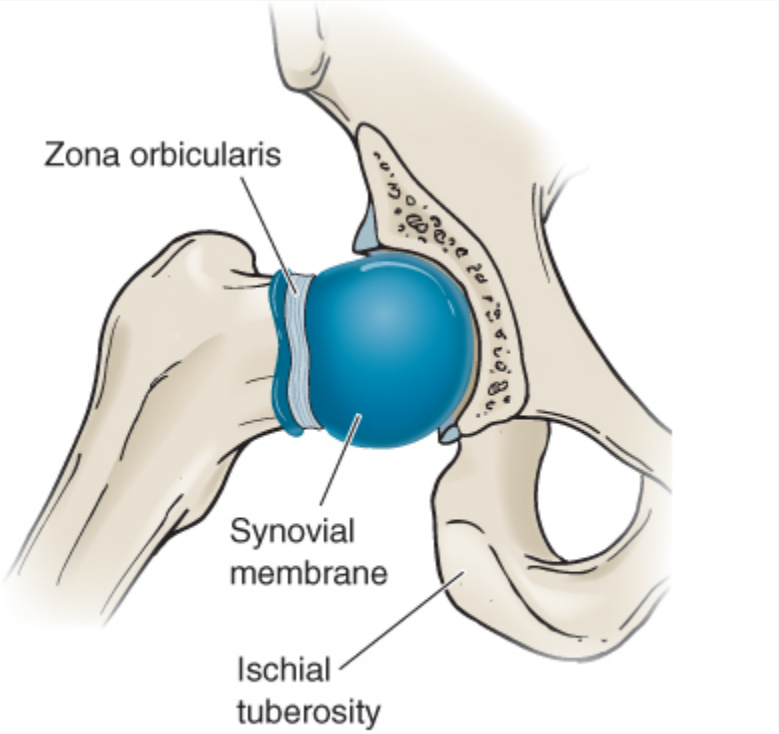
Unlike the relatively weak articular capsule of the shoulder, the hip joint capsule is a substantial contributor to joint stability. The articular capsule of the hip joint is an irregular, dense fibrous structure with longitudinal and oblique fibers and three thickened regions that constitute the capsular ligaments.[59,60] The more extensive

longitudinal fibers of the capsule are attached to the periosteum proximal to the acetabular rim and acetabular labrum.[60] The oblique fibers are aligned near the proximal attachment in a circumferential manner just distal to the femoral head, forming a collar-like structure around the femoral neck.[26,59,60] This area is known as the **zona orbicularis.** These collar-like fibers help to prevent distraction of the femoral head from the acetabulum (Fig 10–13).[61] The capsule is thickened anterosuperiorly, where there is little bony support and the predominant stresses occur. Posteroinferiorly, the capsule is relatively thin and redundant, with some areas of the capsule thin enough to be nearly translucent.[14,62] The capsule covers the femoral head and neck like a cylindrical sleeve and attaches to the base of the femoral neck. The femoral neck is intracapsular, whereas both the greater and lesser trochanters are extracapsular. The synovial membrane lines the inside of the capsule. Anteriorly, there are longitudinal retinacular fibers deep in the capsule that travel along the neck toward the femoral head.[14] The retinacular fibers carry blood vessels that are the major source of nutrition to the femoral head and neck. The retinacular blood vessels arise from a vascular ring located at the base of the neck formed by the medial and lateral circumflex arteries (branches of the deep femoral artery). The superior and inferior gluteal arteries supply more proximal and posterior portions of the joint capsule, whereas the medial and lateral circumflex arteries supply the more distal and anterior aspect of the joint capsule.[63] The femoral head is most commonly supplied through the medial femoral circumflex artery.[63]

FIGURE 10-13

The zona orbicularis is composed of collar-like fibers that encircle the

femoral neck and help to prevent distraction of the femoral head from the acetabulum.



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As with the other joints already described, there are numerous bursae associated with the hip joint. Although as many as 20 bursae have been described, there are commonly recognized to be three primary or important bursae.[64,65] Because the bursae are more strongly associated with the hip joint muscles rather than its capsule, the bursae will be described with their corresponding musculature.

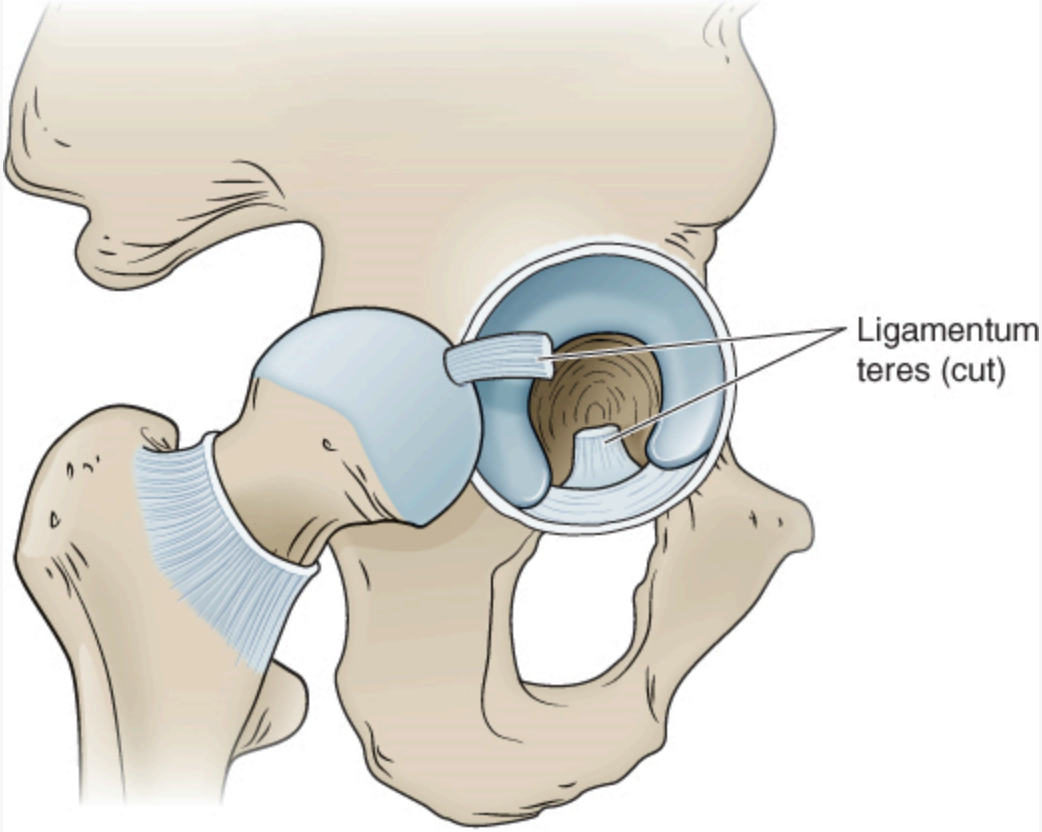
HIP JOINT LIGAMENTS

The ligament of the head of the femur, or the ligamentum teres, is an intra-articular but extrasynovial accessory joint structure. The ligament is a triangular band that attaches to the peripheral edge of the acetabular notch. The ligament then passes under the

transverse acetabular ligament (with which it blends) to attach at its other end to the fovea of the femur (**Fig. 10–14**). The ligamentum teres is encased in a flattened sleeve of synovial membrane so that it does not communicate with the synovial cavity of the joint. The material properties of the ligament of the head are similar to those of other ligaments.[66] Traditionally, it has been believed that the sole purpose of the ligamentum teres was to serve as a conduit for blood vessels to the femoral head and that it did not play a significant role in joint stabilization.[67] However, recent information suggests the ligamentum teres has a role in stabilizing the hip.[68–72] Specifically, the ligamentum teres has been shown to provide restraint of hip medial and lateral rotation when the hip is positioned greater than 90° of flexion.[70–72] Individuals with acquired anterior hip instability and abnormally increased medial/lateral rotation of the hip joint may put excessive strain on the ligamentum teres that may compromise the integrity of the ligament. Once the ligament becomes compromised or lax, it may become pinched between the femoral head and acetabulum, which may result in painful clicking and catching within the joint. As a result, individuals with ligamentum teres deficiency may have apprehension going into a deep squat position. A lesion of the ligamentum teres was found to be the third most common finding in athletes undergoing arthroscopic surgery, occurring in 18% of individuals.[73]

FIGURE 10-14

The ligamentum teres is an intra-articular but extrasynovial accessory joint structure that extends from the peripheral edge of the acetabular notch to the femoral fovea. It is also called ligament of the head of the femur.



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Expanded Concepts 10–3

Blood Supply to the Femoral Head

The importance of the secondary blood supply carried by the ligamentum teres will vary across the life span, with a greater contribution to be made in childhood. While a child is still growing, the primary retinacular vessels (the medial and lateral circumflex arteries) cannot travel through the avascular cartilaginous epiphysis but must travel across the surface, where the vessels are more vulnerable to disruption. Crock proposed that the femoral head was supplied predominantly by the blood vessels of the ligamentum teres until bony maturation and epiphyseal closure.[74] However, Tan and Wong found the ligament was absent in 10% of their examined specimens.[75] The vessels of the ligament of the head are commonly sclerosed in elderly persons.[76] Therefore, the secondary blood supply cannot be counted on to back up the primary retinacular supply when it is disrupted by such problems as

femoral neck fracture.[75] The absence of a secondary blood supply to the head increases the risk of avascular necrosis of the femoral head with femoral neck trauma.

Expanded Concepts 10–4

Legg-Calvé-Perthes Disease

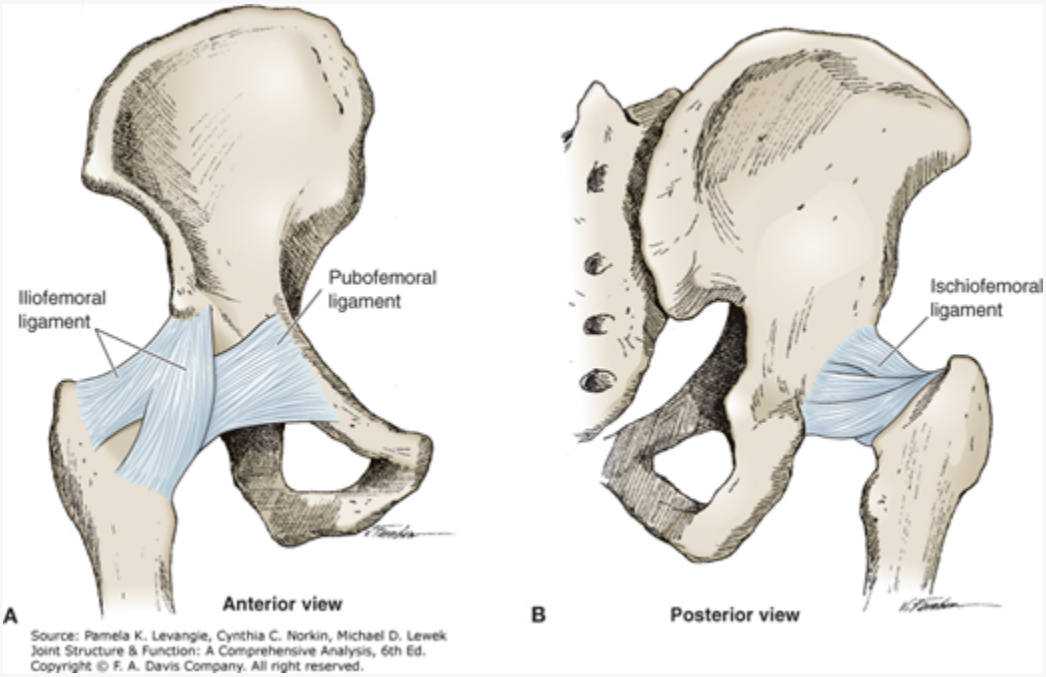
Legg-Calvé-Perthes disease is noted by collapse of the femoral head, resulting in flattening of the head from loss of blood supply. This poorly understood childhood disorder is usually a self-limiting condition with a variable natural history. Impaired growth and abnormal development can result in deformity, joint incongruity, and the early onset of osteoarthritis over the long term if not properly treated.[77,78]

The hip joint capsule is typically considered to have three reinforcing capsular ligaments: two anteriorly and one posteriorly (Fig. 10–15).[52,59,67] The two anterior ligaments are the *iliofemoral ligament* and the *pubofemoral ligament* (Fig. 10–15A). The **iliofemoral ligament** is fan-shaped and resembles an inverted letter *Y* and is often referred to as the **Y ligament of Bigelow.** The apex of the ligament is attached to the anterior inferior iliac spine, and the two arms of the Y fan out to attach along the intertrochanteric line of the femur. The superior band of the iliofemoral ligament is the strongest and thickest of the hip joint ligaments.[52,59,79] The **pubofemoral ligament** is also anteriorly located, arising from the pubic portion of the acetabular rim inferior and medial to the iliopectineal eminence. The pubofemoral ligament forms a sling that supports the inferior aspect of the femoral neck and connects fibers of the iliofemoral and ischiofemoral ligaments.[59] The **ischiofemoral ligament** is the posterior capsular ligament (Fig. 10–15B) and attaches to the posterior surface of the acetabular rim and the acetabular labrum. Some of its fibers spiral around the femoral neck and blend with the fibers of the circumferential fibers of the capsule. Other fibers are arranged

horizontally and attach to the inner surface of the greater trochanter.

FIGURE 10-15

A. Anterior view of the right hip joint shows the two bands of the iliofemoral (Y) ligament and the more inferiorly located pubofemoral ligament. **B.** Posterior view of a right hip joint shows that the spiral fibers of the ischiofemoral ligament are tightened during hyperextension and therefore limit hyperextension.



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There is at the hip joint, as at other joints, some conflicting evidence as to the roles of the joint ligaments. There is consensus, however, that each of the hip joint motions will be restrained by at least one portion of one of the hip joint ligaments.[67] The forces transmitted by the ligaments (and capsule) are dependent on orientation of the femur in relation to the acetabulum.[59,62] The anterior capsuloligamentous structures tend to be stronger (stiffer and withstanding greater force at failure) than the posterior

structures.[79] The primary stabilizing component of the anterior hip joint is the iliofemoral ligament. This ligament is capable of providing resistance to excessive lateral rotation.[59] In particular, the more lateral portion of the iliofemoral ligament is the most significant restraint to lateral rotation when the hip is in neutral position and flexed.[59] The pubofemoral ligament controls lateral rotation in an extended position.[59] The posteriorly located ischiofemoral ligament is the primary restraint to medial rotation of the hip regardless of hip position in flexion or extension.

The capsule and ligaments permit little or no joint distraction even under strong traction forces. When a dysplastic hip is completely dislocated, the capsule and ligaments are strong enough to support the femoral head in weightbearing. In these unusual conditions, the stresses on the capsule imposed by the femoral head may lead to impregnation of the capsule with cartilage cells that contribute to a sliding surface for the head.[80]

Under normal circumstances, the hip joint, its capsule, and ligaments routinely support two-thirds of the body weight (the weight of the head, arms, and trunk). In bilateral stance, the hip joint is typically in neutral position or slight extension. In this position, the capsule and ligaments are under some tension.[52] The normal line of gravity in bilateral stance falls posterior to the hip joint axis, creating an external extension moment. At end-range extension, additional passive tension is created in the capsuloligamentous complex that is sufficient to offset the gravitation extension moment. As long as the line of gravity falls posterior to the hip joint axis, the passive capsuloligamentous structures can typically contribute to the support of the superimposed body weight in symmetrical bilateral stance with minimal active

assistance from the muscles crossing the hip.

CAPSULOLIGAMENTOUS TENSION

Hip joint extension, with slight abduction and medial rotation, is the close-packed position for the hip joint.[14] With increased extension, the ligaments twist around the femoral head and neck, drawing the head into the acetabulum. In contrast to most other joints in the body, the close-packed and most stable position for the hip joint is not the position of optimal articular contact (congruence). As already noted, optimal articular contact occurs with combined flexion, abduction, and lateral rotation. Under circumstances in which the joint surfaces are neither maximally congruent nor close packed, the hip joint is at greatest risk for traumatic dislocation. A position of particular vulnerability occurs when the hip joint is flexed and adducted (as it is when sitting with the thighs crossed). In this position, a strong axial force through the femoral shaft toward the hip joint (as when the knee hits the dashboard in a car accident) may push the femoral head out of the acetabulum (Fig. 10–16).[52] Macrotraumatic injuries associated with high impact are known to cause capsular laxity. Recent information suggests microtrauma associated with sports that involve repeated and forceful end-range movements may also be a cause of laxity.[81]

FIGURE 10-16

With the hip positioned in flexion, a large force applied along the shaft of the femur (as in a dashboard injury) may cause a posterior hip dislocation.



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The capsuloligamentous tension at the hip joint is least when the hip is in mid-range flexion, slight abduction, and mid-rotation. In this position, the normal intra-articular pressure is minimized, and the capacity of the synovial capsule to accommodate abnormal amounts of fluid is greatest.[53] This is the position assumed by the hip when there is pain arising from capsuloligamentous problems or from excessive intra-articular pressure caused by extra fluid (blood or synovial fluid) in the joint, which may be a result of such conditions as **synovitis** of the hip joint or bleeding in the joint from tearing of blood vessels with a femoral neck fracture. Minimizing intra-articular

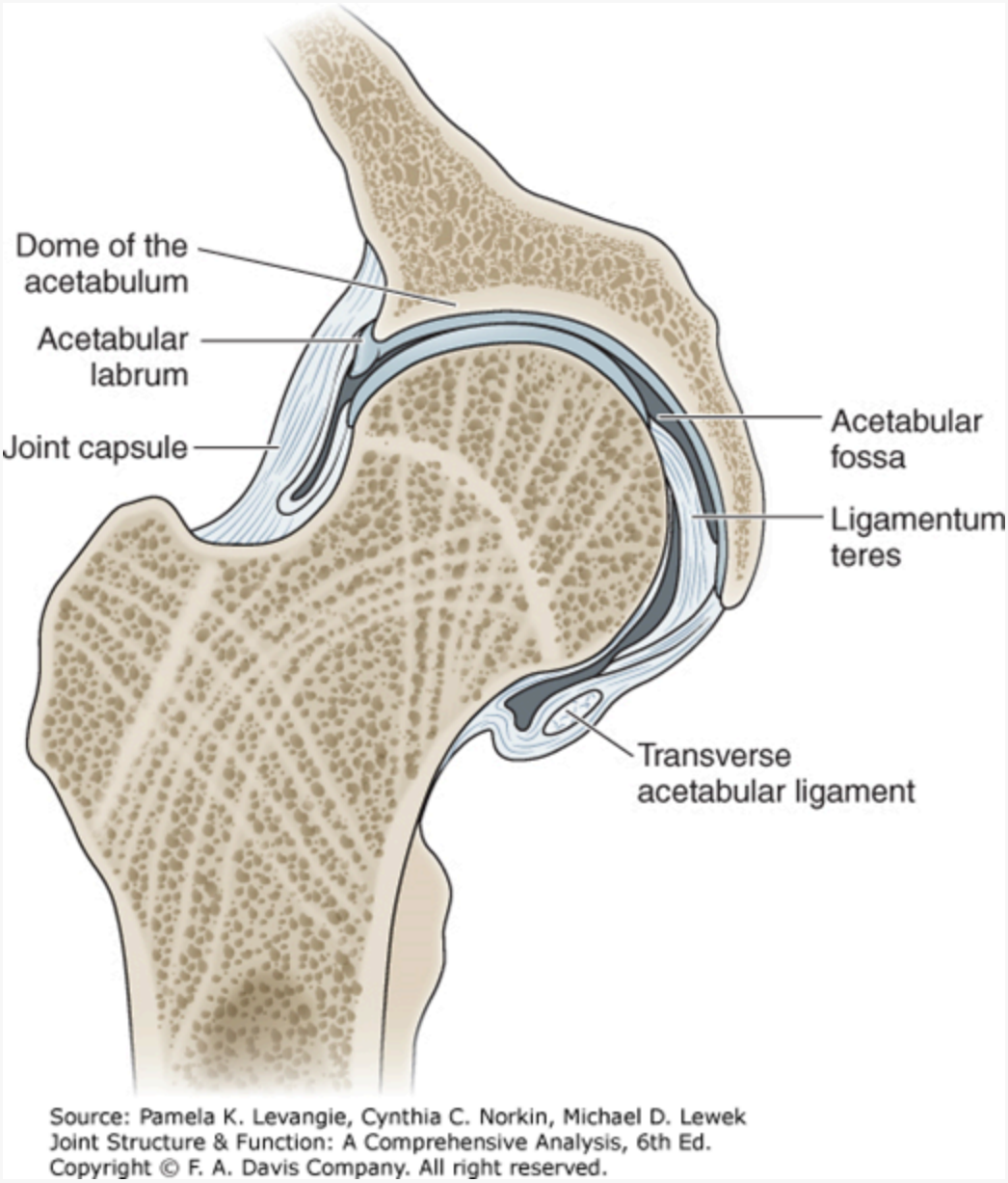
pressure not only decreases pain in the joint but also prevents compression of the intra-articular blood vessels that supply the femoral head.[53]

Structural Adaptations to Weightbearing

The internal architecture of the pelvis and femur reveals the remarkable interaction between mechanical stresses and structural adaptation created by the transmission of forces between the femur and the pelvis. The trabeculae (calcified plates of tissue within the cancellous bone) line up along lines of stress and form systems that normally adapt to stress requirements. The trabeculae are quite evident on bony cross-section, as seen in Figure 10–17, along with some of the other structural elements of the hip joint.

FIGURE 10-17

The trabeculae of the femur line up along lines of stress in the cancellous bone and can be seen on cross-section.



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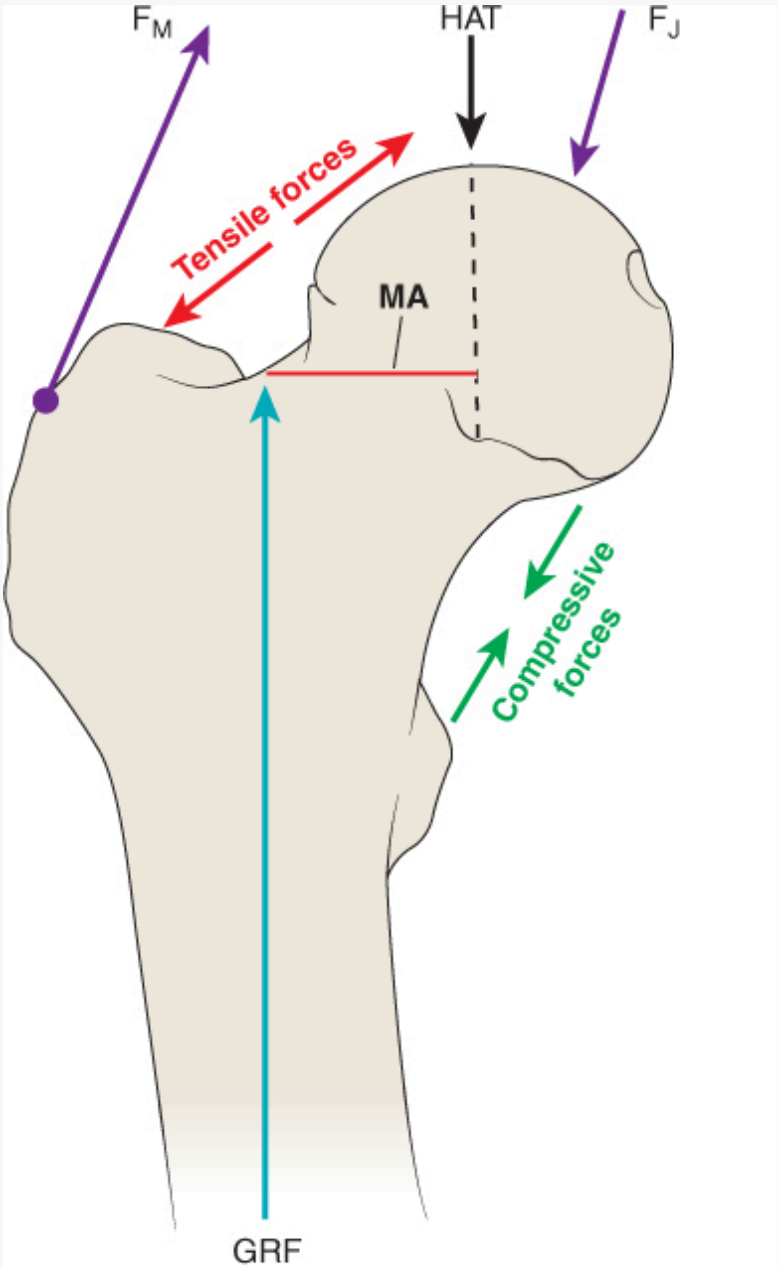
In Chapter 4, we followed the line of weightbearing through the vertebrae of the spinal column to the sacral promontory and on through the sacroiliac joints. Most of the weightbearing stresses in the pelvis pass from the sacroiliac joints to the acetabulum.[14] In standing or upright weightbearing activities, at least half the weight of the HAT (the gravitational force) passes down through the pelvis to each femoral head. Because this

compressive force passes medially to the femoral neck, a moment arm exists that creates a bending moment across the femoral neck (Fig. 10–18). This moment arm is the perpendicular distance between the superimposed body weight on the femoral head (a downwardly directed force) and the intertrochanteric area.[82] As a result of this bending moment the femoral neck is subjected to a tensile force on the superior aspect of the femoral neck and a compressive force on the inferior aspect. Bone naturally responds better to compressive forces than tensile forces. Fractures occur in the area where the bone cannot sufficiently handle the applied load. If the bone cannot handle the bending moment across the femoral neck due to the presence of a large tensile load across the superior portion of the femoral neck, then a fracture will occur. The body is able to minimize this tensile load by the incorporation of muscle activity across the hip (Fig 10–18), which can create large compressive forces across the femoral neck to offset the imposed tensile force due to the weight of the HAT. In addition, a complex set of forces within the femoral neck prevents the rotation of the neck on the shaft; among these forces are the structural resistance of two major and three minor **trabecular systems** (Fig. 10–19).

FIGURE 10-18

The weightbearing line of the head, arms, and trunk (HAT) loads the head of the femur, whereas the ground reaction force (GRF) comes up the shaft of the femur, resulting in a force couple that creates a bending moment, with a moment arm (MA) that is dependent on the length and angle of the neck of the femur. The bending moment creates tensile stress on the superior aspect of the femoral neck and

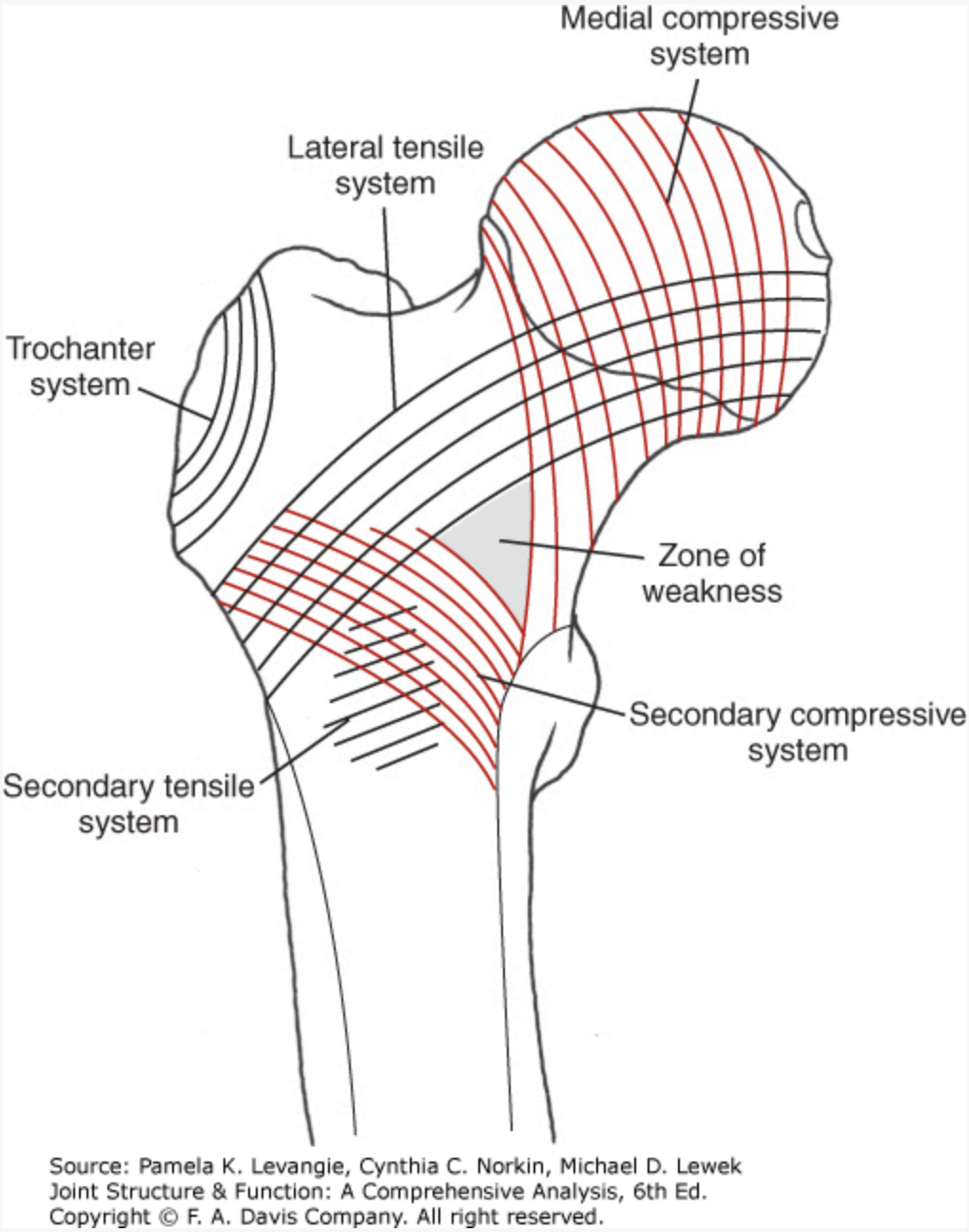
compressive stress on the inferior aspect.



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FIGURE 10-19

Two major (the medial compressive and lateral tensile) trabecular systems show the primary transmission of forces. Additional lines of stress are evident at the secondary compressive and tensile systems and at the trochanteric system. A distinct zone of weakness can be observed in which the trabeculae are thin and do not cross each other.



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The medial (or principal compressive) trabecular system arises from the medial cortex of the upper femoral shaft and radiates through the cancellous bone to the cortical bone of the superior aspect of the femoral head. The medial system of trabeculae is oriented along the vertical compressive forces passing through the hip joint.[52] The lateral (or principal tensile) trabecular system of the femur arises from the lateral cortex

of the upper femoral shaft and, after crossing the medial system, terminates in the cortical bone on the inferior aspect of the head of the femur. The lateral trabecular system is oblique and may develop in response to parallel (shear) forces of the weight of HAT and the ground reaction force.[52] There are two accessory (or secondary) trabecular systems, of which one is considered compressive and the other is considered tensile.[83] Another secondary trabecular system is confined to the trochanteric area of the femur.[52,83] The loading environment in the femur during physical activity is mostly compressive in nature, with relatively small amounts of shear forces.[84]

The areas in which the trabecular systems cross each other at right angles are areas that offer the greatest resistance to stress and strain. There is an area in the femoral neck in which the trabeculae are relatively thin and do not cross each other. This **zone of weakness** (see Fig. 10–19) has less reinforcement and thus more potential for failure. The zone of weakness of the femoral neck is particularly susceptible to the bending forces across the area and can fracture either when forces are excessive or when compromised bony composition reduces the tissue's ability to resist typical forces.



Expanded Concepts 10–5

Femoral Neck Stresses

Although the zone of weakness in the cancellous bone has received a great deal of attention as a factor in hip fracture, Crabtree and colleagues used data from patients and cadavers with a hip fracture to conclude that the cortical bone in the femoral neck supports at least 50% of the load placed on the proximal femur.[85] They suggested that compromise of cortical bone may be more of a factor in fracture than diminished cancellous bone. A more detailed description of the problems of hip fracture will be presented later in the chapter.

The primary weightbearing surface of the acetabulum, or **dome** of the acetabulum, is located on the superior portion of the lunate surface (see Fig. 10–17).[82,86] In the normal hip, the dome lies directly over the center of rotation of the femoral head.[86] Peak contact pressures during unilateral stance are located near the superior aspect of the dome.[26] Stress distribution can be altered with a lack of femoral head coverage by the acetabulum (decreased center edge angle) as well as excessive acetabular anteversion.[2,26] Contact area was found to be significantly smaller in women than in men with associated higher peak contact stress.[26] The dome shows the greatest prevalence of degenerative changes in the acetabulum, which corresponds to the area of greatest stress. The primary weightbearing area of the femoral head is, correspondingly, its superior portion.[82,87] Degenerative changes of the femoral head include loss of the regular "ball" shape, with flattening of the superior portion. Degenerative changes are also consistently noted near the attachment of the ligamentum teres.[25]

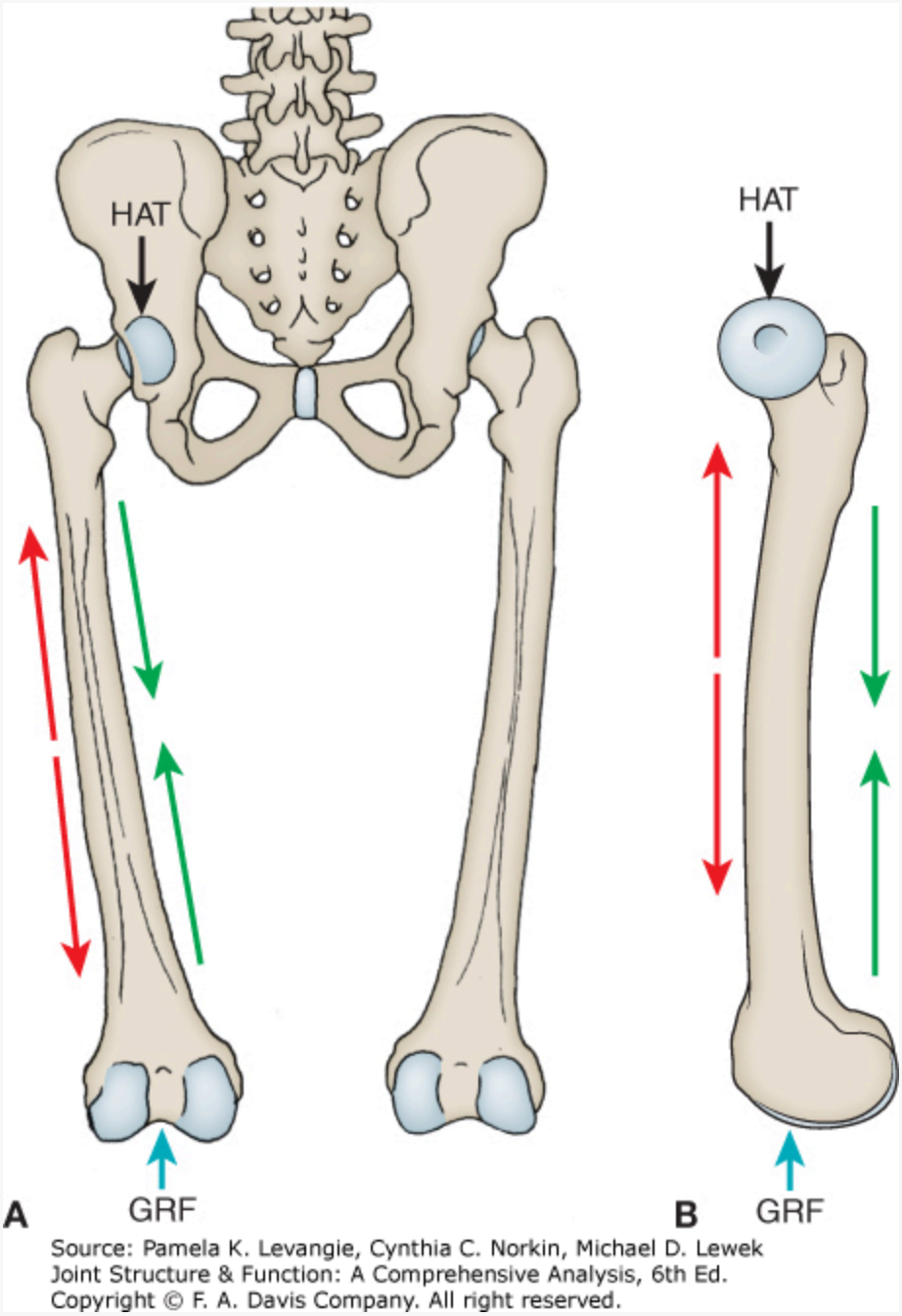
Full loading of the hip joint is presumably necessary to achieve congruence and optimize load distribution between the larger femoral head and the acetabulum.[13] Incongruence of the acetabular dome in the moderately loaded hip (especially in young adults) could result in incomplete compression of the dome cartilage and, therefore, inadequate fluid exchange to maintain cartilage nutrition.[82] The superior femoral head receives compression from the dome in standing, from the posterior acetabulum in sitting, and the anterior acetabulum when the hip is in extension. More frequent and complete compression of the cartilage of the superior femoral head, according to this premise, leads to better nutrition within the cartilage. It must be remembered, however, that avascular articular cartilage is dependent on both *compression* and

release to move nutrients through the tissue; too little compression and excessive compression (magnitude and frequency of loading) can limit nutrient exchange that may lead to compromise of the cartilage structure.

The forces of HAT and the ground reaction force that act on the articular surfaces of the hip joint and on the femoral head and neck also act on the femoral shaft. The shaft of the femur is not vertical but lies at an angle that varies considerably among individuals. However, the relatively vertical loading on the oblique femur results in bending stresses in the shaft.[52] The medial cortical bone in the shaft (diaphysis) therefore must resist compressive stresses, whereas the lateral cortical bone of the shaft must resist tensile stresses (Fig. 10–20).

FIGURE 10-20

The weightbearing line (HAT) from the center of rotation of the femoral head and the ground reaction force (GRF) cause a bending force on the shaft of the femur. **A.** In the frontal plane this results in compressive forces medially and tensile forces laterally. **B.** In the sagittal plane, the compressive forces are seen posteriorly and the tensile forces are anterior.



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Function

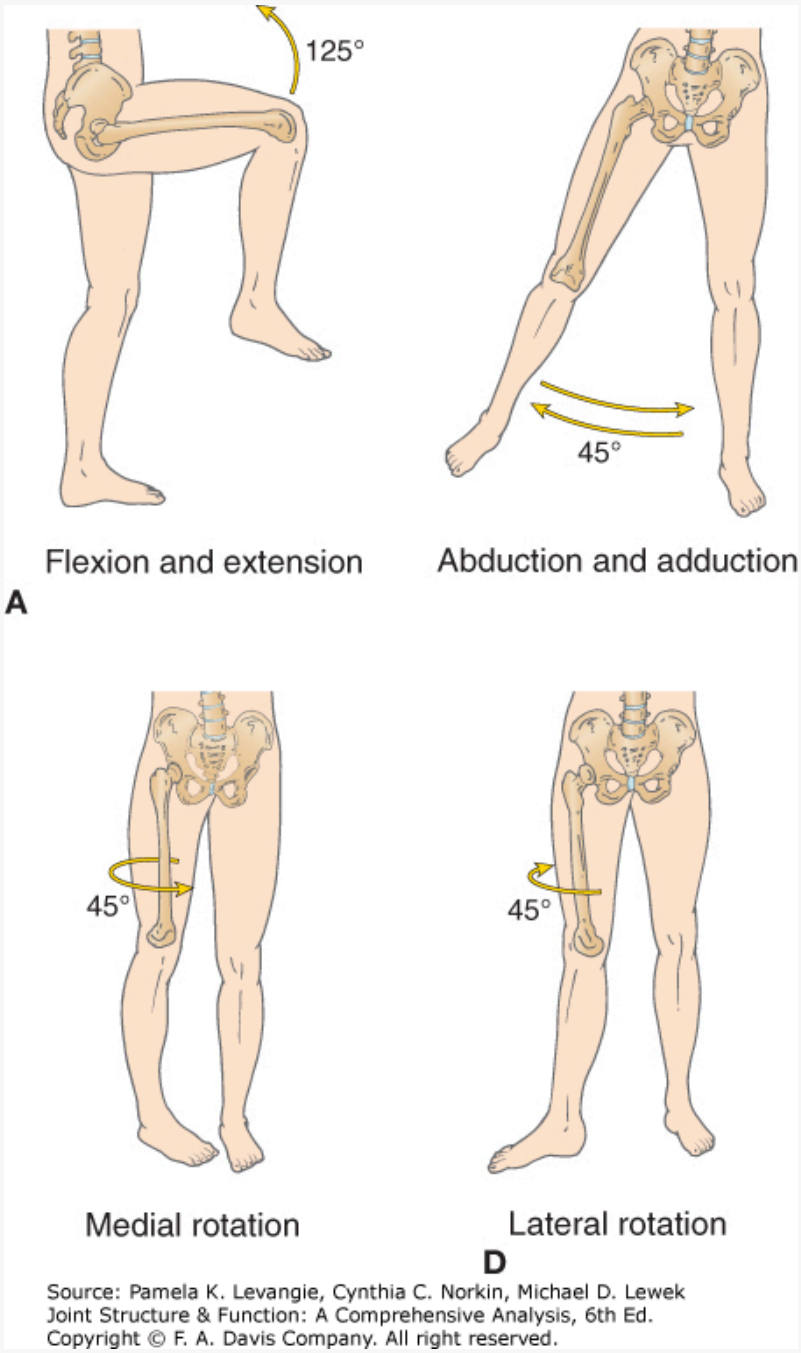
Motion of the Femur on the Acetabulum

The motions of the hip joint are easiest to visualize as movement of the convex femoral head within the concavity of the acetabulum as the femur moves through its three

degrees of freedom: flexion/extension, abduction/adduction, and medial/lateral rotation (Fig. 10–21). The femoral head will glide within the acetabulum in a direction opposite to the motion of the distal end of the femur. Flexion and extension of the femur occur from a neutral position as an almost pure spin of the femoral head around a coronal axis through the head and neck of the femur. The head spins posteriorly in flexion and anteriorly in extension. However, flexion and extension from other positions (e.g., in abduction or medial rotation) must include both spinning and gliding of the articular surfaces, depending on the combination of motions. The motions of abduction/adduction and medial/lateral rotation must include both spinning and gliding of the femoral head within the acetabulum, but the intra-articular motion again occurs in a direction opposite to motion of the distal end of the femur.

FIGURE 10-21

Hip kinematics depicted as the convex femur moving on the concave acetabulum **A.** Flexion and extension. **B.** Abduction and adduction. **C.** Medial rotation. **D.** Lateral rotation.



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As is true at most joints, the ROM of the hip joint is influenced by structural elements, as well as by whether the motion is performed actively or passively and whether passive tension in two-joint muscles is encountered or avoided. The following ranges of passive joint motion are typical of the hip joint.[88] Flexion of the hip is generally about 90° with

the knee extended, and 120° when the knee is flexed so that passive tension in the two-joint **hamstrings** muscle group is released. Hip extension is considered to have a range of 10° to 30°. Hip extension ROM appears to diminish somewhat with age, whereas flexion remains relatively unchanged.[89] When hip extension is combined with knee flexion, passive tension in the two-joint **rectus femoris** muscle may limit the movement. The femur can be abducted 45° to 50° and adducted 20° to 30°. Abduction can be limited by the two-joint **gracilis** muscle and adduction limited by the **tensor fascia lata (TFL)** muscle and the associated **iliotibial (IT) band.** Medial and lateral rotation with the hip joint in 90° of flexion is typically 42° to 50°.

Normal gait on level ground requires at least the following hip joint ranges: 30° flexion, 10° extension, 5° of both abduction and adduction, and 5° of both medial and lateral rotation.[53,84] Walking on uneven terrain or stairs will increase the need for joint range beyond that required for level ground, as will activities such as sitting in a chair or sitting cross-legged.



Patient Application 10–3

Gabriella's passive ROM findings and asymmetries in her gait correspond to femoral anteversion on the left. In the supine position on the examination table (hip extended), she has more hip joint medial than lateral rotation (although medial rotation with hip flexion is limited by pain). In her case, her shallow acetabulum increases the tendency for joint subluxation with lateral rotation of the hip. This would partially explain her complaints that her hip feels like it is "popping out of socket." When she walks, Gabriella's left foot is toed-in more than her right because her involved side is in greater hip medial rotation compared with her uninvolved side. This may be the body's way of maximizing congruence of the anteverted femur on the weightbearing joint, or it may be to minimize stretch on the proprioceptor-rich anterior capsule. The consequence for increased hip joint congruence is that the

torsional deformity now appears as medial femoral torsion, resulting in a deviant positioning of the patella and femoral condyles.[90] Gabriella's foot would be more toed-in than she demonstrates, but it appears that Gabriella, like many persons with femoral anteversion (medial femoral torsion), has also developed an excessive lateral tibial torsion. It is evident in Gabriella's case that altered biomechanical alignment of the hips relates directly to changes throughout the entire kinetic chain, specifically the knee, foot, and ankle.

Motion of the Pelvis on the Femur

Whenever the hip joint is weightbearing, the femur is relatively fixed, and motion is produced by movement of the pelvis on the femur. At all joints, the motion between articular surfaces is the same whether the distal or proximal lever moves. However, the proximal and distal levers move in opposite directions to produce the same articular motion. For example, elbow flexion can be a rotation of the distal forearm upward or, conversely, a rotation of the proximal humerus downward. In examinations of the upper extremity joint complexes thus far, motion of the distal lever tended to predominate functionally, and so this apparent reversal of motions was not a point of discussion. At the hip joint, this reversal of motion of the lever is further complicated by the horizontal orientation and shape of the pelvis (the "levers" of the hip are not in line but lie essentially perpendicular to each other). In contrast to other joints, there is also a new set of terms to identify joint motion when the pelvis (rather than femur) is the moving segment. The terms for pelvic motions are used with weightbearing hip motion because the motions of the pelvis are more apparent to the eye of the examiner and are, in fact, key to what occurs at the joints above and below the pelvis. It must be emphasized, however, that the motions of the pelvis presented in the next sections are *not new motions of the hip joint* but are simply how the same three degrees of freedom for the hip joint are accomplished by movement of the pelvis on the femur, rather than by

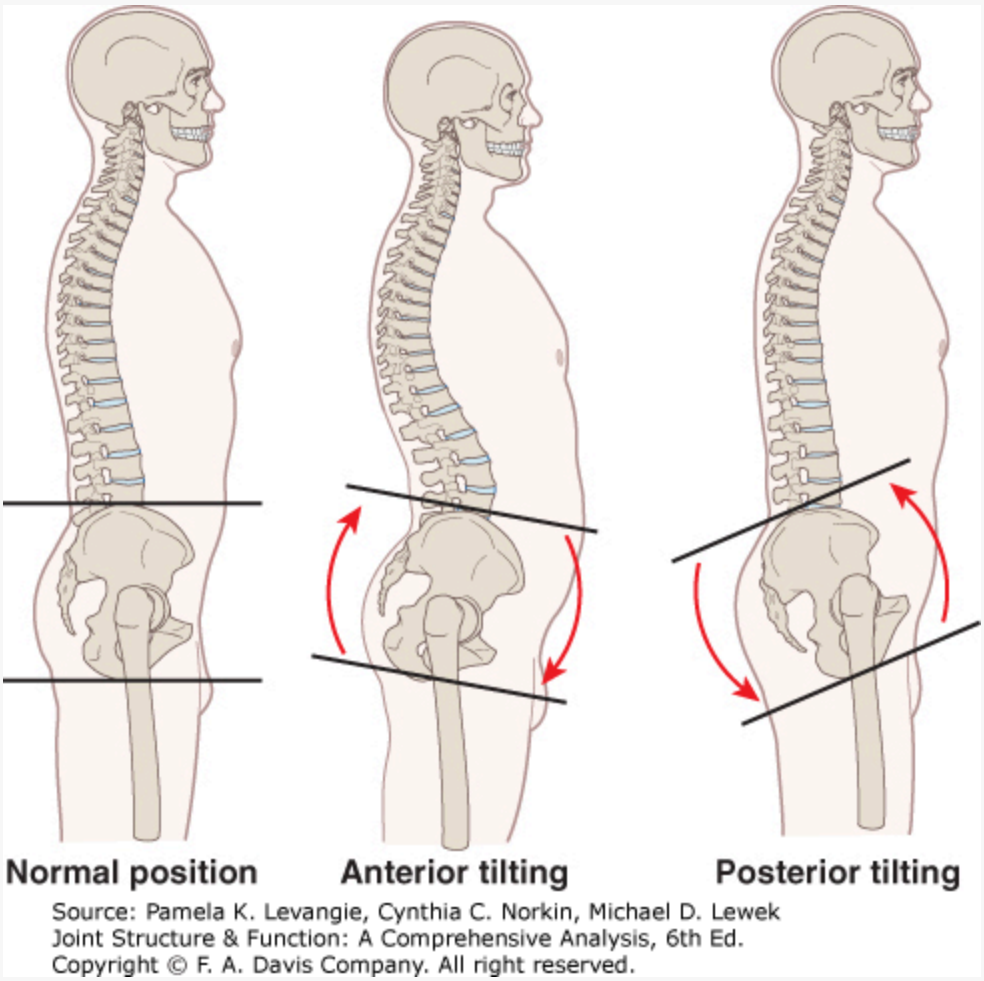
movement of the femur on the pelvis.

ANTERIOR AND POSTERIOR PELVIC TILT

Anterior and **posterior pelvic tilts** are motions of the entire pelvic ring in the sagittal plane around a coronal axis (Fig. 10–22). In the normally aligned pelvis, the anterior superior iliac spines (ASISs) of the pelvis lie on a horizontal line with the posterior superior iliac spines and on a vertical line with the symphysis pubis (Fig. 10–22A).[91] Anterior and posterior tilting of the pelvis on the fixed femur produces hip flexion and extension, respectively. Hip joint extension through posterior tilting of the pelvis brings the symphysis pubis up and the sacrum of the pelvis closer to the femur, rather than moving the femur posteriorly on the pelvis (Fig. 10–22B). Hip flexion through anterior tilting of the pelvis moves the anterior superior iliac spines anteriorly and inferiorly; the inferior sacrum moves farther from the femur, rather than moving the femur away from the sacrum (Fig. 10–22C). Importantly, anterior and posterior tilting will result in flexion and extension of both hip joints simultaneously in bilateral stance or can occur at the stance hip joint alone if the opposite limb is non-weightbearing.

FIGURE 10-22

Flexion and extension of the hip occurring as tilting of the pelvis in the sagittal plane. **A.** The pelvis is shown in its normal position in erect stance. **B.** Anterior tilting of the pelvis moves the symphysis pubis inferiorly on the fixed femur. The hip joint flexes. **C.** In posterior tilting, the anterior superior iliac spines move superiorly on the fixed femur. The hip joint extends.



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Foundational Concepts 10–2

Anterior/Posterior Pelvic Tilt Versus Pelvic Torsion

Clinicians who evaluate and treat sacroiliac joint dysfunction may attempt to diagnose someone as having asymmetry in the sagittal plane between the two halves of the pelvis (ilia or innominate bones). There are a number of terms used to label this imbalance that are beyond the scope of this discussion. However, when the imbalance is referred to as anterior or posterior torsion—or, more confusingly, anterior or posterior tilt—the potential for confusion for the novice is great. When the terms *anterior/posterior torsion* or *anterior/posterior tilt* are used in reference to the *sacroiliac joint* and *sacroiliac joint dysfunction,* these terms generally need to be distinguished as different from anterior/posterior tilt of the entire pelvis, in which the

pelvis is considered to move as a single fixed unit. One way in which clinicians attempt to make this clear is to refer to one side being anteriorly/posteriorly tilted when referring to sacroiliac joint dysfunction. For example, reference to a right side anteriorly tilted innominate makes it clear that only one side may be tilted, rather than the entire pelvic ring. Nevertheless, in this chapter and through this text, **anterior/posterior tilt** of the pelvis will be used exclusively to refer to the pelvis as a fixed unit.

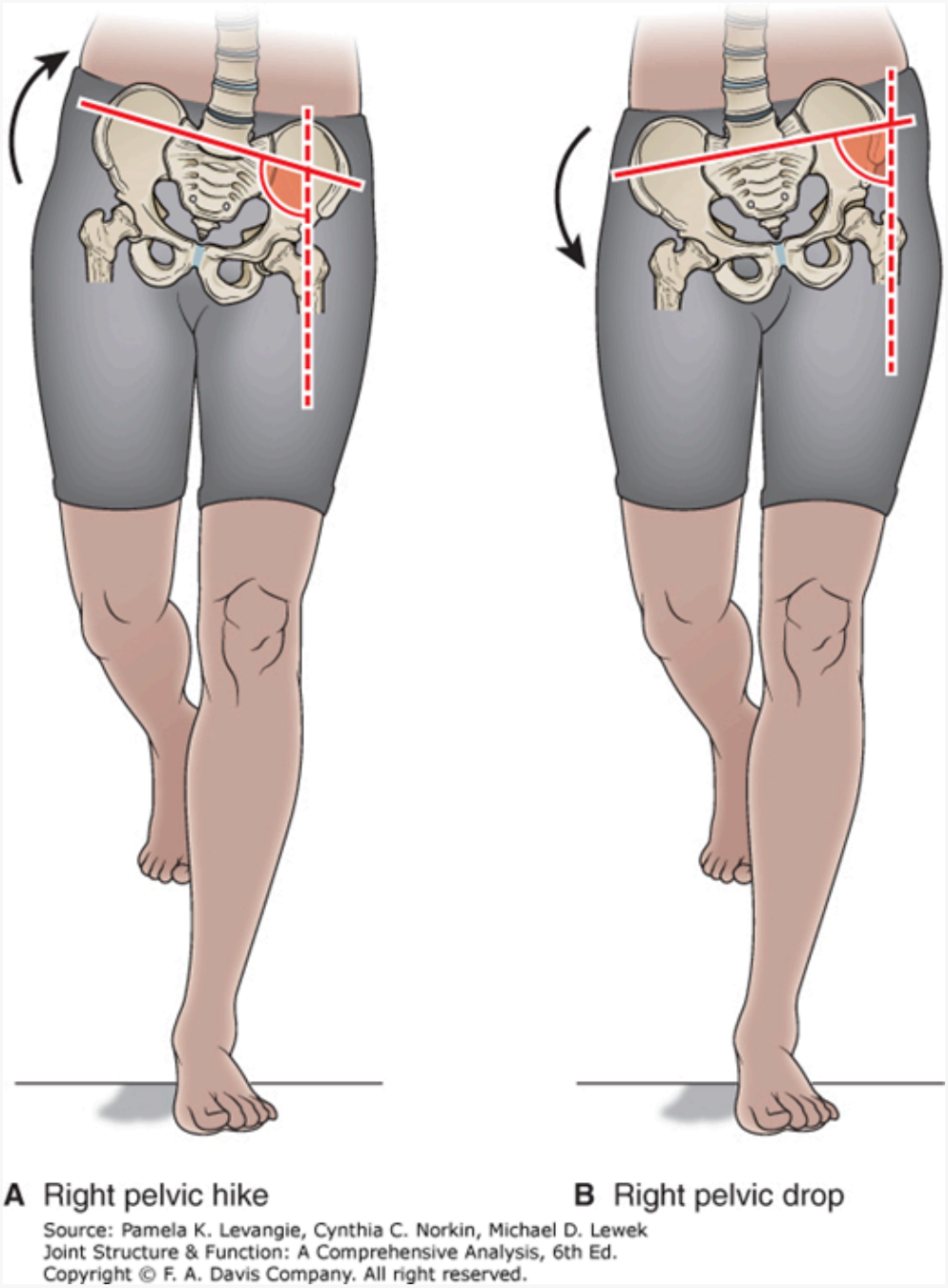
LATERAL PELVIC TILT

Lateral pelvic tilt is a frontal plane motion of the entire pelvis around an anteroposterior axis (Fig. 10–23). In the normally aligned pelvis, a line through the anterior superior iliac spines is horizontal. In unilateral stance, however, the pelvis will often laterally tilt. Lateral pelvic tilt is named by what is happening to the side of the pelvis *opposite* to the weightbearing hip in unilateral stance. The weightbearing hip joint (e.g., the left hip joint) serves as the pivot point or axis for motion of the pelvis as the pelvis either elevates **(pelvic hike)** or drops **(pelvic drop)** in the frontal plane. If a person stands on the left limb and hikes the pelvis (i.e., right iliac crest rises), the left hip joint is being abducted because the medial angle between the femur and a line through the anterior superior iliac spines increases (Fig. 10–23A). If a person stands on the left leg and drops the pelvis (i.e., right iliac crest drops), the left hip joint will adduct because the medial angle formed by the femur and a line through the anterior superior iliac spines will decrease (Fig. 10–23B).

FIGURE 10-23

Lateral tilting of the pelvis around the left can occur either as hip hiking (elevation of the opposite side of the pelvis) or as pelvic drop

(drop of the opposite side of the pelvis). **A.** Hiking of the pelvis around the left hip joint results in left hip abduction. **B.** Dropping of the pelvis around the left hip joint results in left hip joint adduction. Although it is visually tempting to name the direction of lateral tilt by the motion of the side of the pelvis *nearest* the hip, this is incorrect.



The weightbearing hip joint in unilateral stance will always be the axis of rotation, and the opposite side of the pelvis will always be the reference side for naming the movement. In descriptions of the hip joint motions that occur in unilateral stance, the hip joint of the non-weightbearing limb is in an open chain and has no *imposed*

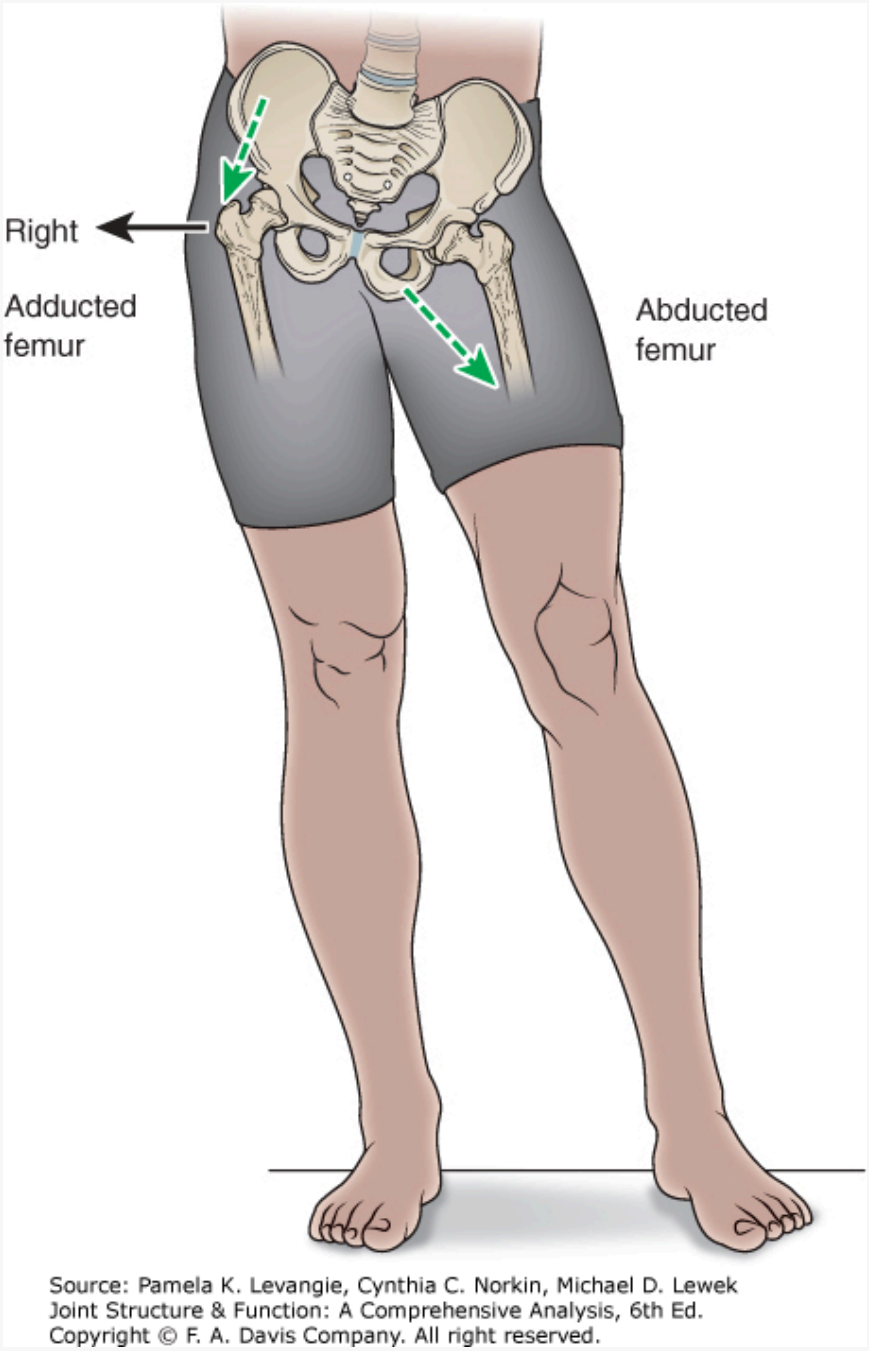
motions on it. However, the non-weightbearing leg typically hangs straight down as the pelvis moves. If the non-weightbearing limb continues to hang vertically, the non-weightbearing hip will adduct and abduct with hike and drop of the pelvis, respectively, around the weightbearing hip joint (see Fig. 10–23).

LATERAL SHIFT OF THE PELVIS

Lateral pelvic tilt can also occur in bilateral stance. If both feet are on the ground and the hip and knee of one limb are flexed, the opposite limb is largely the weightbearing limb and the terminology is the same as for unilateral stance. However, if both limbs are weightbearing, lateral tilt of the pelvis will cause the pelvis to shift to one side or the other. With **pelvic shift,** the pelvis cannot hike; it can only drop. Because there is a closed chain between the two weightbearing feet and the pelvis, both hip joints will move in the frontal plane in a predictable way as the pelvic tilt (or pelvic shift) occurs. If the pelvis is shifted to the right in bilateral stance, the left side of the pelvis will drop, the right hip joint will be adducted, and the left hip joint will be abducted (Fig. 10–24).

FIGURE 10-24

When the pelvis is shifted to the right in bilateral stance, the right hip joint will be adducted and the left hip joint will be abducted. To return to neutral position while continuing to bear weight on both feet, the right abductor and left adductor muscles work synergistically to shorten and shift the weight back to center.



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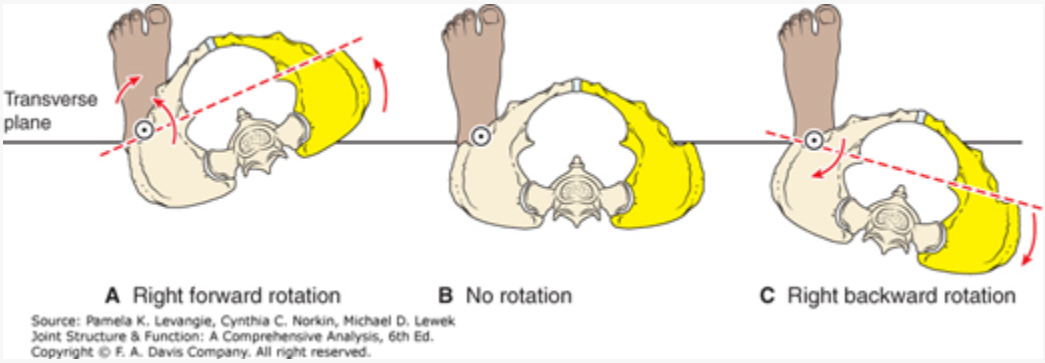
FORWARD AND BACKWARD PELVIC ROTATION

Pelvic rotation is motion of the entire pelvic ring in the transverse plane around a vertical or longitudinal axis. Although rotation can occur around a vertical axis through the middle of the pelvis in a bilateral stance, it most commonly and more importantly

occurs in single-limb support around the axis of the supporting or weightbearing hip joint (Fig. 10–25). **Forward** (anterior) **rotation** of the pelvis occurs in unilateral stance when the side of the pelvis opposite to the weightbearing hip joint moves anteriorly (Fig. 10–25A) from the neutral position. Forward rotation of the pelvis produces medial rotation of the weightbearing hip joint. **Backward** (posterior) **rotation** of the pelvis occurs when the side of the pelvis opposite the weightbearing hip moves posteriorly (Fig. 10–25C). Backward rotation of the pelvis produces lateral rotation of the weight-bearing hip joint.

FIGURE 10-25

A superior view of rotation of the pelvis in the transverse plane. **A.** Forward rotation of the pelvis around the left hip joint results in medial rotation of the left hip joint. **B.** Neutral position of the pelvis and the left hip joint. **C.** Backward rotation of the pelvis around the left hip joint results in lateral rotation of the left hip joint.



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Pelvic rotation can occur in bilateral stance as well as unilateral stance, as is true for lateral pelvic tilt. If both feet are bearing weight and the axis of motion occurs around a vertical axis through the *center* of the pelvis, the terms *forward rotation* and *backward*

rotation must be used by referencing a side (e.g., forward rotation on the right and backward rotation on the left).

Foundational Concepts 10–3

Pelvic Rotation and Hip Joint Rotation

Similar to a lateral pelvic tilt (e.g., pelvic hike or pelvic drop), when referencing forward and backward rotation, the reference is the side of the pelvis opposite to the rotating (weightbearing) hip joint (see Fig. 10–25). If it is known which leg a person is standing on in unilateral stance, forward or backward rotation of the pelvis always references the side of the pelvis opposite to the weightbearing hip joint.

The relative rotation of the hip that occurs with forward or backward rotation of the pelvis in unilateral stance is often difficult for the novice to identify. The rotation of the hip joint that occurs during rotation of the pelvis can best be appreciated by performing the motion yourself. Standing on one leg and rotating the pelvis and trunk forward as much as possible will give a clear "feeling" of the relative medial rotation of the supporting limb. Similarly, rotating the pelvis backward as far as possible will give the feeling of the relative lateral rotation of the stance hip joint.

Expanded Concepts 10–6

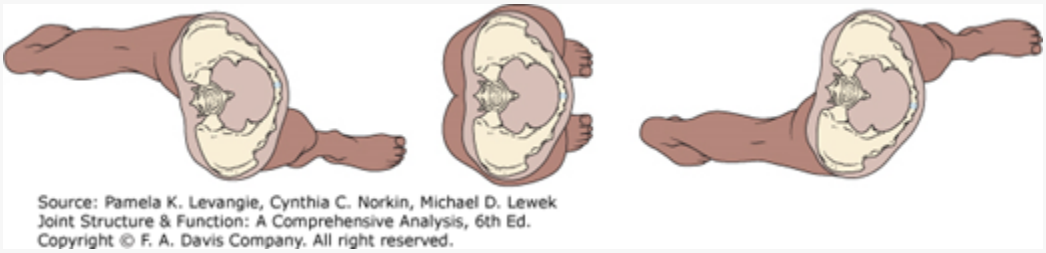
Pelvic Rotation in Gait

In normal gait, the pelvis rotates forward around the weightbearing hip in alternating fashion to advance the lower extremity during each step. The pelvis is forward rotating on the weightbearing limb (stance phase) and is in relative backward rotation during the swing phase of gait (**Fig. 10–26**).

FIGURE 10-26

This schematic representation of a superior view of the pelvis is shown forwardly rotating sequentially around the left and right hips

during gait (the rotation is exaggerated to make the point more clearly). Observation of the right side of the pelvis alone will give the illusion of the pelvis forwardly and backwardly rotating sequentially.



Coordinated Motions of the Femur, Pelvis, and Lumbar Spine

When the pelvis moves on a relatively fixed femur, there are two possible outcomes to consider. Either the head and trunk will follow the motion of the pelvis (moving the head through space) or the head will continue to remain relatively upright and vertical despite the pelvic motions. These are open- and closed-chain responses, respectively. Each of these two situations produces very different reactions from the joints and segments proximal and distal to the hip joints and pelvis and must be examined separately.

PELVIFEMORAL MOTION

When the femur, pelvis, and spine move in a coordinated manner to produce a larger ROM than is available to one segment alone, the hip joint is participating in what will predominantly (but not exclusively) be an open-chain motion termed **pelvifemoral motion.** Pelvifemoral motion can be considered analogous to scapulohumeral motion because the combination of motions at several joints serves to increase the range available to the distal segment. In the case of scapulohumeral motion, the joints are serving the hand. In the case of pelvifemoral motion, the joints may serve either end of

the chain: the foot or head.



Foundational Concepts 10–4

Moving the Head and Arms Through Space

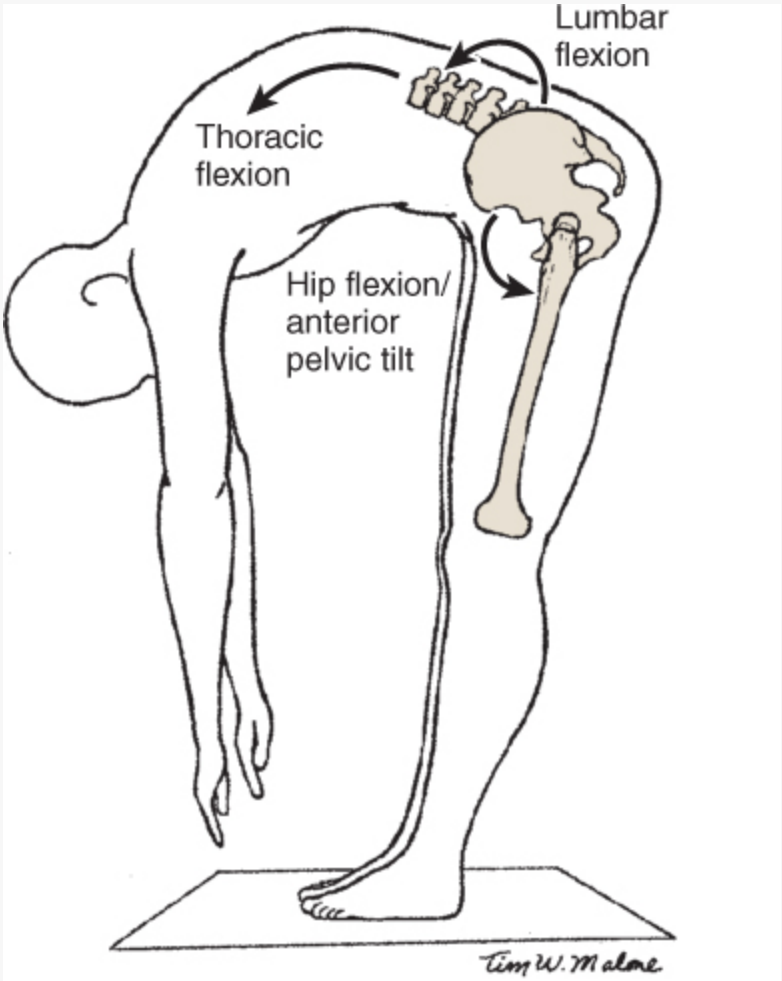
If the goal is to bend forward to bring the hands (and head) toward the floor, isolated flexion at the hip joints (anteriorly tilting the pelvis on the femurs) is generally insufficient to reach the ground. *If* the knees remain extended, the hips will typically flex no more than 90° (and often less, depending on extensibility of the hamstrings). The addition of flexion of the lumbar spine (and, perhaps, flexion of the thoracic spine) will add to the total ROM (**Fig. 10–27**). The combination of hip and trunk flexion is generally sufficient for the hands to reach the ground—as long as the hamstrings and lumbar extensors allow sufficient lengthening. The combination of hip and lumbar motion to achieve a greater ROM for the hands and head is an example of a largely open-chain response in the hips and trunk.

The open-chain response (the ability of each joint in the chain to move independently) is somewhat constrained (largely at the ankles) by the need to keep the line of gravity within the base of support.

Note: Please note that this is not an example of how to reach the floor to pick up an object!

FIGURE 10-27

Pelvifemoral motion can increase the range of forward flexion of the head and arms by combining hip flexion, anterior pelvic tilt, and flexion of the lumbar spine. This combination permits the hands to maximize the reach toward the ground.



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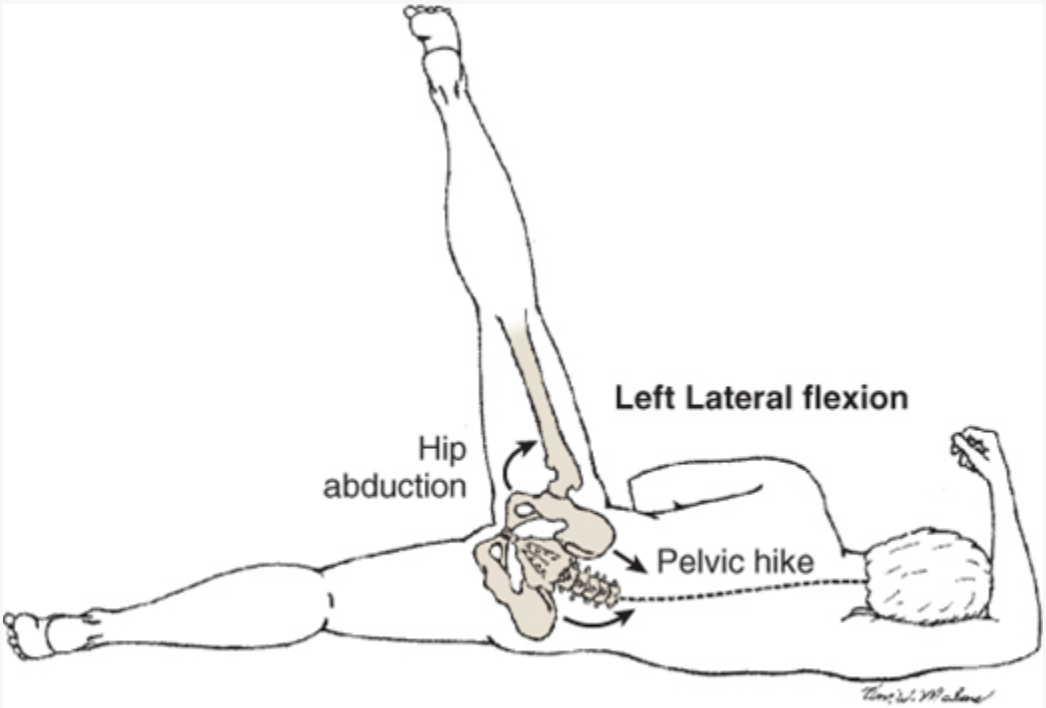
Expanded Concepts 10–7

Moving the Foot Through Space

When a person is lying on the right side, the left foot may be moved through an arc of motion approaching 90° (Fig. 10–28). This is clearly not all from the left hip joint, which can typically abduct only to 45°; motion of the foot through space also includes lateral tilting of the pelvis (hiking around the right hip joint) and lateral flexion of the lumbar spine to the left. The abducting limb is in an open chain; the lumbar spine (and thoracic spine) is constrained by the body weight and contact with the ground.

FIGURE 10-28

Pelvifemoral motion increases the range through which the foot can be moved in space by combining left hip abduction, lateral pelvic tilt (left hike), and lateral flexion of the lumbar spine to the left.



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Pelvifemoral motion has also been referred to as **pelvifemoral "rhythm,"** which implies a continuous relationship between the two segments that is arguable because the relative contributions can vary among individuals and in different activities. Pelvic tilt contributes between 30% and 46% of the total range of a passive straight leg raise.[92] During active maximal hip flexion (knee flexed) in standing (e.g., marching in place), pelvic tilt contributed between 8% and 32% of the total motion, with an even greater variability among individuals (9% to 53%) when a 4.53-kg ankle weight was added.[93] The link between hip, pelvis, and lumbar motion is the basis of using provocation of

pain with active straight-leg raising as a test for severity of dysfunction in persons with low back pain.[94,95]

CLOSED-CHAIN HIP JOINT FUNCTION

The joints of the right and left lower limbs are part of a true closed chain when both lower limbs are weightbearing and the chain is defined as all the segments between the right foot, up through the pelvis, and down through the left foot. A true closed chain is formed because both ends of the chain (both feet in this example) are "fixed" and movement at any one joint in the chain invariably involves movement at one or more other links in the chain. It is important not to confuse the idea of weightbearing and closed chain as interchangeable terms. In *Foundational Concepts 10–4,* pelvic motion on the fixed femur results in moving the head and trunk through space. This is an open-chain movement despite the feet remaining securely to the floor in a weightbearing position.

For the hips (and other lower limb joints) to be in a closed chain in standing, both ends of the chain (the head and feet) must be fixed. The feet are, in fact, fixed by weight-bearing. The head, however, is often (but not necessarily) functionally "fixed." Although the head is certainly free to move in space, it most often remains upright and vertically oriented during upright activities. The drive to keep the head upright is due, in part, to the influence of the tonic labyrinthine and optical righting reflexes that are normally evident almost immediately at birth and continue to operate throughout life.[96] The desire to keep the head upright and over the sacrum will effectively *fix the head in relative space*; that is, the head is functionally, rather than structurally, fixed. When the head (one end of the chain) is held upright and over the feet (the other end of the

chain), all the segments in the axial skeleton and lower limbs function as part of a closed chain; movement at one joint will create movement in at least one other link in the chain. Consequently, in this functional closed-chain premise, hip flexion does not occur independently (which would move the head forward in space) but is accompanied by mandatory motion in one or more interposed segments to ensure that the head remains upright over the base of support and that the body remains stable. Table 10–1 presents the compensatory motions of the lumbar spine that accompany given motions of the pelvis and hip joint in a functional closed chain.

Table 10-1

Relationship of Pelvis, Hip Joint, and Lumbar Spine During Right Lower Extremity Weightbearing and Upright Posture



Expanded Concepts 10–8

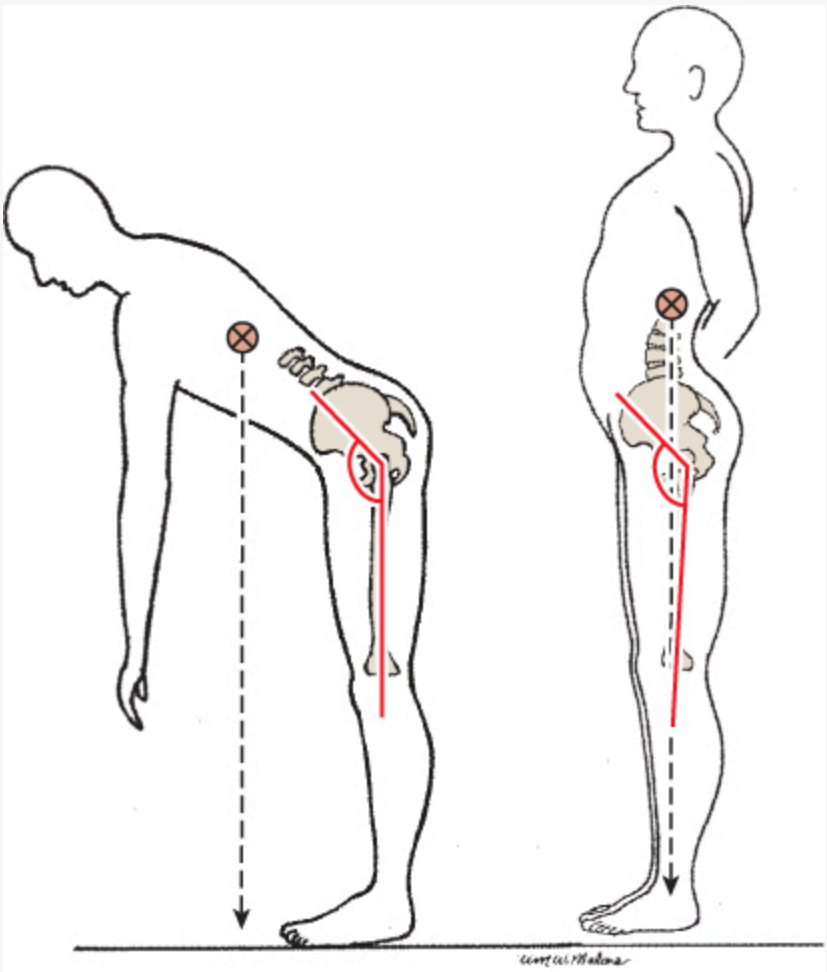
Closed-Chain Hip Joint Function

A common example of closed-chain (versus open-chain) function is seen when the hip flexor musculature is tight and the hip joint is maintained in flexion (Fig. 10–29). A person standing with fixed hip flexion is shown in Figure 10–29A (an open-chain response) and **B** (a closed-chain response). A true open-chain response to isolated hip flexion would displace the head and trunk forward, with the line of gravity falling in front of the supporting feet. More commonly, hip flexion in stance is not isolated to the hips but is accompanied by compensatory movements of the vertebral column (including extension or lordosis of the lumbar spine) that maintain the head in the upright position and keep the line of gravity within the base of support. In a functional closed-chain, motion at the hip (one link in the chain) is accompanied by

an essentially mandatory lumbar *extension* to maintain the head over the sacrum (see Fig. 10–29B). In contrast, hip flexion in open-chain pelvifemoral motion is accompanied by *lumbar flexion* because the goal is to achieve more range for the head in space (see Fig. 10–27).

FIGURE 10-29

A. In an open-chain response to tight hip flexors that is isolated to the hip joints, the trunk will be inclined forward. The line of gravity (LoG) will fall outside the base of support if no other adjustments are made. **B.** In a functional closed-chain response to tight hip flexors, the head seeks to maintain a vertical position; the lumbar spine will extend (become lordotic) to return the head to a position over the sacrum and maintain the LoG within the base of support.



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Patient Application 10–4

Skeletal shortening of the limb with a developmental hip dysplasia is not unusual. As Gabriella stands with her weight evenly distributed between her feet, her pelvis will be laterally tilted (down on the left) as a result of a measured 1-in. shortening of her left leg. To keep the line of gravity within the center of her base of support in bilateral stance, Gabriella's lumbar spine will be slightly laterally flexed to the right (away from the side of shortening). This is the opposite lumbar motion to what we saw in *Expanded Concepts 10–7* because Gabriella's goal is to keep her head upright, not to gain ROM. The lateral flexion of the spine with asymmetrical leg lengths puts a person at risk for low back pain, although this is not one of Gabriella's presenting

complaints. Interestingly, the relative abduction of the left leg that occurs in stance with shortening of Gabriella's dysplastic limb may reduce stress on the hip. The abducted position of the hip has the potential to increase congruence slightly, diminishing the peak pressure at the hip joint by distributing the forces over a larger contact area.

Hip Joint Musculature

There have been numerous studies of the muscles of the hip joint. Most confirm underlying principles of muscle physiology seen at the other joints we have examined so far. That is, hip joint muscles work best in the middle of their contractile range or on a slight stretch (at so-called optimal length–tension); two-joint muscles generate greatest force when not required to shorten over both joints simultaneously; and tension generation is optimal with eccentric contractions, followed by isometric and then concentric contractions.

The muscles of the hip joint make their most important contributions to function during weightbearing. In weight-bearing, the muscles are called on to move or support the HAT (approximately two-thirds of body weight) rather than the weight of one lower limb (approximately one-sixth of body weight). Consequently, the hip joint muscles adapt their structure to the required function, as can be seen in their large areas of attachment, their length, and their large cross-section. The alignment of the hip joint muscles and the large ROM available at the hip joint result in muscle functions that are strongly influenced by hip joint position. For example, the adductor muscles may assist with hip flexion in the neutral hip joint but will function as hip extensors when the hip joint is already flexed.[97] Delp and colleagues used computer modeling to determine that the medial rotators were capable of generating greater torque in positions of increased hip flexion, whereas the torque-generating capacity of the lateral rotators

decreased in positions of increased hip flexion.[98] They similarly determined that the **piriformis muscle** was a lateral rotator at 0° of hip flexion but a medial rotator at 90° of hip flexion. Such alterations in function are found in a few muscles at the shoulder (the clavicular portion of the pectoralis major, for example), but are fairly common in the hip joint. As a consequence, results of various studies may appear to be contradictory, but in fact, the different testing conditions can be used to explain the differing results. Some gender-related differences may also explain differential findings.[99]

It is best to examine muscle action at the hip joint in the context of specific functions such as single-limb support, posture, and gait. The next section will briefly review muscle function, but we will leave more detailed analyses for later in this and other chapters. Although the traditional action of each muscle on the distal femoral segment is described for the most part, it must be emphasized that any of the muscles are as likely (or more likely) to produce joint action by moving the proximal pelvic segment instead.

FLEXORS

The flexors of the hip joint function primarily as mobility muscles in open-chain function; that is, they function primarily to bring the swinging limb forward during ambulation or in various sports-specific movements. The flexors may function secondarily to resist strong hip extension forces that occur as the body passes over the weightbearing foot. Nine muscles have action lines crossing the anterior aspect of the hip joint: **rectus femoris, iliacus, psoas major, tensor fascia latae, sartorius, pectineus, adductor longus, adductor magnus,** and **gracilis** (Fig 10–30). Of these, the primary muscles of hip flexion are the iliopsoas, rectus femoris, tensor fascia lata, and sartorius.

The iliopsoas muscle is considered to be the most important of the primary hip flexors. It consists of two separate muscles, the iliacus muscle and the psoas major muscle, both of which attach to the femur by a common tendon. The two components of the iliopsoas muscle have many points of origin, including the iliac fossa and the disks, bodies, and transverse processes of the lumbar vertebrae. Given the attachments of the psoas major muscle to the anterior vertebrae and the iliacus muscle to the iliac fossa, activity of or passive tension in these muscles would anteriorly tilt the pelvis (iliacus muscle) and, apparently, pull the lumbar vertebrae anteriorly into flexion (psoas major muscle). In closed-chain function (head vertical), however, these muscles seem to create a paradoxical lumbar lordosis (lumbar extension) that results from the body's attempt to keep the head over the sacrum with anterior pelvic tilt and lower lumbar flexion. The role of the iliopsoas muscle in hip flexion may be particularly critical when active hip flexion from a sitting position is required. The hip may not be capable of actively flexing beyond 90° when the iliopsoas muscle is paralyzed, because the other hip flexor muscles are effectively actively insufficient in that position.[100] Both segments of the iliopsoas muscle, however, are likely active in various stages of hip flexion.[97] The moment arm of the iliopsoas muscle for medial or lateral rotation is very small and probably not functionally relevant.[97,98]

FIGURE 10-30

Nine muscles have action lines crossing the anterior aspect of the hip joint. The major muscles that contribute most of the flexion torque are the rectus femoris, iliopsoas, tensor fascia latae, and sartorius. In addition, the pectineus, adductor longus, adductor magnus, and

gracilis have the capability of assisting with hip flexion.



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The rectus femoris muscle is the only portion of the quadriceps muscle that crosses both the hip and knee joints. It attaches proximally to the anterior inferior iliac spine and distally by way of a common tendon into the tibial tuberosity. The rectus femoris muscle flexes the hip joint and extends the knee joint. Because it is a two-joint hip

flexor, the position of the knee during hip flexion will affect its ability to generate force at the hip. Simultaneous hip flexion and knee extension considerably shorten this muscle and increase the likelihood of active insufficiency. Consequently, the rectus femoris muscle makes its best contribution to hip flexion when the knee is maintained in flexion.

The sartorius muscle is a strap-like muscle, which is considered to be a flexor, abductor, and lateral rotator of the hip, as well as a flexor and medial rotator of the knee. The sartorius muscle, although a two-joint muscle, should be relatively unaffected by the position of the knee, given the relatively small proportional change in length with increased knee flexion.[101] Its function is probably most important when the knee and hip need to be flexed simultaneously (as in climbing stairs), but its small cross-sectional area argues against a critical role at the hip joint.[14]

The tensor fascia lata muscle has a proximal attachment that is more lateral than the sartorius muscle, on the anterolateral lip of the iliac crest. The muscle fibers attach distally into the iliotibial band, the thickened lateral portion of the **fascia lata** of the hip and thigh, about one-fourth of the way down the lateral aspect of the thigh. The iliotibial band attaches proximally to the iliac crest lateral to the tensor fascia lata and **gluteus maximus** muscle.[102] The iliotibial band continues distally on the lateral thigh to insert into the lateral condyle of the tibia. The tensor fascia lata muscle is considered to flex, abduct, and medially rotate the femur at the hip.[97] However, the tensor fascia lata's contribution to hip abduction may be dependent on simultaneous hip flexion.[103] The most important contribution of the tensor fascia lata muscle may be in maintaining tension in the iliotibial band, in combination with the gluteus maximus. The iliotibial

band assists in relieving the femur of some of the tensile stresses imposed on the shaft by weightbearing forces.[103,104] Because bone more effectively resists compressive than tensile stresses, reduction of tensile stresses is important in maintaining integrity of the bone.

Although the importance of the tensor fascia lata muscle and iliotibial band is controversial, evidence suggests they contribute to stability of the hip joint.[102] Excessive tension in the iliotibial band may also contribute to reduced hip adduction ROM when the hip is extended. Gajdosik and colleagues performed the **Ober test,** presumed to test flexibility of the iliotibial band, on men and women without impairments.[105] They found an average passive hip adduction of 9° for men and 4° for women when both the hip and knee were extended. When the knee was flexed during the maneuver, the hip did not get past neutral and remained in 4° of abduction for men and 6° of abduction for women, which implied that there was greater tension in the lateral hip joint structures (potentially with the iliotibial band as a key factor) when the knee was flexed.[105] The Ober test presumably moves the iliotibial band from its position anterior to the greater trochanter to a position posterior to the greater trochanter by extending the hip. Movement of the iliotibial band anteriorly and posteriorly over the greater trochanter during functional activities has been implicated in **"snapping hip" syndrome** and in inflammation of the **trochanteric bursae.**

The secondary hip flexors are the **pectineus, adductor longus, adductor magnus,** and the **gracilis** muscles. These muscles are described in the next section because they are predominantly adductors of the hip. Each, however, is capable of contributing to hip joint flexion, but that contribution is dependent on hip joint position. These muscles

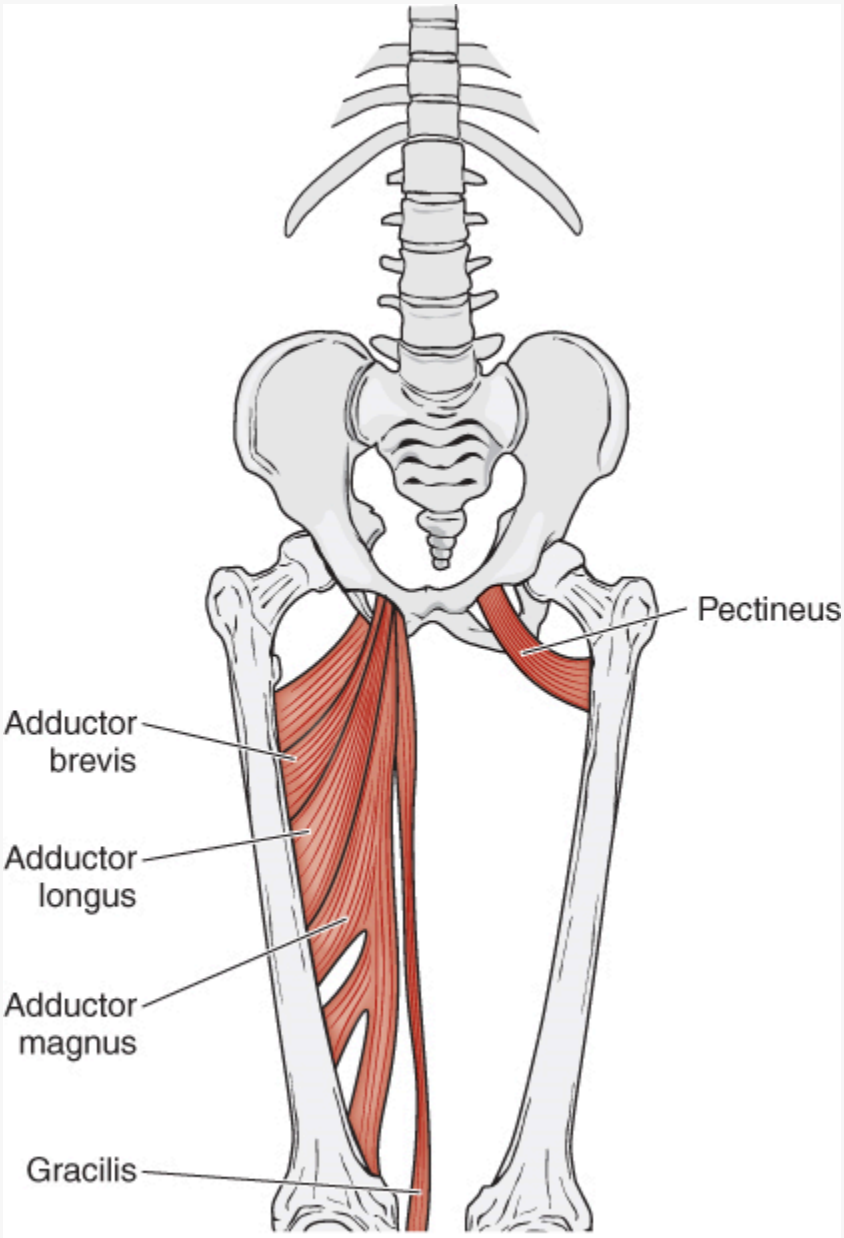
are believed to contribute up to 40° to 50° of hip flexion.[52] The gracilis, a two-joint muscle, is active as a hip flexor when the knee is extended but not when the knee is flexed.[101]

ADDUCTORS

The hip adductor muscle group is generally considered to include the **pectineus, adductor brevis, adductor longus, adductor magnus,** and the **gracilis** muscles (Fig 10–31). These adductors are located anteromedially. The contribution of the adductor muscles to hip joint function has been debated for many years. One of the reasons for debate is a question concerning the degree to which the flexed, adducted, and medially rotated posture assumed by many individuals with cerebral palsy is attributable to adductor spasticity. Arnold and Delp (using kinematic data from children with cerebral palsy and excessive medial rotation of the hip, and a "deformable femur" model) concluded that, in the normal hip in standing, the adductor brevis, adductor longus, pectineus, and posterior adductor magnus muscles had only small moment arms for medial rotation, whereas the gracilis and anterior adductor magnus muscles had small moment arms for lateral rotation.[42] With excessive femoral anteversion, the moment arms of the adductor brevis, pectineus, and the middle adductor magnus muscles switched from medial rotational to lateral rotational lines of pull. After examining the changes in moment arms with femoral anteversion or combined hip medial rotation and knee flexion, the investigators concluded that the adductors were unlikely to have a strong influence on the medially rotated hip position during the gait cycle.[42]

FIGURE 10-31

The hip adductor muscle group lies on the anteromedial aspect of the thigh and includes the pectineus, adductor brevis, adductor longus, adductor magnus, and the gracilis muscles.



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Basmajian and DeLuca believed that the adductors function not as prime movers but by reflex response to gait activities.[97] As shall be seen in our discussion of muscle function in bilateral stance, the adductors may be synergists to the abductor muscles when both feet are on the ground, enhancing side-to-side stabilization of the pelvis. Although the role of the adductor muscles may be less clear than that of other hip muscle groups, the relative importance of the adductors should not be underestimated. The adductors as a group contribute 22.5% to the total muscle mass of the lower extremity, in comparison with only 18.4% for the flexors and 14.9% for the abductors.[107] The adductors are relevant in sports-related injuries because of the high frequency of adductor longus strains, particularly in sports such as hockey.[108]

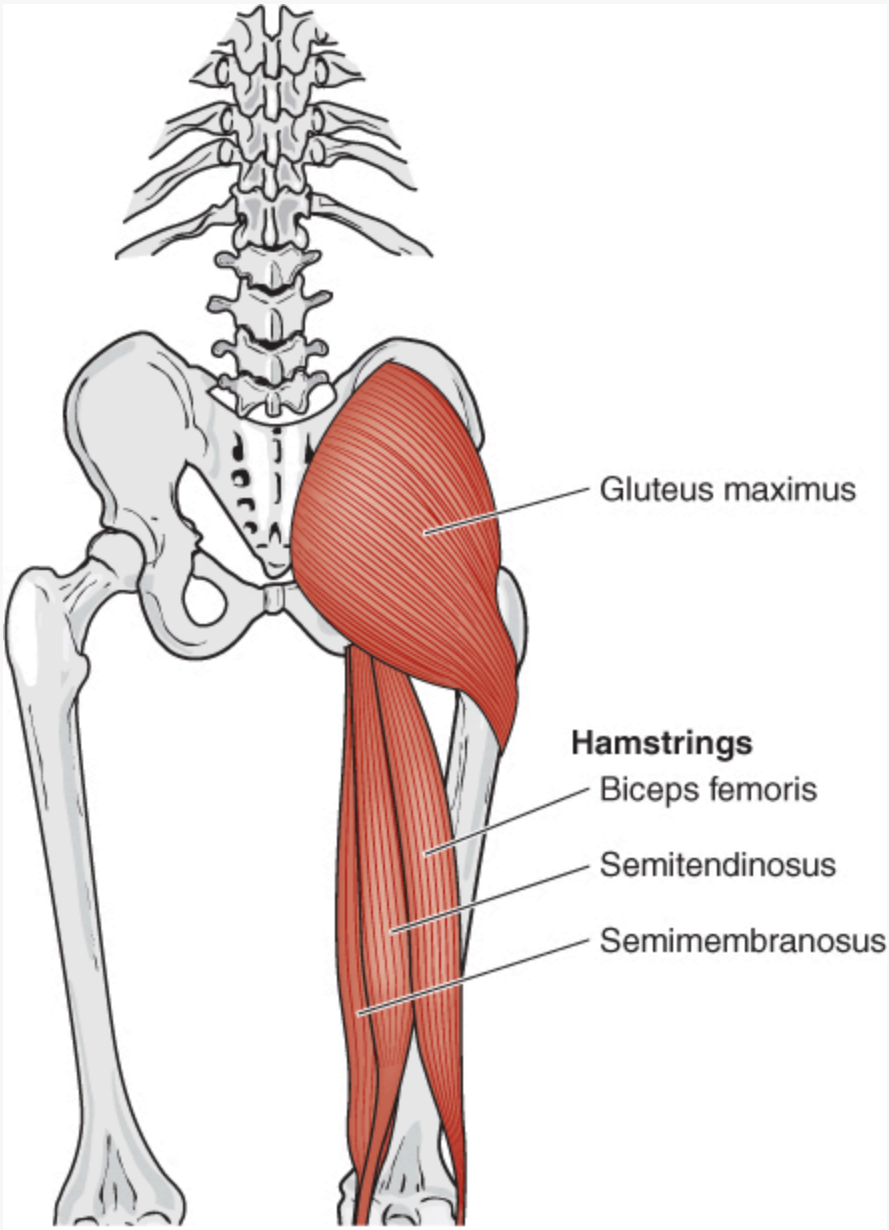
EXTENSORS

The one-joint gluteus maximus muscle and the two-joint hamstrings muscle group are the primary hip joint extensors (Fig 10–32). These muscles may receive assistance from the posterior fibers of the **gluteus medius,** from the posterior fibers of the adductor magnus muscle, and from the piriformis muscle. The **gluteus maximus** is a large, quadrangular muscle that crosses the sacroiliac joint before its most superior fibers attach to the iliotibial band (as do the fibers of the tensor fascia lata muscle) and its inferior fibers attach to the gluteal tuberosity. The gluteus maximus is the largest of the lower extremity muscles; this muscle alone constitutes 12.8% of the total muscle mass of the lower extremity.[107] The gluteus maximus is a strong hip extensor that appears to be active primarily against a resistance greater than the weight of the limb. Its moment arm for hip extension is considerably longer than that of either the hamstrings or the adductor magnus muscles and is maximal in the neutral hip joint position.[99] A favorable length–tension relationship, however, allows it to exert its peak extensor moment at 70°

of hip flexion.[109] The fibers of the gluteus maximus have a substantial capacity to laterally rotate the femur, although the moment arms for lateral rotation diminish with increased hip flexion.[98]

FIGURE 10-32

The primary hip extensors cross the joint posteriorly and include the one-joint gluteus maximus muscle and the two-joint hamstrings muscle group.



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The three two-joint extensors are the long head of the **biceps femoris,** the **semitendinosus,** and the **semimembranosus** muscles, known collectively as the **hamstrings.** All three muscles extend the hip with or without resistance, as well as serving as important knee flexors. The hamstrings increase their moment arm for hip

extension as the hip flexes to 35° and decrease it thereafter. This is somewhat in contrast to the moment arm of the gluteus maximus that is maximal at neutral position and decreases with any hip flexion thereafter.[99] Regardless of these changes in moment arm with joint position, the moment arm of the combined hamstrings for hip extension is smaller than that of the gluteus maximus at all points in the hip flexion/extension ROM. As two-joint muscles, the role of the hamstrings in hip extension is also strongly influenced by knee position. Chleboun and colleagues (using ultrasonography) determined that the moment arm for the long head of the biceps femoris was greater for hip extension than for knee flexion, with hip position affecting its excursion capability more than did knee position.[110] Although these investigators reported only on the long head of the biceps femoris, the anatomy of the medial hamstrings (semimembranosus and semitendinosus) makes it likely that these muscles have similar attributes. Importantly, if the hip is extended and the knee is flexed to 90° or more, the hamstrings may not be able to contribute much to hip extension force because of active insufficiency. Extension forces of the hip increase by 30% if the knee is extended.[109] The optimal length–tension relationship for the long head of the biceps is estimated to be at 90° of hip flexion and 90° of knee flexion, and it is likely that the medial hamstrings show similar behavior.[110] The medial hamstrings have a small moment arm for medial rotation in the neutral hip but appear to switch to lateral rotators with the knee joint in flexion.[42] The biceps femoris may contribute to lateral rotation of the hip.[97]

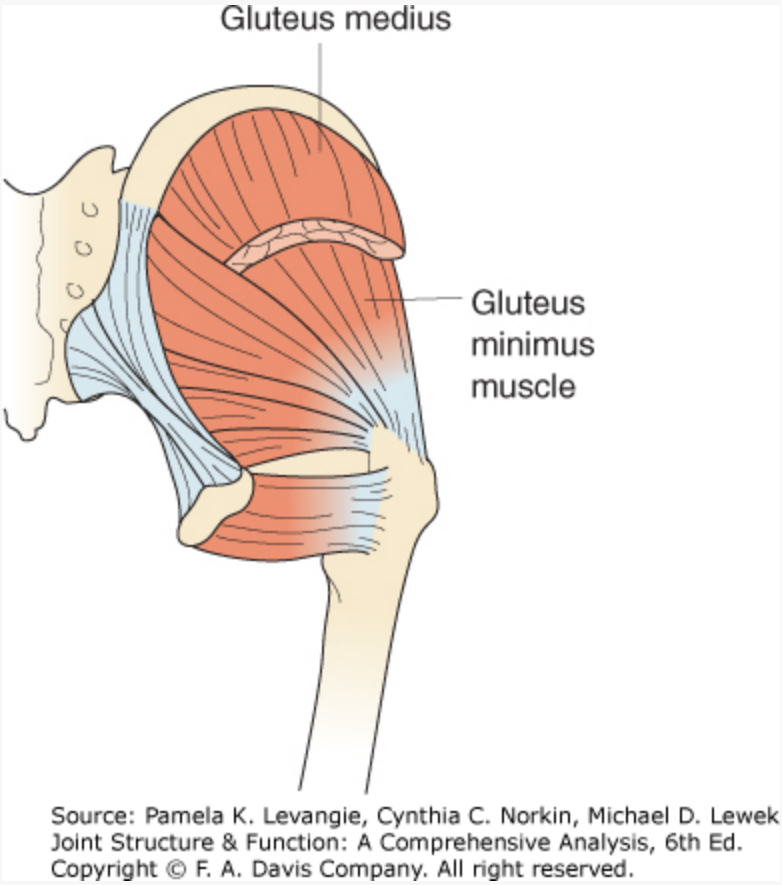
ABDUCTORS

The abductors have been likened to the “rotator cuff of the hip,” with the gluteus medius and **gluteus minimus** being analogous to the supraspinatus and subscapularis

muscles, respectively (Fig 10–33).[111–113] The gluteus medius tendon attaches distally on the lateral and posterior superior portion of the greater trochanter, resulting in a moment arm similar in direction and force to the supraspinatus.[114] Active abduction of the hip is brought about predominantly by the gluteus medius and the gluteus minimus muscles. The superior fibers of the gluteus maximus and the sartorius muscles may assist when the hip is abducted against strong resistance. The tensor fascia lata muscle may be effective as an abductor only during simultaneous hip flexion. The gluteus medius has anterior, middle, and posterior parts that function asynchronously during movement at the hip.[115] Analogous to the deltoid muscle of the glenohumeral joint, the anterior fibers of the gluteus medius are active in hip flexion, whereas the posterior fibers assist with hip extension. In the neutral hip, the posterior portion of the gluteus medius will produce a lateral rotational moment, whereas the middle and anterior portions have small medial rotational moments. In hip flexion, all portions medially rotate the hip.[98] All portions of the gluteus medius are primary abductors of the hip joint, regardless of hip joint position.

FIGURE 10-33

The muscles responsible for producing hip abduction lie on the lateral side of the joint. The prime abductors are the gluteus medius and gluteus minimus muscles.



The gluteus minimus muscle lies deep to the gluteus medius, and is best positioned to be an abductor and flexor of the hip joint, but can exert several different moments including medial rotation or lateral rotation, depending on the position of the femur. There appears to be a consensus that the gluteus minimus has a tendinous insertion into the hip joint capsule as it passes to the greater trochanter. It is hypothesized that this attachment retracts the capsule during hip abduction to prevent entrapment, or tightens the capsule to add to the gluteus minimus's function of stabilizing the femoral head in the acetabulum.[116,117] Based on these observations, it follows that one of the primary functions of the gluteus minimus is to act as a femoral head stabilizer.[118]

The gluteus minimus and medius muscles function together either to abduct the femur

(distal segment free to move: non-weightbearing) or, more important, to stabilize the pelvis (and superimposed HAT) in a unilateral stance against the effects of gravity. As will be presented later, the gluteus medius and minimus muscles will offset the gravitation adduction torque on the pelvis (pelvis drop) around the weightbearing hip in unilateral stance. The abductors are physiologically designed to work most effectively in a neutral or slightly adducted hip (slightly lengthened abductors).[119,120] Isometric abduction torque in the neutral hip position is 82% greater than abduction torque when the hip is in 25° of abduction (shortened abductors).[121] Because the gluteus medius is an important pelvic stabilizer, its weakness has been implicated in lower extremity overuse injuries such as bursitis and hip osteoarthritis, as well as disorders at the knee.[122–124]



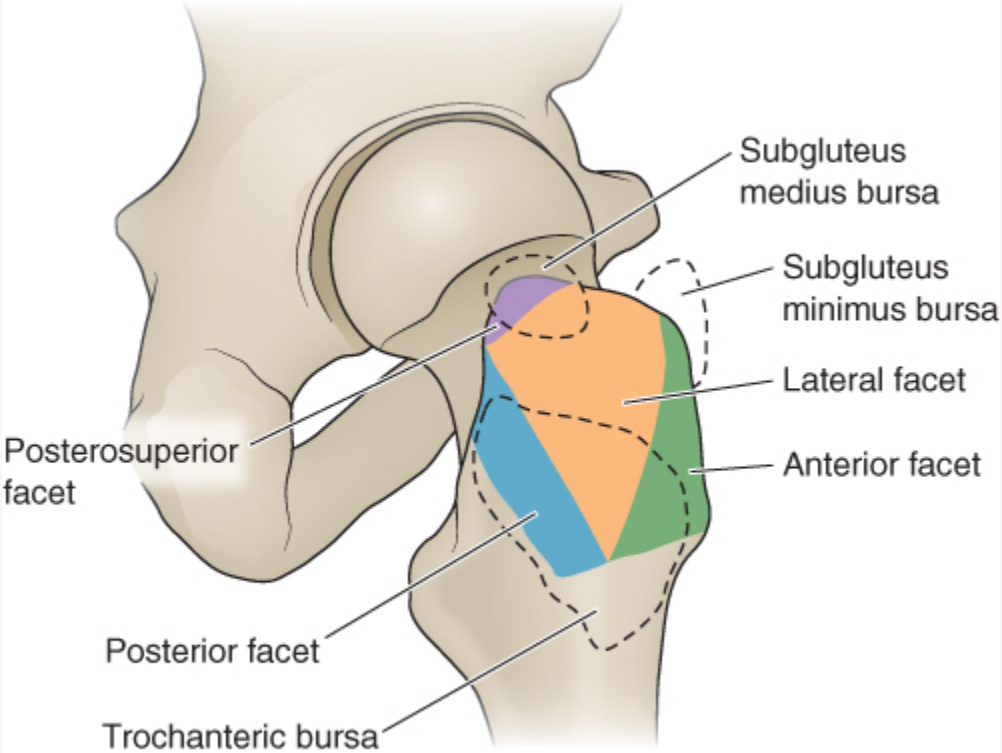
Expanded Concepts 10–9

Trochanteric Bursae

The greater trochanter has become the focus of increased interest as lateral hip pain syndromes among both the elderly and athletes are diagnosed more often.[33,64,65] Although a number of possible pathologies of both intra-articular and extra-articular origin are probably involved, there is consensus that the bursae around the greater trochanter are commonly implicated. There does not appear to be consensus on how many discrete bursae exist or how to name them. Pfirrmann and colleagues used MRI, bursography, and conventional radiography to study the greater trochanter and its associated bursae in cadavers and asymptomatic volunteers.[65] They concluded that the greater trochanter consisted of four facets: the gluteus minimus attached to the anterior facet, with the subgluteus minimus bursa beneath the tendon; the gluteus medius attached to the posterosuperior and lateral facets, with the subgluteus medius bursa beneath the tendon at the lateral facet; and the large trochanteric bursa covered the posterior facet, which was free of tendinous attachments (Fig. 10–34). Presumably, the trochanteric bursa serves to reduce friction between the posterior facet and the overlying gluteus maximus, as well as between the iliotibial band and the trochanter.

FIGURE 10-34

The greater trochanter has four facets: the anterior facet, the lateral facet, the posterior facet, and the posterosuperior facet. Also seen on cross-section are three bursae which can all become compressed due to muscle activity or a fall on the lateral hip.



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Patient Application 10–5

In addition to her other problems, Gabriella was complaining of lateral hip pain that was tender to palpation. Although other explanations for her pain exist, greater trochanter pain syndrome is a common complaint in middle-aged women (with a 4:1 ratio of women to men) and in those diagnosed with osteoarthritis.[33,64,125] The altered biomechanics associated with her leg length discrepancy may further predispose her to pathology of the lateral hip. Trochanteric bursitis and lesions of the abductor (gluteus medius and gluteus minimus) tendons commonly coexist and are

analogous to rotator cuff tears and bursitis in the shoulder.[33,64,65,126] As we continue to explore the role of the hip abductors in standing and the possible effects of hip dysplasia on the hip abductors, it will become clear that Gabriella's hip abductors and associated bursae are likely at risk for overuse injury and perhaps the start of degenerative changes.

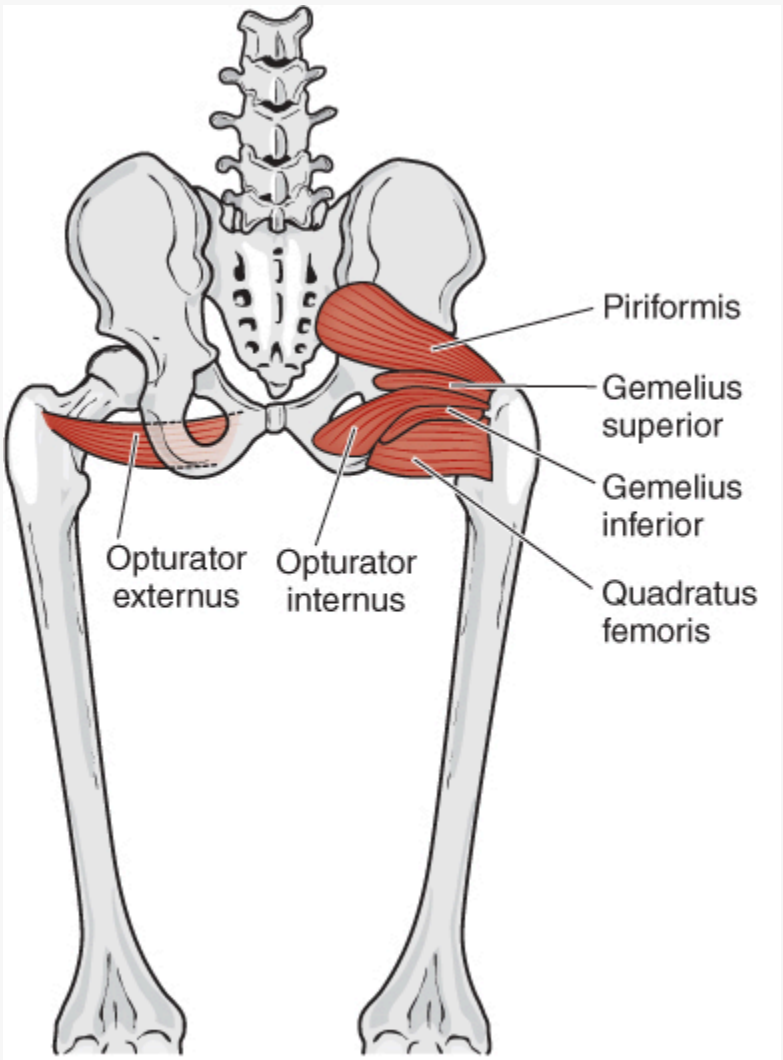
LATERAL ROTATORS

Six short muscles function primarily as lateral rotators of the hip joint. These muscles are the **obturator internus** and **externus,** the **gemellus superior** and **inferior,** the **quadratus femoris,** and the **piriformis** muscles. Other muscles that have fibers posterior to the axis of motion at the hip (the posterior fibers of the gluteus medius, minimus, and maximus) may produce lateral rotation combined with the primary action of the muscle (although it has already been noted that the lateral rotary function of these muscles decreases or becomes medial with increased hip flexion).[98] Of the primary lateral rotators, each inserts either on or in the vicinity of the greater trochanter (Fig. 10–35). The piriformis and gluteus maximus are the only two muscles that cross the sacroiliac joint. The sciatic nerve, the largest nerve in the body, enters the gluteal region just inferior to the piriformis muscle as both structures pass through the greater sciatic notch. This close proximity of the piriformis to the sciatic nerve has the potential to contribute to radiating leg pain arising from impairments associated with the piriformis muscle.

FIGURE 10-35

The lateral rotators pass posterior to the joint axis in a mediolateral direction. These short muscles include the obturator internus and externus, the gemellus superior and inferior, the quadratus femoris,

and the piriformis muscles.



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The lateral rotator muscles are positioned to perform their rotary function effectively, given the nearly perpendicular orientation to the shaft of the femur (see Fig. 10–33). However, exploration of function of these muscles has been restricted because of the relatively limited access to electromyography (EMG) surface or wire electrodes. Like their rotator cuff counterpart at the glenohumeral joint, these muscles would certainly

appear to be effective joint compressors because their combined line of pull parallels the head and neck of the femur. Using modeling, Delp and colleagues determined that the obturator internus, like the gluteal muscles, decreased its moment arm for lateral rotation with increased hip flexion.[98] The piriformis was estimated to have a large moment arm for lateral rotation with the hip joint at 0° but switched to a medial rotator with half the moment arm when the hip reached 90° of flexion. The obturator externus and quadratus femoris were the only lateral rotators that did not diminish their moment arm for lateral rotation with increased hip joint flexion.[98] Hypothetically, the lines of pull of the deep one-joint lateral rotators should make them ideal tonic stabilizers of the joint during most weightbearing and non-weightbearing activities. Although their ability to perform lateral rotation may decrease with hip flexion in some instances, the action lines of these muscles should remain largely compressive (parallel to the femoral neck) throughout the hip joint ROM.

MEDIAL ROTATORS

There are no muscles with a *primary* function of producing medial rotation of the hip joint. The more consistent medial rotators are the anterior portion of the gluteus medius, gluteus minimus, and the tensor fascia lata muscles. Although the role of the adductors in hip joint rotation is controversial, the majority of evidence appears to support the adductor muscles as medial rotators of the joint, with the possible exception of the gracilis muscle.[97,100] The ability of hip joint muscles to shift function with changing position of the hip joint is evident when medial rotation of the hip is examined. There is a trend toward increased medial rotation torques (or decreased lateral rotation torques) with increased hip flexion among many of the hip joint muscles, with three times more medial rotation torque in the flexed hip than in the

extended hip.[42,98,100] Research has suggested that the medial rotation that may accompany a “crouched” gait seen in many individuals with cerebral palsy may be attributable more to hip flexion than to adductor spasticity.[98]



Expanded Concepts 10–10

“Rotator Cuff” of the Hip

The shoulder rotator cuff can be used to help understand the function of muscles around the hip. Lateral rotation of the shoulder is performed by the infraspinatus, teres minor, and suprasinatus and is compared with the piriformis, obturator externus and internus, and superior and inferior gemelli, as well as the posterior gluteus medius at the hip. Medial rotation of the shoulder is performed by the subscapularis compared with the anterior fibers of the gluteus medius and gluteus minimus. Abduction of the shoulder is performed by the supraspinatus and is compared with the gluteus medius of the hip.[127]

Hip Joint Forces and Muscle Function in Stance

Bilateral Stance

In erect bilateral stance, both hips are in neutral or slight extension, and weight is evenly distributed between both legs. The line of gravity falls just posterior to the axis for flexion/extension of the hip joint. The posterior location of the line of gravity creates an external extension moment around the hip that tends to tilt the pelvis posteriorly on the femoral heads.

The gravitational extension moment is largely checked by passive tension in the hip joint capsuloligamentous structures, although slight or intermittent activity in the iliopsoas muscles in relaxed standing may assist the passive structures.[97]

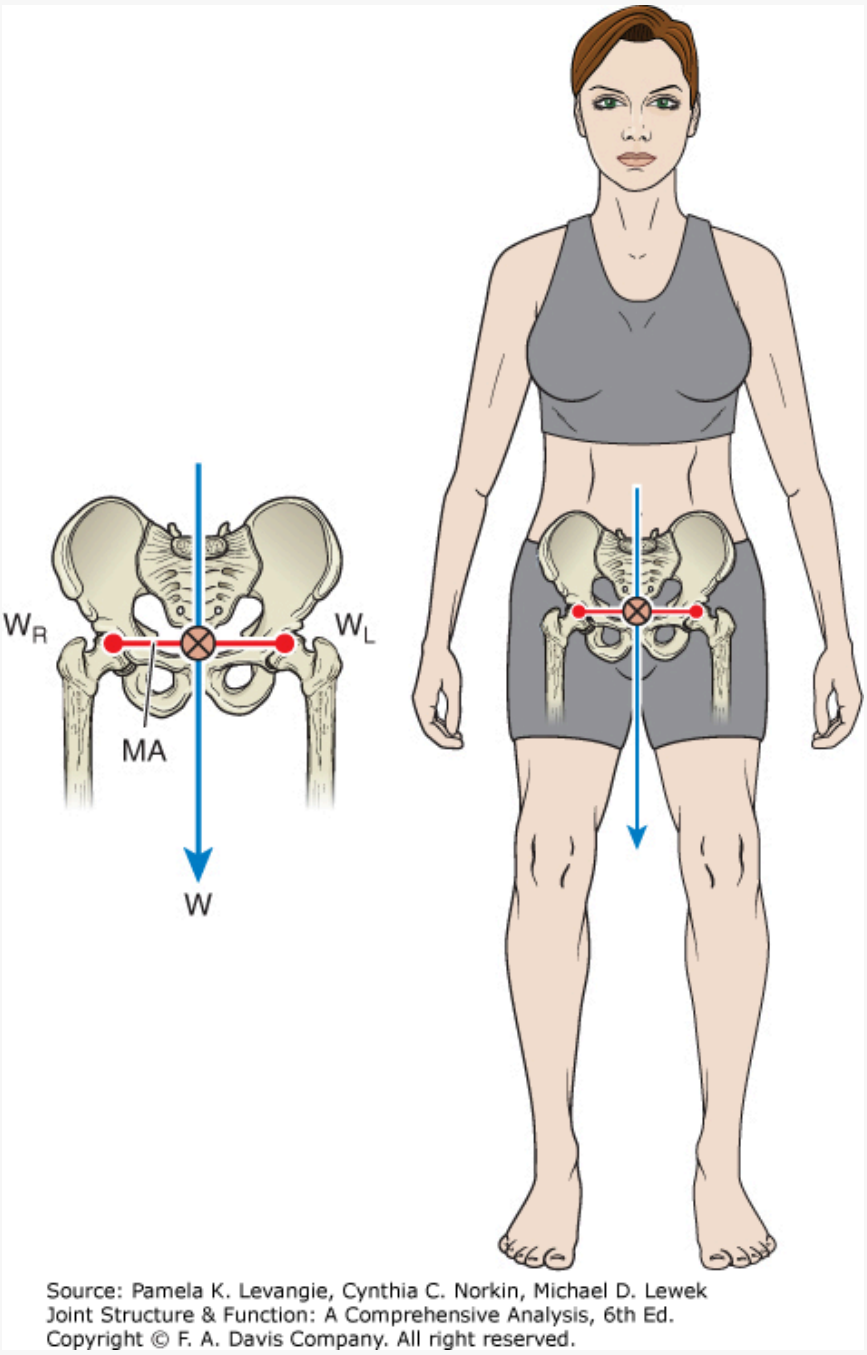
In the frontal plane during bilateral stance, the superincumbent body weight is transmitted through the sacroiliac joints and pelvis to the right and left femoral heads. Hypothetically, the weight of the HAT (two-thirds of body weight) should be distributed so that each femoral head receives approximately half of the superincumbent weight.[128] As shown in Figure 10–36, the joint axis of each hip lies at an equal distance from the line of gravity of HAT; that is, the gravitational moment arms for the right hip (MAR) and the left hip (MA_L) are equal. Because the body weight (W) on each femoral head is the same ($W_R = W_L$), the magnitude of the gravitational torques around each hip must be identical ($W_R \times MA_R = W_L \times MA_L$). The gravitational torques on the right and left hips, however, occur in opposite directions. The weight of the body acting around the right hip tends to drop the pelvis down on the left (right adduction moment), whereas the weight acting around the left hip tends to drop the pelvis down on the right (left adduction moment). These two opposing gravitational moments of equal magnitude balance each other, and the pelvis is maintained in equilibrium in the frontal plane without the assistance of active muscles. Assuming that muscular forces are not required to maintain either sagittal or frontal plane stability at the hip joint in bilateral stance, the compression across each hip joint in bilateral stance should simply be half the superimposed body weight (or one half of HAT to each hip).

FIGURE 10-36

An anterior view of the pelvis in normal erect bilateral stance. The weight acting on the right hip joint (W_R) multiplied by the distance from the right hip joint axis to the body's center of gravity (MAR) is equal to the weight acting at the left hip joint (W_L) multiplied by the

distance from the left hip to the body's center of gravity (MA_L).

Therefore, $W_R \times MA_R = W_L \times MA_L$.



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Hip joint compression can be altered by different means. This includes leaning over one lower extremity during single limb stance and the use of a cane either ipsilaterally or

contralaterally. The magnitude of hip joint compression as it relates to bilateral stance, unilateral stance, compensatory leaning, ipsilateral cane use, and contralateral cane use is presented in Examples 10–1, 10–2, 10–3, 10–4, and 10–5, respectively. A summary of the calculations is presented after the examples in Table 10–2 for direct comparison.

Table 10-2

Summary of Calculations of Hip Joint Compression

The rationale presented in Example 10–1 for assuming that each hip receives one-third of body weight in bilateral stance is reasonable. However, Bergmann and colleagues showed in several subjects with an instrumented pressure-sensitive hip prostheses that the joint compression across each hip in bilateral stance was 80% to 100% of body weight, rather than one third (33%) of body weight, as commonly proposed.[129] When they added a symmetrically distributed load to the subject's trunk, the forces at both hip joints increased by the full weight of the load, rather than by half of the superimposed load as might be expected. Although the mechanics of someone standing who has a prosthetic hip may not fully represent normal hip joint forces, the findings of Bergmann and colleagues call into question the simplistic view of hip joint forces in bilateral stance. The slight activity in the iliopsoas muscle may account for more joint compression than previously thought. Alternatively, capsuloligamentous tension or activation of secondary stabilizing muscles may contribute to joint compression.

Example 10–1

Calculating Hip Joint Compression in Bilateral Stance

Using a hypothetical case of someone weighing 825 N (~185 lb), the weight of HAT (two-thirds body weight) will be 550 N (~124 lb). Of that 550 N, half will presumably be distributed through each hip. Because we are assuming no additional compressive force produced by hip muscle activity, the total hip joint compression at each hip in bilateral stance is estimated to be 275 N (~62 lb); that is, total hip joint compression through each hip in bilateral stance is one-third of body weight.

When bilateral stance is not symmetrical, frontal plane muscle activity will be necessary either to control the side-to-side motion or to return the hips to symmetrical stance. If the pelvis is shifted to the right (see Fig. 10–24), there is a relative adduction of the right hip and abduction of the left hip. To return to neutral position, an active contraction of the right hip abductors would be expected. However, a contraction of the left hip adductors would accomplish the same goal. Thus, in bilateral stance when both lower limbs bear at least some of the superimposed weight, the contralateral abductors and adductors may function as synergists to control the frontal plane motion of the pelvis. In unilateral stance, activity of the adductors either in the weightbearing or non-weightbearing hip *cannot* contribute to stability of the stance limb. Hip joint stability in unilateral stance is the sole domain of the hip joint abductors. In the absence of adequate hip abductor function, the adductors can contribute to stability—but only in bilateral stance.

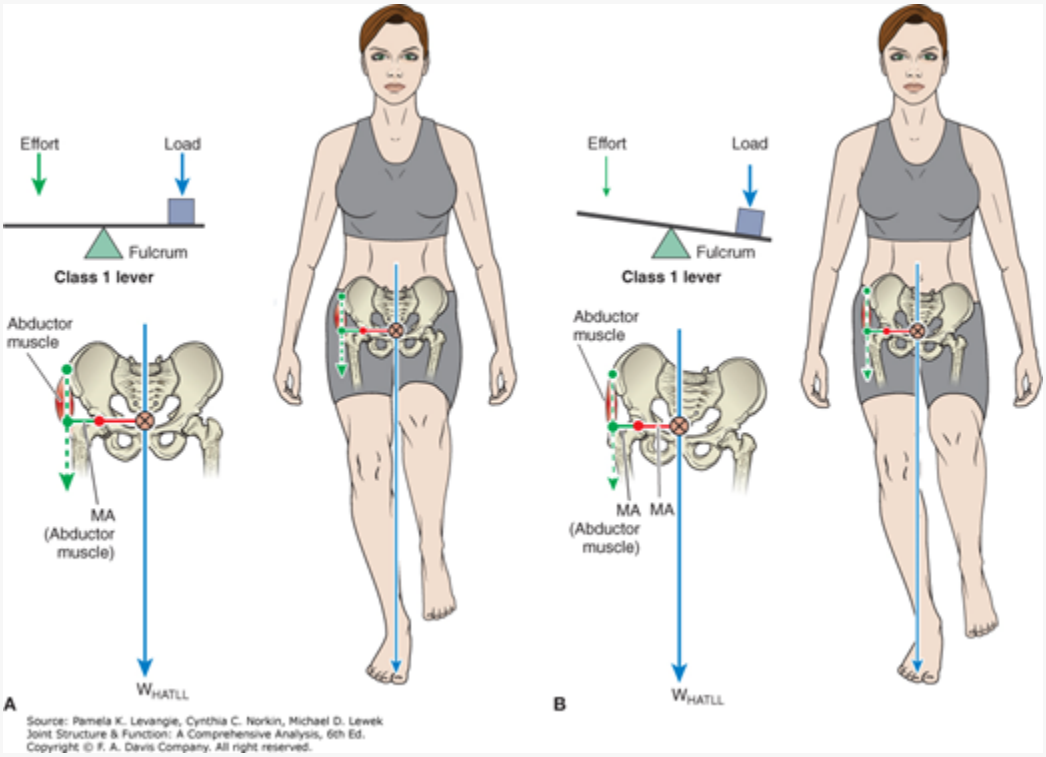
Unilateral Stance

In Figure 10–37, the left leg has been lifted from the ground and the full superimposed body weight (HAT) is being supported by the right hip joint. Rather than sharing the compressive force of the superimposed body weight with the left limb, the right hip

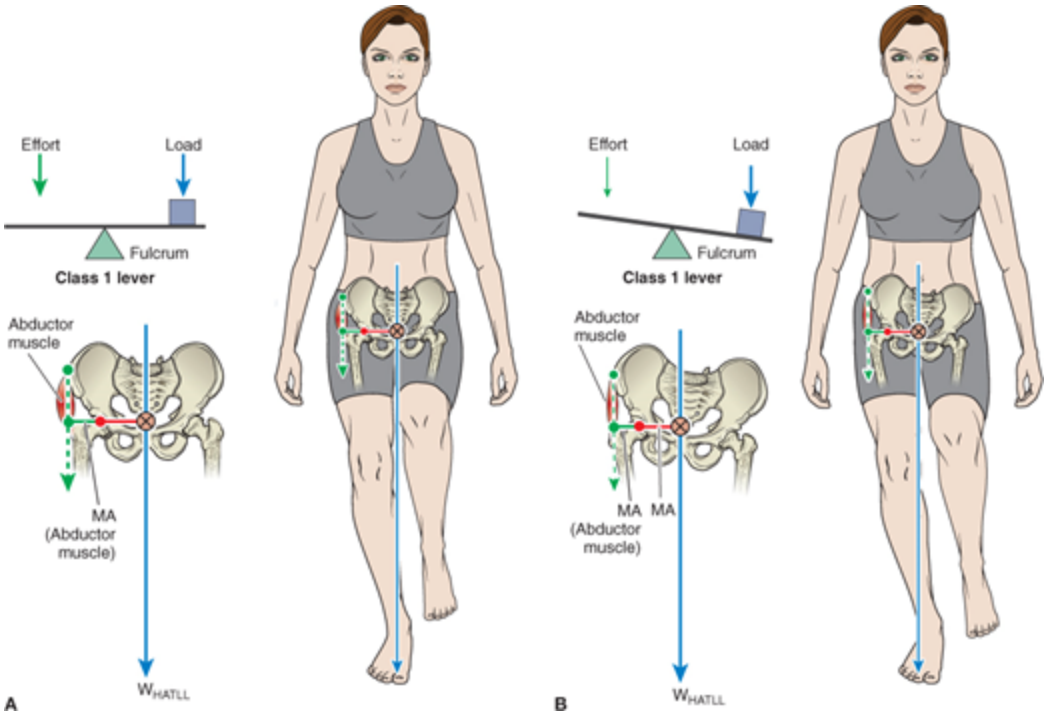
joint must now carry the full burden. In addition, the weight of the non-weightbearing left limb that is hanging on the left side of the pelvis must be supported along with the weight of HAT. Of the one-third of the portion of body weight found in the lower extremities, the non-weightbearing limb must account for half of that, or one-sixth of the full body weight.[130] The magnitude of body weight (W) compressing the right hip joint in right unilateral stance, therefore, is

FIGURE 10-37

A. In right unilateral stance, the weight of HAT acts 10 cm from the right hip joint. The hip abductors have a moment arm of approximately 5 cm and must be active to keep the pelvis level. Inset. **B.** If inadequate abduction torque is created (e.g., from a weakened gluteus medius), the pelvis will drop on the contralateral side.



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In our hypothetical subject from Example 10–1 who weighs 825 N, HAT accounts for 550 N. One lower extremity weighs one-sixth of body weight, or 137.5 N. Therefore, when this individual lifts one leg off the ground, the supporting hip joint will undergo 687.5 N (or five-sixths of body weight) of compression *from body weight alone.*[5]

Although we have accounted for the increase in hip joint compression from body weight as a person moves from double-limb support (bilateral stance) to single-limb support, the problem is more complex. Not only is the hip joint in unilateral stance being compressed by body weight (gravity), but body weight is creating a torque around the stance limb hip joint that needs to be balanced.

The combined force of gravity acting on HAT and the non-weightbearing left lower limb (HATLL) will create an adduction torque around the weightbearing hip joint; that is, gravity will attempt to drop the pelvis around the right weightbearing hip joint axis. The

abduction counter torque to maintain equilibrium will have to be supplied by the right hip abductor musculature. When muscles contract across a joint, they produce joint compression. Therefore, the total joint compression or joint reaction force is a combination of both body weight and abductor muscular compression. The total joint compression can be calculated for our hypothetical 825-N patient. The line of gravity of HATLL can be estimated to lie 10 cm (0.1 m, or ~4 in.) from the right hip joint axis. That is, the moment arm (MA) = 0.1 m, although the actual distance will vary among individuals. The 10-cm estimate of the gravitational moment arm is for symmetrical stance. The actual moment arm is likely to be somewhat smaller, however, because the weight of the hanging left leg will pull the center of gravity of the superimposed weight slightly to the right, and a compensatory trunk lean will likely be needed to shift the line of gravity slightly right to get the line of gravity within the now smaller (i.e., single foot) base of support.



Example 10–2

Calculating Hip Joint Compression in Unilateral Stance

For simplicity, the possible change in the moment arm of HATLL from the non-weightbearing limb will be ignored, so our torque calculation here is simply an estimate of the actual gravitational torque. In our simplified hypothetical example, the magnitude of the gravitational adduction torque at the right hip will be as follows:

$$\text{HATLL torque}_{\text{adduction}}\text{: } 687.5\ \text{N} \times 0.1\ \text{m} = 68.75\ \text{Nm}$$

To maintain the single-limb support position, there must be a counter torque (abduction moment) of equivalent magnitude. The counter torque must be produced by the force of the hip abductors (gluteus medius, minimus, and tensor fascia lata muscles) acting on the pelvis. Assuming that the abductor muscles act through a typical moment arm of 5 cm (0.05 m, or 2 in.) and knowing that the muscles must generate an abduction torque equivalent to the adduction torque of gravity (68.75

Nm), we can solve for the magnitude of muscle contraction (F_{ms}) needed to maintain equilibrium in our hypothetical example.[78]

$$\text{Torque}_{\text{abduction}}: 68.75 \text{ Nm} = F_{ms} \times 0.05 \text{ m}$$

$$F_{ms} = 68.75 \text{ Nm} \div 0.05 \text{ m} = 1{,}375 \text{ N}$$

Assuming that all the abductor muscular force is transmitted through the acetabulum to the femoral head, the 1,375-N abductor muscular compressive force is now added to the 687.5 N of compression caused by body weight passing through the supporting hip. Thus, the total hip joint compression, or joint reaction force, at the stance hip joint in unilateral support can be estimated for our hypothetical patient as:

$$\begin{array}{r} 1{,}375 \text{ N abductor joint compression} \\ \underline{+\ 687.5 \text{ N body weight (HATLL) compression}} \\ 2{,}062.5 \text{ N total joint compression} \end{array}$$

The hypothetical figures used in the examples oversimplify and underestimate the forces involved in hip joint stresses as already noted. However, this example illustrates nicely that the hip joint compressive force is heavily influenced by muscle activity, and that the joint compressive force increases substantially during unilateral stance compared with bilateral stance. Total hip joint compression or joint reaction forces are generally found to be two to three times body weight in unilateral stance.[109,128,131] Some investigators have found peak forces are higher than typically calculated for unilateral stance and can achieve four times body weight depending on measurement technique and individual characteristics.[132,133] Individual differences in hip structure, (i.e., angulation of the femur) may substantially alter forces and pressures across the hip joint.[57,84,134]

If total joint compression in unilateral stance is approximately three times body weight, a loss of 1 N (~4.5 lb) of body weight will reduce the joint reaction force by 3 N (13.5 lb).

For painful hip joints, however, the reductions in compression generally required are greater than can be realistically achieved through weight loss. The solution must be in a reduction of abductor muscle force requirements. If less muscular counter torque is needed to offset the effects of gravity, there will be a decrease in the amount of *muscular compression* across the joint, although the body weight compression will remain unchanged. Several options are available when there is a need to decrease abductor muscle force requirements. Some compression reduction strategies occur automatically, but at a cost of extra energy expenditure and structural stress. Other strategies require intervention such as assistive devices, but minimize the energy cost.

Compensatory Lateral Lean of the Trunk

Gravitational torque at the pelvis is the product of body weight and the distance that the line of gravity lies from the hip joint axis (its moment arm). If there is a need to reduce the torque of gravity in unilateral stance and if body weight cannot be reduced, the moment arm of the gravitational force can be reduced by laterally leaning the trunk over the pelvis toward the side of pain or weakness when in unilateral stance on the painful limb. Although leaning toward the side of pain might appear counterintuitive, the compensatory lateral lean of the trunk toward the painful stance limb will shift the line of gravity closer to the hip joint, thereby reducing the gravitational moment arm. Because the weight of HATLL must pass through the weightbearing hip joint regardless of trunk position, leaning toward the painful or weak supporting hip does not increase the joint compression caused by body weight. However, it does reduce the gravitational torque. If there is a smaller gravitational adduction torque, there will be an equivalent reduction in the need for an abductor counter torque. Although it is theoretically possible to laterally lean the trunk enough to bring the line of gravity through the

weightbearing hip (reducing the torque to zero) or to the opposite side of the supporting hip (reversing the direction of the gravitational torque), these are relatively extreme motions that require high energy expenditure and would result in excessive wear and tear on the lumbar spine. More energy efficient and less structurally stressful compensations can still yield dramatic reductions in the hip abductor force.

The 1,031.25-N joint reaction force estimated in Example 10–3 is half the 2,062.5 N of hip joint compression previously calculated for our hypothetical patient in single-limb support (see Table 10–2). This 50% reduction in joint compression may be enough to relieve some of the pain symptoms experienced by a person with arthritic changes in the hip joint or to provide some relief to a weak or painful set of abductors. The compensatory lean is instinctive and commonly seen in people with hip joint disability.



Example 10–3

Calculating Hip Joint Compression With Lateral Lean

Returning to our hypothetical patient weighing 825 N, let us assume that he can laterally lean to the right enough to bring the line of gravity within 2.5 cm (0.025 m) of the right hip joint axis (Fig. 10–38). The gravitational adduction torque would now be

$$\text{HATLL torque}_{\text{adduction}} = [5/6\ (825\ \text{N})] \times 0.025\ \text{m}$$

$$\text{HATLL torque}_{\text{adduction}} = 17.2\ \text{Nm}$$

If only 17.2 Nm of adduction torque were produced by the superimposed weight, the abductor force needed would be as follows:

$$\text{Torque}_{\text{abduction}}\text{: } 17.2\ \text{Nm} = F_{ms} \times 0.05\ \text{m}$$

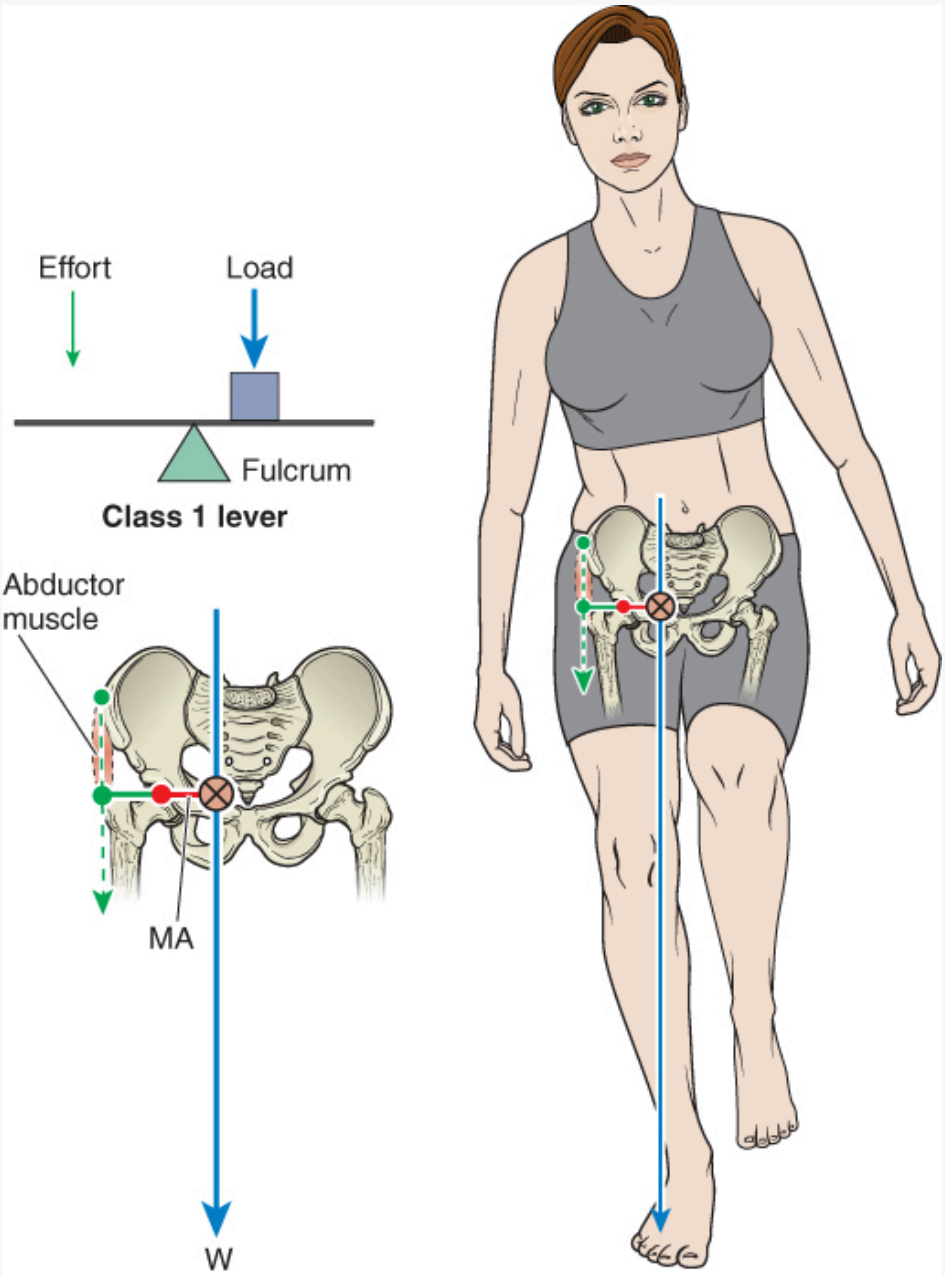
$$F_{ms}\text{: } 17.2\ \text{Nm} \div 0.05\ \text{m} = 343.75\ \text{N}$$

If only ~344 N (~77 lb) of abductor force were required, the total hip joint compression in unilateral stance using the compensatory lateral lean would now be

343.75 N abductor joint compression
+ 687.5 N body weight (HATLL) compression
1,031.25 N total joint compression

FIGURE 10-38

When the trunk is laterally flexed toward the stance limb, the moment arm of HATLL is substantially reduced (e.g., 2.5 cm, in comparison with 10 cm with the neutral trunk), whereas that of the abductors remains unchanged (e.g., 5 cm). The result is a substantially diminished torque from HATLL and, consequently, a substantially decreased need for hip abductor force to generate a countertorque.



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Expanded Concepts 10–11

Pathological Gaits

When a lateral trunk lean is seen during gait and is due to hip abductor muscle weakness, it is known as a **gluteus medius gait.** If the same compensation is due to

hip joint pain, it is known as an **antalgic gait.** In some instances, the 344-N abductor force we calculated as necessary to stabilize the pelvis is still beyond the work capacity of very weak or completely paralyzed hip abductors. In such cases of extreme abductor muscle weakness, the pelvis will drop to the unsupported side even in the presence of a lateral trunk lean to the supported side. If lateral lean and pelvic drop occur during walking, the gait deviation is commonly referred to as a **Trendelenburg gait.** The lateral lean that accompanies the drop of the pelvis must be sufficient to keep the line of gravity within the supporting foot.

Whether a lateral trunk lean is due to muscular weakness or pain, a lateral lean of the trunk during walking still uses more energy than required for normal single-limb support and may result in stress changes within the lumbar spine if used over an extended time period. Use of a cane or some other assistive device offers a realistic alternative to the person with hip pain or weakness.

Use of a Cane Ipsilaterally

Pushing downward on a cane held in the hand on the side of pain or weakness should reduce the superimposed body weight by the amount of downward thrust; that is, some of the weight of HATLL would follow the arm to the cane, rather than arriving on the weightbearing hip joint. Inman and colleagues suggested that it is realistic to expect that someone can push down on a cane with approximately 15% of his body weight.[109] The proportion of body weight that passes through the cane will not pass through the hip joint and will not create an adduction torque around the supporting hip joint.



Example 10–4

Calculating Hip Joint Compression With a Cane Ipsilaterally

If our 825-N patient can push down on the cane with 15% of his body weight, 123.75 N of body weight (825 N × 0.15) will pass through the cane. The magnitude of HATLL is thereby reduced to 563.75 N (687.5 N – 123.75 N). If the gravitational force of HATLL

works through our estimated “normal” moment arm of 10 cm or 0.10 m (remember, the cane is intended to prevent the trunk lean), the torque of gravity is reduced to 56.38 Nm (563.75 N × 0.10 m). With a gravitational adduction torque of 56.38 Nm, the required force of the abductors acting through the usual 5 cm (0.05 m) moment arm is reduced to 1,127.6 N (56.38 Nm ÷ 0.05 m). The new hip joint reaction force using a cane *ipsilaterally* would then be:

1,127.6 N abductor joint compression
+ 563.75 N body weight (HATLL-cane) compression
1,691.35 N total joint compression

Total hip joint compression of 1,691.35 N calculated in Example 10–4 when a cane is us ed ipsilaterally provides some relief over the total hip joint compression of 2,062.5 N ordinarily experienced in unilateral stance. The total hip joint compression when the cane is used ipsilaterally is still greater, however, than the total joint compression of 1,031.25 N found with a compensatory lateral trunk lean (see Table 10–2). Although a cane used ipsilaterally provides some benefits in energy expenditure and structural stress reduction, it is not as effective in reducing hip joint compression as the undesirable lateral lean of the trunk. *Moving the cane to the opposite hand, however, produces substantially better results.*

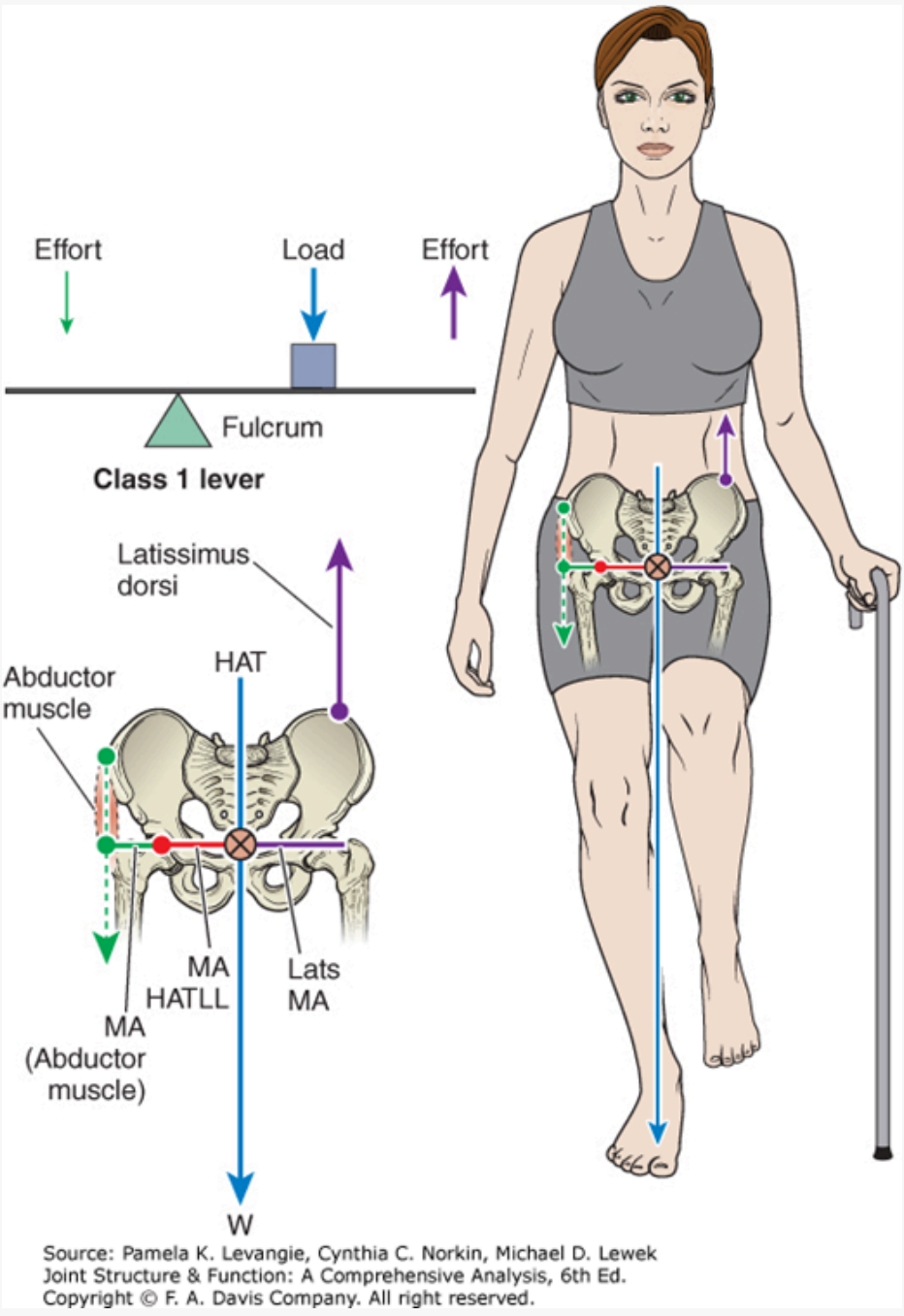
Use of a Cane Contralaterally

When the cane is moved to the side opposite the painful or weak hip joint, the reduction in the magnitude of HATLL is the same as it is when the cane is used on the same side as the painful hip joint; that is, the superimposed body weight passing through the weightbearing hip joint is reduced by approximately 15% of body weight. However, the cane is now substantially farther from the painful supporting hip joint (Fig. 10–39) than it would be if the cane is used on the same side; that is, in addition to

relieving some of the superimposed body weight, the cane is now in a position to assist the abductor muscles in providing a counter torque to the torque of gravity.[135] A classic description of the benefit of using a cane in the hand opposite to the hip impairment presumes that the downward force on the cane acts through the full distance between the hand and the weightbearing (impaired) hip joint.[135] We will first look at an example using the classic analysis and then determine how this analysis might be misleading.

FIGURE 10-39

When a cane is placed in the hand opposite the painful supporting hip, the weight passing through the right hip is reduced, and activation of the left latissimus dorsi provides a counter torque to that of HATLL and diminishes the need for a contraction of the right hip abductors. The moment arm (MA) of the cane is erroneously estimated to be 50 cm, whereas the moment arm of the more relevant latissimus dorsi is estimated to be 20 cm.



According to the classic analysis of the value of a cane in the opposite hand in Example 10–5, the hip joint reaction force would be due exclusively to body weight (563.75 N). This is, of course, an improvement over our calculated total hip compression with a lateral lean (1,031.25 N) and an even greater improvement over joint compression in

normal unilateral stance (2,062.5 N) for a person weighing 825 N (see Table 10–2).

Example 10–5

Classic Calculation of Hip Joint Compression With a Cane Contralaterally

Our sample 825-N patient has a superimposed body weight (HATLL) of 687.5 N, of which 123.75 N (W × 0.15) passes through the cane. Consequently, 563.75 N of body weight will pass through the right stance hip joint and the gravitation adduction torque will be:

$$\text{HATLL torque}_{\text{adduction}}\text{: } 563.75\ \text{N} \times 0.10\ \text{m} = 56.38\ \text{Nm}$$

The downward force on the cane of 123.75 N acts through an estimated moment arm of 50 cm (0.5 m) between the cane in the left hand and the right weightbearing hip joint (see Fig. 10–37). The cane, therefore, would generate an opposing abduction torque as follows:

$$\text{Cane torque}_{\text{abduction}}\text{: } 123.75\ \text{N} \times 0.5\ \text{m} = 61.88\ \text{Nm}$$

The torque around the right stance hip produced by a cane in the left hand (61.88 Nm) exceeds the torque produced by the remaining weight of HATLL (56.38 Nm). Because the gravitational torque (HATLL) may be underestimated, let us assume that the gravitational adduction torque and the counter torque provided by the cane offset each other. If the cane completely offset the adduction effect of gravity, there would be no need for hip abductor muscle force. The total hip joint compression in unilateral stance when a cane is used in the opposite hand would then be:

$$\begin{array}{r} 0\ \text{N abductor joint compression} \\ +\ 563.75\ \text{N body weight (HATLL-cane) compression} \\ \hline 563.75\ \text{N total joint compression} \end{array}$$

As explained in the text, however, this classic calculation is misleading and does not agree with experimental data.

Unfortunately, the classic treatment of biomechanics of cane use appears to overestimate substantially the effects of the cane. Krebs and colleagues (monitoring patients with an instrumented hip prosthesis) found reductions in peak pressure magnitudes of 28% to 40% during cane-assisted gait.[136] Although they reported

pressures rather than forces, these values do not match the nearly 75% reduction in force that the classic calculation would indicate. Furthermore, Krebs and colleagues found a 45% reduction in gluteus medius EMG, not an elimination of activity.[136] The discrepancy in the classic analysis and laboratory and modeling data can be resolved by examining *how* the force applied to the cane by a person provides a counter torque to gravity. Although this is conjectural, we propose that the force of the downward thrust on the cane arrives on the pelvis through an equivalent contraction of the latissimus dorsi muscle (123.75 N or 15% of body weight), which is necessary during cane use to maintain the shoulder in extension, and has an attachment to the pelvis on the posterior iliac crest, lateral to the erector spinae.[14] Given this attachment site, the line of pull of the muscle can be approximated to have a point of application on the pelvis above the ipsilateral acetabulum (20 cm or 0.20 m from the right hip joint axis). If the adduction torque around the right weightbearing hip continued but was of diminished magnitude because of the counter torque provided by the latissimus dorsi (123.75 × 0.20m, or 24.75 Nm), contraction of the right hip abductors would still be needed to offset the remaining 31.63 Nm of adduction torque. Acting through their 0.05-M moment arm, the right hip abductors would need to contract with a force of 632.6 N. The total joint compression would be estimated as 1,193.35 N (see Table 10–2). The hypothesized action of the latissimus dorsi on the side of the cane could account for the difference in compressive forces between classic calculations, with the cane used on the contralateral side and actual force measured in vivo.



Patient Application 10–6

Gabriella has osteoarthritic changes in her left hip that, if sufficient, can be a source of her pain. Furthermore, she has evidence of a labral tear, lateral hip pain, and a lean

during left stance that may be at least partially attributable to degenerative changes in the gluteus medius/minimus tendons and trochanteric bursa inflammation. There is little doubt that Gabriella can benefit from reducing the demands on her left hip abductor muscles. Reduction in demand may both minimize aggravation of any tears or inflammation and reduce hip joint compressive force that, with her hip dysplasia, are likely to be carried over a reduced area of articular contact. Using a cane in her right hand would be appropriate. Although a lateral lean actually reduces the demands on the hip abductors a bit more, the energy expenditure and structural stress on the spine must be considered.

Life Span and Clinical Considerations

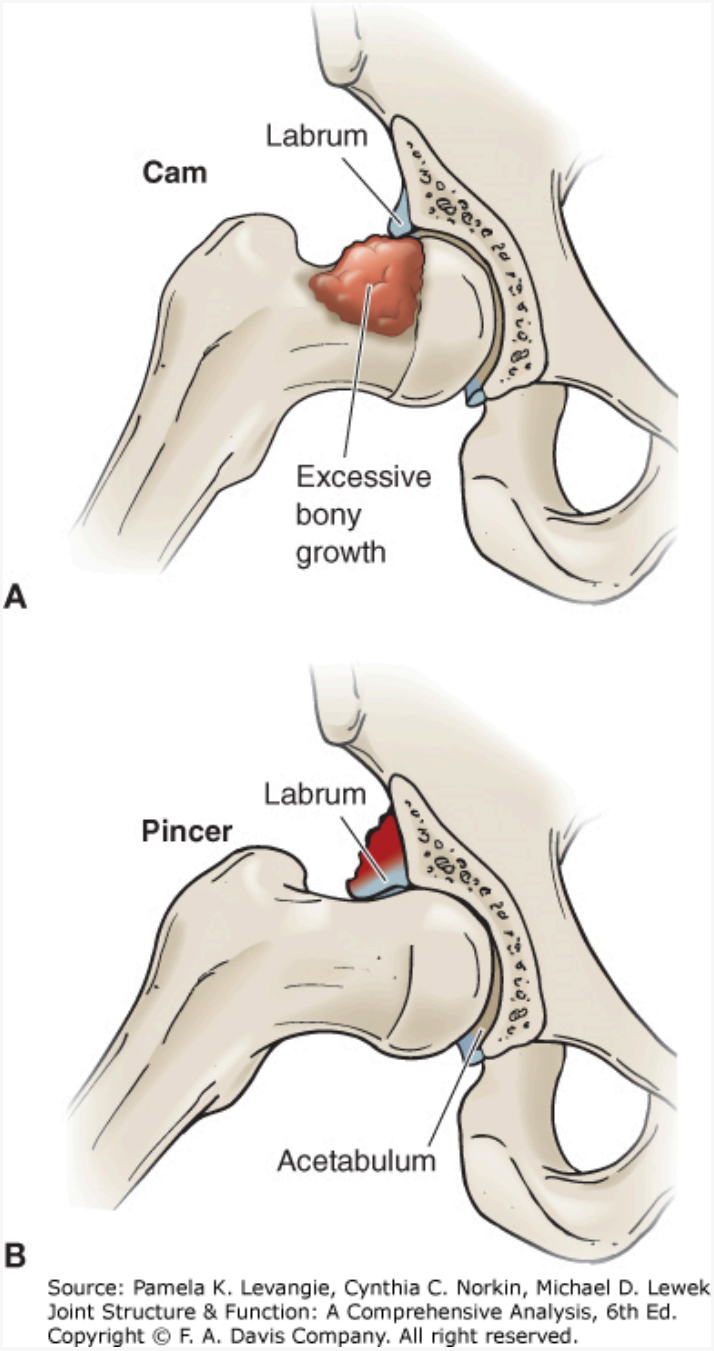
The very large active and passive forces crossing the hip joint make the joint's structures susceptible to wear and tear of normal components and to failure of weakened components. Small changes in the biomechanics of the femur or the acetabulum can result in increases in passive forces above normal levels or in weakness of the dynamic joint stabilizers. Some of the more common problems and the underlying mechanisms are discussed in this section.

Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is described as the dysfunctional abutment of the proximal femur and the acetabulum (Fig. 10–40). The result of such impingement causes pain and can lead to progressive degenerative changes in the hip joint, specifically the labrum. The causes of femoroacetabular impingement are thought to be multifactorial and the structure and deviant anatomy of the proximal hip joint are thought to be the primary causes of impingement. Femoroacetabular impingement has been classified as having one of two origins, each with its own etiologic and pathoanatomical characteristics.

FIGURE 10-40

A. Cam impingement occurs as a result of an abnormal widening of the femoral neck that leads to the abutment of the anterior superior labrum and the articular cartilage of the acetabulum during flexion or abduction of the hip. **B.** Pincer impingement occurs as a result of an over coverage of the acetabulum on the femoral head, causing compression of the superior labrum between the acetabular rim and the femoral head-neck junction with flexion or abduction.



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Cam impingement is thought to originate from what is described as a pistol-grip deformity of the femoral neck.[137] The deformity is likened to a pistol-grip in that the femoral neck fails to taper as the femoral head merges laterally into the femoral neck (Fig. 10–40A). The origin of the deformity is largely unknown, but researchers have

suggested that it may evolve from a subtle and asymptomatic slippage and resultant calcification of the proximal femoral epiphysis.[138] Others have suggested that it is the body's natural and protective response to strengthen a vulnerability to stresses across the femoral neck. Radiographs taken from a lateral frog-leg angle may identify a cam deformity in which the junction of the femoral head and neck becomes indistinguishable. This thickening leads to poor clearance of the femur, specifically the femoral head-neck junction as the hip joint flexes or abducts. The abnormal junction or cam causes undersurface wear and tear to the anterosuperior region of the labrum and the adjacent articular cartilage of the acetabulum. Histological observation is distinguished by a lack of inflammatory cell markers and by the appearance of a chronic degenerative process. Cystic changes, disorganization, and fraying of the cartilaginous labral structures as well as fibrillation, malacia, and balding of the articular cartilage of the anterosuperior acetabulum has been consistently observed among those diagnosed with cam impingement.[138]

Pincer impingement in femoroacetabular impingement (in contrast to cam impingement) is caused by aberrations of the acetabulum. Patients presenting with pincer-type impingement may demonstrate greater coverage, or overhang, of the acetabulum on the femoral head due to excessive retroversion of the acetabulum, or a deeper acetabular fossa, referred to as *coxa profunda* (**Fig. 10–40B**).[139,140] The excessive coverage of the femoral head causes compression of the superior labrum between the acetabular rim and the femoral head/neck in abduction.[139,141] The condition is analogous to primary subacromial impingement of the shoulder, in which a hooked acromion invades the subacromial space and creates a mechanical impingement. Similarly, in pincer-type impingement of the hip, the over coverage of the femoral head

by the acetabulum leads to a mechanical impingement of the superior labral-acetabular complex.[137] Repetitive impingement of the labrum may result in ossification of the labrum that may further worsen the over coverage of the femoral head.[141] This calcification may further lead to articular lesions to the femoral head and acetabulum. Researchers have suggested that the mechanical blocking of the natural arthrokinematics of hip flexion leads to abnormal posterior displacement of the femoral head as the hip is forced into greater flexion angles.[137] This would explain articular erosion to the posterior-inferior acetabulum and the posterior-medial surface of the femoral head associated with pincer-type impingement.

Although femoroacetabular impingement is typically considered to result from either cam or pincer deformities, current research indicates that it may be difficult and erroneous to classify femoroacetabular impingement into one category versus the other.[140] Researchers have studied surgically dislocated hips with radiological evidence of either a pistol-grip deformity (cam impingement) or coxa profunda abnormality (pincer impingement). Although the imaging results indicated classification into one of the two categories, the surgical observation revealed that the majority of the studied hips had a combination of both abnormal findings. This suggests that the classification scheme is more complex and that many patients may in fact have varying degrees of both types of impingement.[137]

Aberrant anatomical findings, however, may not always present in those with symptoms of femoroacetabular impingement. Ito and colleagues studied subjects with inguinal pain related to femoroacetabular impingement who had significant loss of ROM into flexion, abduction, medial rotation, and lateral rotation without findings of

dysplasia.[138] This suggests that other factors related to hip ROM deficits, including capsuloligamentous or musculotendinous restriction and arthritic changes, may exist as etiologic factors of femoroacetabular impingement and should be considered in the evaluation and treatment of the condition.

Labral Pathology

Labral pathology is often discussed synonymously with femoroacetabular impingement, as the two conditions may often coexist. As previously discussed, the mechanisms of labral pathology are associated with femoroacetabular impingement. Burnett found 95% of patients with labral tears confirmed by hip arthroscopy had clinical signs of femoroacetabular impingement.[142] Femoroacetabular impingement, however, is not the only bony abnormality associated with a torn labrum. Guerva and colleagues reported that in comparison to the unaffected side, hips with symptomatic labral tears displayed significant differences in several radiologic measures, including decreased center edge angle, acetabular retroversion, and coxa vara compared with the unaffected side.[139] Collectively, these abnormalities lead to a lateral and superior displacement of the femoral head and predispose to an anterior superior impingement of the acetabulum and femoral neck.[138,139]

The labrum is a legitimate source of pain. Kim and colleagues identified that the labrum is innervated tissue with the capability of causing and referring pain in patients with labral pathology.[7] The onset of labral pathology may be caused either by a single traumatic event or more commonly by the cumulative effects of microtrauma. A traumatic labral injury may be the result of a rapid and forceful rotation of the hip. Although this may occur in open-chain lower limb function, such trauma is more often a

result of long axis rotation in a closed-chain position, such as commonly seen in athletics that may require a rapid change of direction with planting and pivoting on a fixed leg. Despite the common mechanism of injury involving sport-related activities, labral damage can occur across the life span. Another mechanism for traumatic labral injury may be the result of a dislocating force of the hip. Although the hip is an inherently stable joint, a blunt force to a flexed hip, such as during a fall on the knee may lead to a posterior dislocation and subsequent injury to the posterior labrum.

Cumulative microtrauma appears to be the more common mechanism of injury to the labrum. As evidenced by the absence of inflammatory markers in symptomatic labral tears, it appears that the labrum is vulnerable to a progressive degenerative process that leads to pathology.[138,139] As discussed earlier, the repetitive mechanical stress induced by femoroacetabular impingement contributes to nontraumatic labral injury. Thus, the morphologic factors that characterize femoroacetabular impingement are also common in those patients with labral pathology. Collectively, these factors may ultimately lead to either an under coverage or an over coverage of the femoral head. Under coverage may also be the result of a shallow acetabulum or dysplastic changes to the femoral head-neck junction as described in cam impingement. This leads to disproportionate loading of the acetabular rim and labral structures. Under coverage may also be produced functionally through laxity or an acquired instability of the hip joint.

The role of the labrum as a key stabilizing component of the hip joint also helps explain the vulnerability to injury. Tension through the capsuloligamentous structures creates extreme strain through the anterior labrum. This is accentuated when the hip is moving

into positions of abduction and lateral rotation. Further tension may be exhibited on the capsulolabral structures while these movements are performed with combined extension of the hip joint.[143] These positions may be common in athletic movements. Thus throwing and swinging movements as well as quick planting and explosive changes in direction may lead to cumulative damage to the anterior capsulolabral complex. Athletes participating in baseball, golf, gymnastics, dance, and martial arts are particularly vulnerable to capsular and ligamentous redundancy (laxity) and labral injury. Crawford and colleagues re-created capsular redundancy and a simulated labral tear in cadaver specimens and found a 60% reduction of force needed to distract the hip joint.[143] Researchers suggest that a labral tear with capsular redundancy causes a loss in intra-articular pressure of the joint that ultimately compromises joint stability.

Arthrosis

Our understandings of more recently discovered pathologies associated with hip joint dysfunction are also helping us to understand the mechanisms and interrelationships to other pathologies of the hip. Femoroacetabular impingement has been implicated as a potential etiologic factor in the presence of **osteoarthritis (OA)** of the hip joint. Beck and colleagues described the process of articular deterioration as it relates to findings of cam impingement.[137] The presence of a labral tear has also been identified as an important risk factor in the development of OA of the hip.[58,144] Chronic synovial irritation, joint fluid leakage, and the relationship to micro-instability of the hip joint may contribute to abnormal joint loading and deterioration of the articular surfaces of the joint.

Regardless of the etiology, osteoarthritis is the most common painful condition of the

hip. It is defined as deterioration of the articular cartilage and to subsequent related changes in articular tissues.[145] OA of the hip is a leading cause of disability worldwide.[146] Hip OA affects 0.9% to 3.9% of the male population and 0.7% to 5.1% of females, and is particularly prevalent in middle and older adulthood.[147] Juhakoski and colleagues conducted a 22-year prospective study that attempted to identify the key risk factors associated with the onset of hip OA symptoms.[148] Many variables were analyzed, including body mass index, work history, injury history, participation in exercise programs, and history of smoking and alcohol use. Of these factors, a previous history of musculoskeletal injury and work history, including extensive physical and manual labor, were the only two factors predictive of hip OA. One study noted that more than 90% of hip osteoarthritis cases were attributed to anatomical abnormalities.[149] Changes may be due to subtle deviations present from birth, to tissue changes inherent in aging, to the repetitive mechanical stress of loading the body weight on the hip joint over a prolonged period, to impingement between the femur and labrum or adjacent acetabulum, or to interactions between these factors.[33,150,151] The factors most closely associated with idiopathic hip joint arthrosis are increased age and increased weight/height ratio.[152] Contrary to popular belief, repetitive weightbearing exercise, specifically running, was found to have no association between OA changes and running status among older subjects.[153]

The mechanism for cartilaginous degeneration in the hip joint is not clear-cut. In the absence of preexisting trauma or biomechanical abnormality, degenerative changes may be the result of inadequate joint forces, rather than excessive forces at the hip joint. Articular cartilage is nourished through joint movement and compression. When a person is inactive, nourishment to the articular cartilage is diminished and more

vulnerable to injury and deterioration. This would explain why there is little or no association between increased activity level with sports or recreational activities and hip arthrosis.[153,154] In fact, there is evidence to suggest that lower activity levels are associated with hip OA. Hirata and colleagues found that 40% of women diagnosed with hip OA were classified as physically inactive and spent less than 6 minutes per day in what would be considered moderately intense physical activity.[155] Rosemann and colleagues suggested that moderate activity can actually be preventive in the progression of OA symptoms.[156] Forces in excess of half body weight are needed to fully compress the femoral head into congruent contact with the dome of the acetabulum.[157] Using a number of other studies as a base, Bullough and associates hypothesized that we typically spend no more than 5% to 25% of our time in unilateral lower extremity weightbearing activities in which the load may be sufficient to compress the articular cartilage of the dome of the acetabulum.[157] Lower loads and infrequent high joint forces may be inadequate to maintain flow of nutrients and wastes through the avascular cartilage. The theory of inadequate compression as a contributing factor to hip joint degeneration is supported by the fact that the more common degenerative changes in the femur are at the periphery of the head and the perifoveal area, rather than at the superior primary weightbearing area.[82,157] The periphery of the head receives only about one third the compressive force of the superior portion of the head.[128] Conversely, the superior portion of the head is compressed not only in standing but is also in contact with the posterior acetabulum during sitting activities.[82] Yet, the area of the femoral head around the fovea is most commonly in the non-weightbearing acetabular notch and would undergo compression relatively infrequently. Wingstrand and colleagues proposed that excessive intra-articular fluid

from relatively benign synovitis or trauma may reduce articular congruence and the stabilizing effect of atmospheric pressure, resulting in micro instability and unfavorable cartilage loads.[53] This theory has been substantiated by evidence from Tarasevicius and colleagues, who demonstrated lower intra-capsular pressures to be related to the severity of hip OA.[158]

Fracture

To maintain integrity of the hip joint, the bony components must be of sufficient strength to withstand the forces that are acting around and through the hip joint. As noted in the section on the weightbearing structure of the hip joint, the vertical weightbearing forces that pass down through the superior margin of the acetabulum in both unilateral and bilateral stance create a bending force across the femoral neck (see Fig. 10–18). Normally the trabecular systems are capable of resisting the bending forces, but abnormal increases in the magnitude of the force or weakening of the bone can lead to bony failure. The site of failure is likely to be in areas of thinner trabecular distribution, such as the zone of weakness (see Fig. 10–20). Thomas and colleagues found the superior, lateral, and posterior portions of the femoral neck suffered the most significant amount of trabecular bone loss.[159] It was estimated that the women they studied experienced a 42% decrease in the trabecular density to this region over the age span of 50 years of adult life.[159] Crabtree and colleagues studied both patients and cadavers with a fracture to conclude that a loss in cortical bone, not cancellous bone (trabeculae), may be the source of the problem.[85] Although cancellous bone mass was similar for cases and controls in similar age groups, there was a 25% reduction in cortical bone mass in the fracture cases.[85] Thomas and colleagues reported similar findings in that there was cortical thinning of 37% to 49% in cases where fractures

occurred versus controls, but trabecular density was similar between groups.[159] The authors acknowledged, however, the importance of the trabecular infrastructure in resisting the deforming forces that cause a fracture, especially during a fall. Although cortical thinning is a more significant finding in cases of proximal hip fracture, the use of therapies and treatment directed toward increasing trabecular density may have a substantial impact on reducing the risk of fracture.[159]

Bony failure in the femoral neck is uncommon in the child or young adult, even with large applied loads. However, femoral neck fractures occur at the rate of about 98/100,000 people in the United States, with the average age at occurrence being in the 70s. It is estimated that three out of four hip fractures occur in an elderly population. Adults over the age of 75 are over five times more likely to sustain a hip fracture than those aged 65 to 74, and over 30 times more common than those aged 45 to 64.[160] There is a predominance of fractures in women, although this is certainly influenced by their greater longevity. Of middle-aged people, women actually suffer fewer hip fractures than men, although the fractures in this age group are usually attributable to substantial trauma.[161]

In 87% of cases of hip fracture among the elderly population, the precipitating factor appears to be moderate trauma such as that caused by a fall from standing, from a chair, or from a bed. There is consensus that hip fracture is associated with, but not exclusively due to, diminished bone density.[162] Bone density decreases about 2% per year after age 50 and trabeculae clearly thin and disappear with aging.[163] Cummings and Nevitt believed the exponential increase in hip fractures with age could not be accounted for by decreased bone density alone and proposed that the slowed gait

characteristic of the elderly may play an important part.[163] They contended that the slowing of gait makes it less likely that momentum will carry the body forward in a fall (generally onto an outstretched hand) and more likely that the fall will occur backward onto the hip area weakened by bone loss and no longer padded by the fat and muscle bulk of youth. Gait speed and characteristics have been shown to have a directional relationship to falls and subsequently may be meaningful in assessing the risk for a hip fracture. Individuals with higher gait speed and longer step length show greater bone mineral density and thus have less risk for femur fracture.[164] Researchers have identified clinical predictors that categorize females into a high- or low-risk group for proximal femur fracture to include gait speed as well as a tandem walk test that tests dynamic balance.[165]

Hip fracture will continue to receive considerable attention because of the high health-care costs of both conservative and operative treatment. Of all fall-related fractures, hip fractures cause the greatest number of deaths and lead to the most severe health problems and reduced quality of life. Hip fractures alone account for nearly half of fractures leading to mortality and 4% of all injury-related deaths.[160] Mortality within the year following a femoral neck fracture has been estimated to be between 21% to 24%.[166] Although mortality rates among the elderly are high, younger patients are more vulnerable to complications after a fracture. This may include nonunion of the fracture and femoral head osteonecrosis, which occurs in 23% of cases.[167] Not only are these conditions painful, but malunion of the fracture can also lead to joint instability or cartilaginous deterioration (or both) as a result of poorly aligned bony segments. Although the femoral head may receive some blood supply via the ligament of the femoral head, an absent or diminished supply through the ligament of the head (as

occurs with aging) means reliance on anastomoses from the circumflex arteries. This circumflex arterial supply may be disrupted by femoral neck fracture, which leaves the femoral head susceptible to avascular necrosis and necessitates replacement of the head of the femur with an artificial implant. Due to high complication and mortality rates, careful consideration in the management of patients with hip fracture is necessary. Several factors, including age, level of functional activity, and comorbidities, must be considered in the effective management of these conditions.[168]

Patient Application 10–7

Although we have probably covered the potential sources of Gabriella's problems fairly well at this point, it is worth taking note of the most likely underlying causes. Gabriella's persisting coxa valga and femoral anteversion are the likely sources of many, if not all, of her problems. With a valgus and anteverted femur, the articular contact within Gabriella's hip joint is substantially reduced, increasing the contact pressures and locating them atypically within the acetabulum and on the head of the femur. This is consistent with her complaints of instability and may also have contributed to her labral tear. Wenger and colleagues found that over half of the hips studied that had an identifiable labral tear also had a coxa valga deformity of the femur.[32] Both her hip dysplasia and her history of dance, gymnastics, and years of working at floor level with young children have exacerbated the likelihood of anterior impingement of the labrum, resulting in probable labral tear and chondral lesions. The mechanical disadvantage to her hip abductors is likely to contribute to overuse and degenerative lesions of the abductor mechanism, further affecting abductor function and resulting in what might be referred to either as a gluteus medius or antalgic gait. It appears that Gabriella's hip symptoms have been the result of an ongoing and progressive phenomenon that is leading to the early stages of joint degeneration. It likely began during repetitive movements and activities that required excessive and extremes of ROM. These repetitive movements, combined with altered hip mechanics due to her hip dysplasia, resulted in chronic and repeated trauma that damaged her anterior capsule and labrum. The degradation and ultimate failure of her capsule and labrum likely contributed to her feeling of instability of the hip. A labral tear causes a leakage of joint fluid and disrupts the

"seal" of the hip joint that creates intra-articular pressure.[143] This pressure helps stabilize the hip joint. Combined, all of these factors make an individual more susceptible to osteoarthritis. If Gabriella continues without intervention it is likely that her pain will worsen and her joint may continue to deteriorate. This may lead to an eventual total joint replacement. However, Gabriella is not without treatment options for her current condition. Medications and injections, as well as physical therapy, remain options for the conservative management of Gabriella's symptoms.[169] The emergence of hip arthroscopy has resulted in new treatment options for patients with similar symptoms, as long as more advanced osteoarthritis is not present. For example, labral pathology and tears to the abductor mechanism have shown to be managed successfully through minimally invasive techniques of arthroscopy. Philippon and colleagues reported excellent results that included a 24% improvement in self-reported functional outcomes and a median satisfaction score of 9 out of a possible 10 for patients 2 years after hip arthroscopy.[170] Addressing her dysplastic hip findings may also suggest a surgical option. However, the procedures to correct her acetabular and femoral abnormalities would require much more aggressive and invasive surgery with a likely protracted recovery period. Although there has been no concrete evidence to suggest that any of these surgical approaches and techniques prevent the progression of osteoarthritis of the hip, surgery may improve the patient's associated biomechanical faults or abnormities and may eliminate the diseased source of pain.

It used to be thought that patients like Gabriella would likely continue to progress in the process of degeneration of the hip joint until the pain and functional limitations would eventually necessitate a need for joint replacement surgery. Our understanding of this process is helping to discover more effective, conservative approaches to the management of such conditions. When conservative measures have failed, current surgical approaches and the focus of future treatment options in bioengineering and restorative techniques for the articular surfaces of joints are encouraging in that viable and effective treatment options exist and will evolve into even better treatment options for patients like Gabriella.[171]

Summary

- The normal hip joint is well designed to withstand the large forces that act through

and around it, assisted by the trabecular systems, labrum, cartilaginous coverings, muscles, and ligaments.

- Alterations in the direction or magnitude of forces acting around the hip create abnormal concentrations of stress that predispose the joint structures to injury and degenerative changes. The degenerative changes, in turn, can create additional alterations in function that not only affect the hip joint's ability to support the body weight in standing, in locomotor activities, and in other activities of daily living but may also result in adaptive changes at more proximal and distal joints.

- Consequently, the reader must understand both the dysfunction that might occur at the hip *and* the associated dysfunctions that may result in or from dysfunction elsewhere in the lower extremity and spine.

The remaining chapters of this text will focus not only on primary dysfunction at a joint complex but also on associated dysfunction related to proximal and distal joint problems.

Study Questions

1. Which side of the femoral neck and which side of the femoral shaft are subjected to compressive stresses during weightbearing? How does the bone respond to these stresses?

2. What is the primary weightbearing area of the femoral head? Of the acetabulum? Where are degenerative changes most commonly found in the femoral head and acetabulum?

3. Describe why using a cane on the side opposite hip joint pain or weakness is more effective than using the cane on the same side.

4. Demonstrate how variations in the angle of inclination affect the moment arm of the hip abductors by drawing the following: a normal angle of inclination at the hip, the angle in coxa vara, and the angle in coxa valga. Please include the action line and the moment arm of the hip abductors in the diagram.

5. Describe what would happen to the pelvis in left unilateral stance when the left hip abductors are paralyzed. How is equilibrium maintained in this situation?

6. Describe motion at the right and left hip joints and at the lumbar spine during hiking of the pelvis in right limb stance, assuming that the person is to remain upright.

7. Contrast the close-packed versus maximally congruent position for the hip joint.

8. Calculate the minimum joint reaction force (total hip joint compression) at the right hip joint that would occur for a 200-lb person standing symmetrically on both legs versus one leg (assuming a gravitational moment arm of 4 in. and the abductor muscle moment arm of 2 in.).

9. Under what circumstances does the hip joint participate as part of an open chain? As part of a closed chain?

10. What bony abnormality or abnormalities of the femur or pelvis predispose the hip joint to the possibility of dislocation? Why?

11. Which structures at the hip joint, given their location, appear likely to limit the extremes of motion in flexion? In extension? In lateral rotation? In medial rotation? In abduction and adduction?

12. Which muscles of the hip joint are affected by knee joint position? Which position of the knee makes these muscles less effective at the hip joint?

13. If someone were in a unilateral stance on the left limb, what hip joint motion would result from forward rotation of the pelvis?

14. If a person has a painful right hip, in which direction should he or she lean his or her trunk to reduce the forces on the right hip during right unilateral support? Explain the reasons for your answer.

15. What position does the hip joint tend to assume when there is joint pain? Why is this?

16. Identify several factors that might predispose someone to a femoral neck fracture.

17. How does the femoral head receive its blood supply? What problems might jeopardize that supply?

18. Relate femoral anteversion to medial femoral torsion.

19. Under what circumstances might the hip adductors work synergistically with the hip abductors?

20. Why is the acetabular notch nonarticular? In what ways does this serve hip joint function?

21. Explain the differences between cam and pincer-type impingement.

22. Describe how the presence of femoroacetabular impingement may lead to osteoarthritis of the hip.

23. What factors contribute to stability of the hip joint?

24. How are abnormalities at the femur and acetabulum related to femoroacetabular impingement? Labral tears? Fractures?

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Chapter 11: The Knee

Elizabeth Wellsandt; David S. Logerstedt; Lynn Snyder-Mackler

Anatomy Overview

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MUSCLE ACTIONS OF THE KNEE JOINT		
TABLE KEY: Primary muscle Secondary muscles		
Sagittal Plane	Flexion	Extension
	Semimembranosus Semitendinosus Biceps femoris (long and short heads) Sartorius Gracilis Popliteus Gastrocnemius	Vastus lateralis Vastus medialis Vastus intermedius Rectus femoris
Transverse Plane	Medial Rotation	Lateral Rotation
	Popliteus Semimembranosus Semitendinosus	Biceps femoris

	Sartorius Gracilis	

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KNEE MUSCLE ATTACHMENT		
Muscle	**Proximal Attachment**	**Distal Attachment**
Rectus femoris	AIIS and ilium superior to acetabulum	Quadriceps tendon to patella and ultimately tibial tuberosity
Vastus lateralis	Greater trochanter and lateral lip of linea aspera (femur)	Quadriceps tendon to patella and ultimately tibial tuberosity
Vastus medialis	Intertrochanteric line and medial lip of linea aspera (femur)	Quadriceps tendon to patella and ultimately tibial tuberosity
Vastus intermedius	Anterior and lateral surfaces of shaft of femur	Quadriceps tendon to patella and ultimately tibial tuberosity
Sartorius	ASIS and superior part of notch inferior to it	Superior part of medial surface of tibia
Gracilis	Body and inferior ramus of pubis	Superior part of medial surface of tibia
Tensor faciae latae	ASIS; anterior part of iliac crest	Iliotibial (IT) band to lateral condyle of tibia
Semitendinosus	Ischial tuberosity	Medial surface of superior part of tibia

Semimembranosus	Ischial tuberosity	Posterior aspect of medial condyle of tibia; reflected attachment forms oblique popliteal ligament (to lateral femoral condyle)
Biceps femoris	Long head: Ischial tuberosity Short head: Linea aspera and lateral supracondylar line of femur	Lateral side of head of fibula
Gastrocnemius	Lateral head: Lateral aspect of lateral condyle of femur Medial head: Popliteal surface of femur; superior to medial condyle	Posterior surface of calcaneus via calcaneal tendon
Popliteus	Lateral surface of lateral condyle of femur and lateral meniscus	Posterior surface of tibia, superior to soleal line

Introduction

The knee complex is one of the most often injured regions in the human body and the most frequently injured joint during sports activities.[1] The myriad of ligamentous

attachments, along with numerous muscles crossing the joint, provide insight into the joint's complexity. This anatomical complexity allows for elaborate interplay between the simultaneous mobility and stability roles required by the knee complex. The knee joint works in conjunction with the hip, ankle, and joints in the feet to support the body's weight during static erect posture. Dynamically, the knee complex is responsible for moving and supporting the body during a variety of both routine and difficult activities. The knee's complex structure and multiple functions reflect its major roles in stability and mobility that are needed to complete many human movements.

The knee complex is composed of two distinct articulations located within a single joint capsule: the tibiofemoral joint and the patellofemoral joint. The **tibiofemoral joint** is the articulation between the distal femur and the proximal tibia. The **patellofemoral joint** is the articulation between the distal femur and the posterior patella. Although the patella enhances the effectiveness of the tibiofemoral mechanism, the characteristics, responses, and challenges of the patellofemoral joint are uniquely different from the tibiofemoral joint, warranting separate attention within this chapter. Also, despite its adjacent location, the superior tibiofibular joint is not considered to be a part of the knee complex because it is not contained within the knee joint capsule. Because it is functionally related to the ankle joint, it will be discussed in Chapter 12.

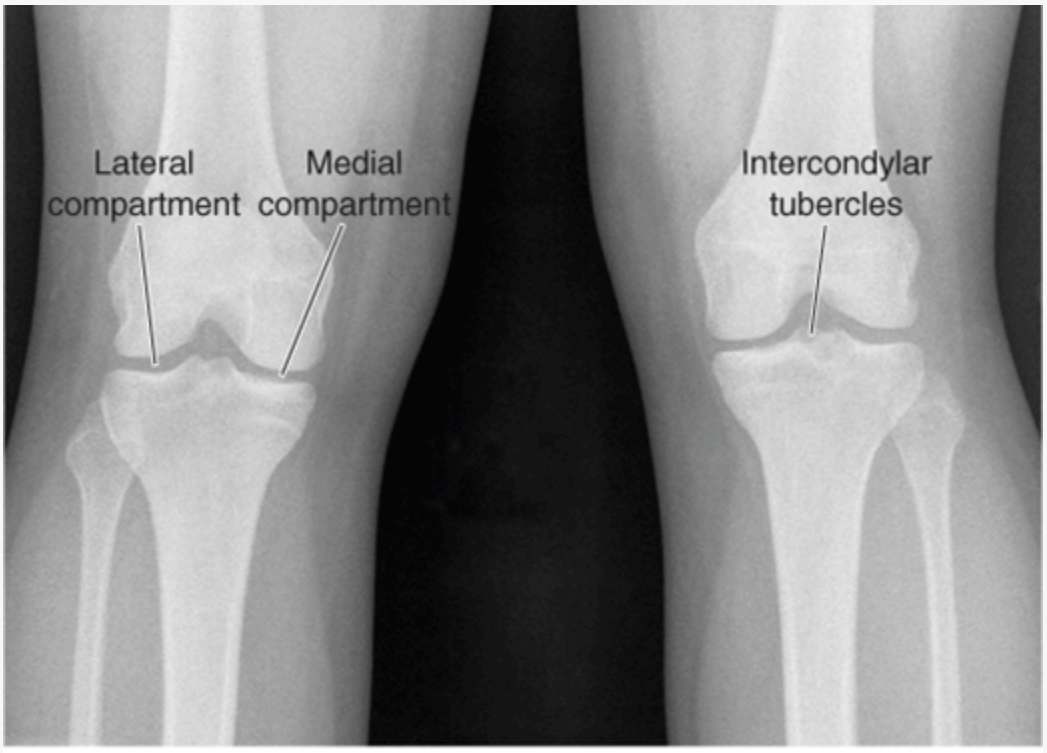
Tibiofemoral Joint Structure and Function

The tibiofemoral, or knee, joint is a double condyloid joint with three angular (rotary) degrees of freedom. Flexion and extension occur in the sagittal plane around a medial-lateral (frontal) axis through the epicondyles of the distal femur, medial/lateral

(internal/external) rotation occurs in the transverse plane about a longitudinal axis through the lateral side of the medial tibial condyle, and abduction and adduction occur in the frontal plane around an anteroposterior (sagittal) axis.[2–4] The double condyloid knee joint is defined by its medial and lateral articular surfaces, also referred to as the **medial** and **lateral compartments** of the knee (Fig. 11–1).

FIGURE 11-1

A weightbearing knee radiograph showing the medial and lateral tibiofemoral knee compartments. The medial and lateral compartments are separated centrally by the intercondylar tubercles.



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Structure

A full understanding of the tibiofemoral joint's common functions and dysfunctions requires careful examination of its articular surfaces, ligaments, and musculotendinous structures, along with an understanding of their relationship to each other.

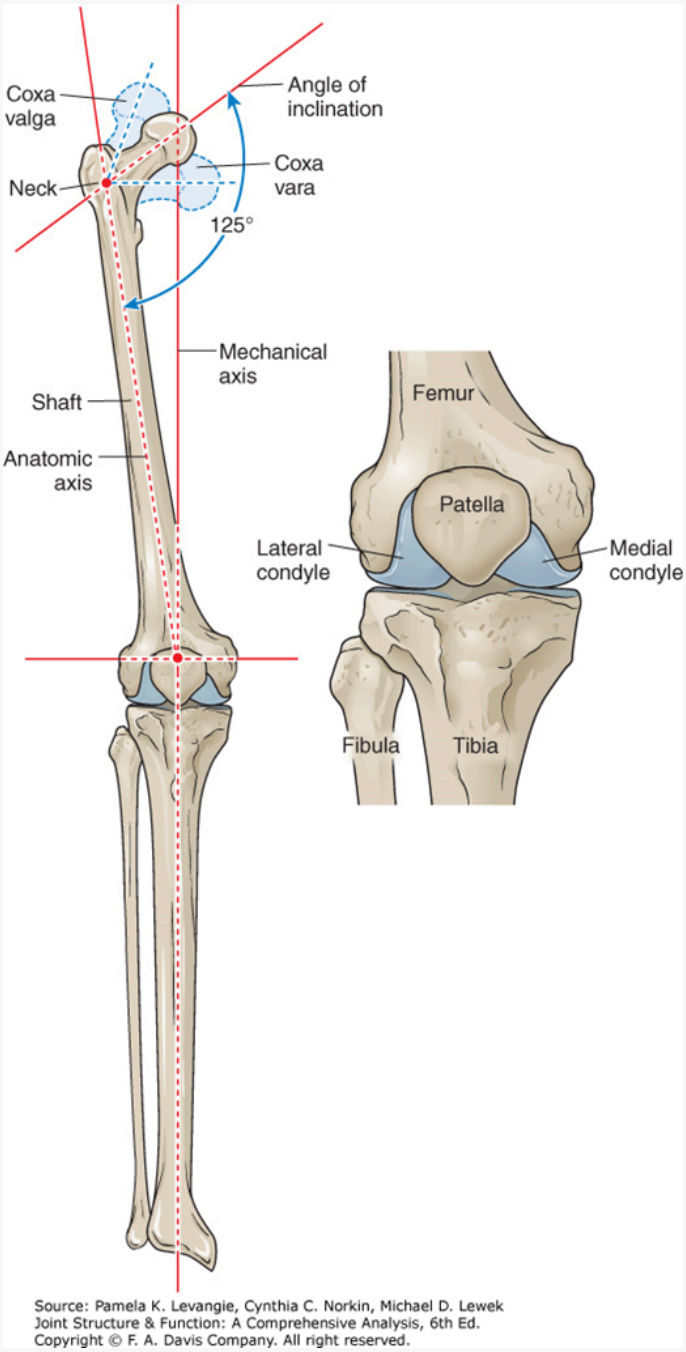
Femur

The proximal articular surface of the knee joint is composed of the large medial and lateral condyles of the distal femur (**Fig. 11–2**). The medial condyle is larger than the lateral condyle and has a greater radius of curvature.[5] If the shaft of the femur were aligned vertically, then the longer medial condyle would extend farther distally than the lateral condyle. However, because of the natural oblique angle of the shaft of the femur, the distal ends of the femoral condyles remain essentially horizontal. In the sagittal plane, the condyles have a convex shape, with a smaller radius of curvature posteriorly (**Fig. 11–3**).[3,6] In the frontal plane, the distal femur has very little curvature as a whole: however, both the medial and lateral condyles individually exhibit slight frontal plane convexity. There are also important size and positional differences between the medial and lateral femoral condyles. For example, the lateral femoral condyle is shifted anteriorly with respect to the medial femoral condyle.[7] This anterior shift of the lateral femoral condyle coincides with a tibial articular surface of the lateral femoral condyle that is shorter from anterior to posterior than the tibial articular surface of the medial femoral condyle (**Fig. 11–4**). The difference in anterior to posterior size of the condyles can be appreciated when examining the femur from an inferior view (Fig. 11–4, top). Although the lateral femoral condyle appears at first glance to be longer, when the patellofemoral surface is excluded it is evident that the medial tibial surface of the femur extends farther anteriorly than the lateral condyle. The two condyles are

separated inferiorly by the **intercondylar notch** through most of their length but are joined anteriorly by an asymmetric, shallow groove called the **femoral sulcus, patellar groove,** or **patellar surface** (see Figs. 11–2B and 11–4).

FIGURE 11-2

Because of the obliquity of the shaft of the femur, the more prominent medial condyle is aligned horizontally with the lateral femoral condyle.

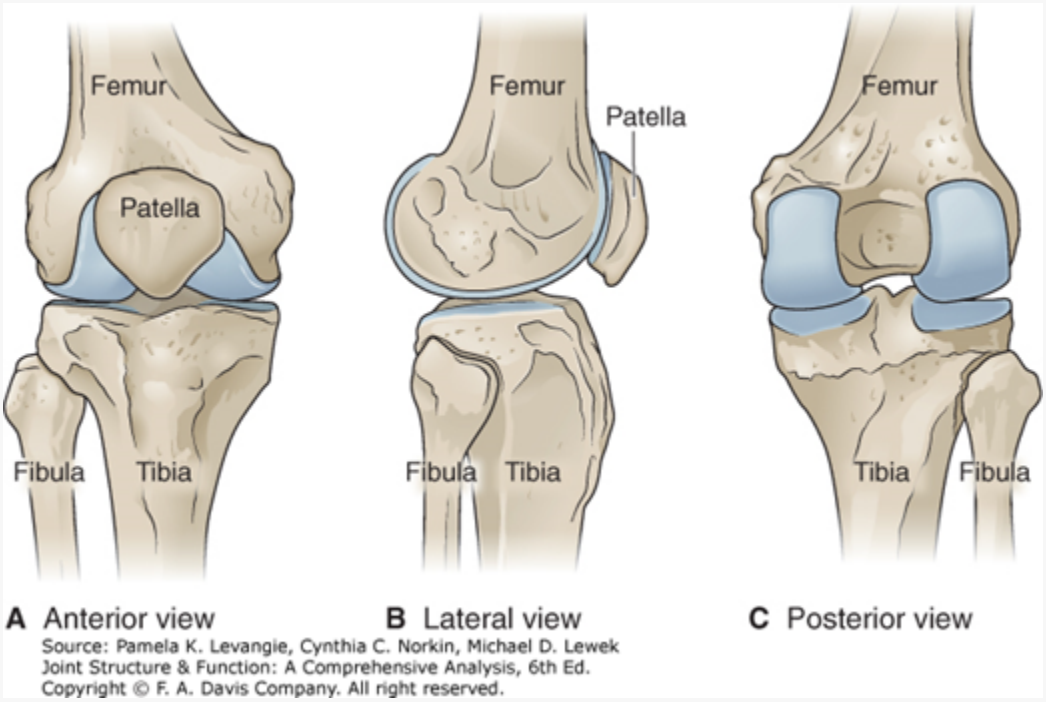


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FIGURE 11-3

A. Viewed anteriorly, the separate medial and lateral compartments of the tibiofemoral joint can be viewed with its slight femoral convexity.

B. In this lateral view of the tibiofemoral joint, it can be seen that the anteroposterior convexity of the condyles is not consistently spherical, having a smaller radius of curvature posteriorly. The tibial plateau overhangs the shaft of the tibia posteriorly and is inclined posteriorly 7° to 10°. **C.** Viewed posteriorly, the large femoral condyles can be easily distinguished.

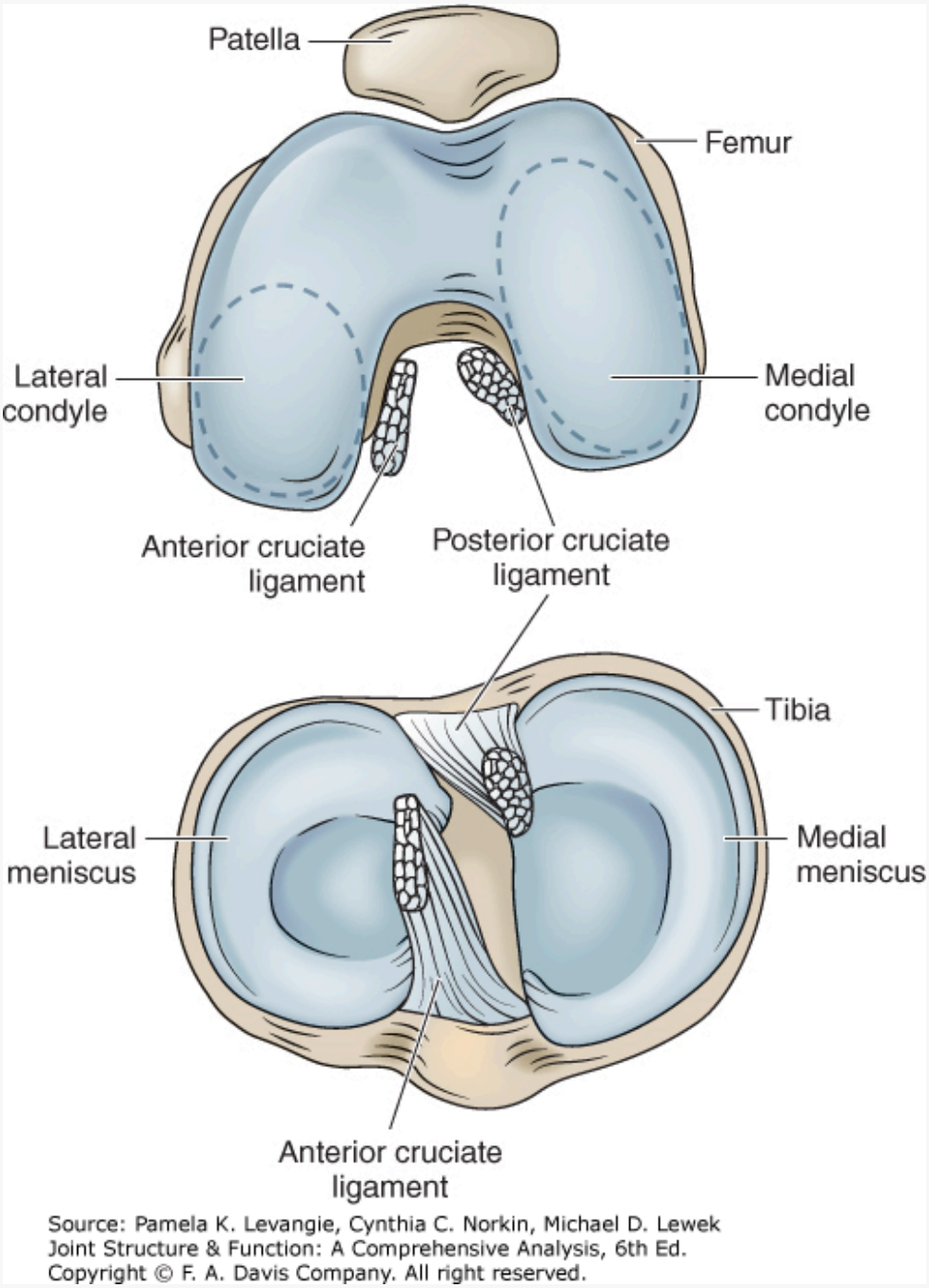


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FIGURE 11-4

Top: Inferior view of femur. The intercondylar notch separates the medial and lateral femoral condyles where tibial articulation occurs. Articulating surfaces for the lateral and medial patellar facets are separated by the femoral sulcus. Bottom: Superior view of tibial

plateau. The larger medial plateau is separated from the smaller lateral surface by the intercondylar ridge.



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Tibia

The large convex femoral condyles sit on the relatively flat tibial condyles. The

asymmetrical medial and lateral tibial condyles or plateaus compose the distal articular surface of the tibiofemoral joint (Fig. 11–4, bottom). Similar to the distal femur, the medial tibial plateau is longer in the anteroposterior direction than the lateral tibial plateau; however, the lateral tibial articular cartilage is thicker than the articular cartilage of the medial tibial plateau.[6,8,9] The proximal tibia is larger than the tibial shaft and, consequently, overhangs the tibial shaft posteriorly (see Fig. 11–3). Associated with the posterior overhang is approximately a 7° to 10° posterior slope of the tibial plateaus, making it conducive for flexing the tibiofemoral joint.[6,7] The medial and lateral tibial condyles are separated by a roughened area and two bony spines called the **intercondylar tubercles** (see Figs. 11–1 and 11–2B). These tubercles become lodged in the intercondylar notch of the femur when the knee is in an extended position. The predominantly flat tibial plateaus, with a slight convexity at the anterior and posterior margins, in combination with the convex femoral condyles, create a bony architecture that is not conducive for joint stability.[3] Because the tibiofemoral joint lacks bony stability, accessory joint structures **(menisci)** are necessary to improve joint congruency.

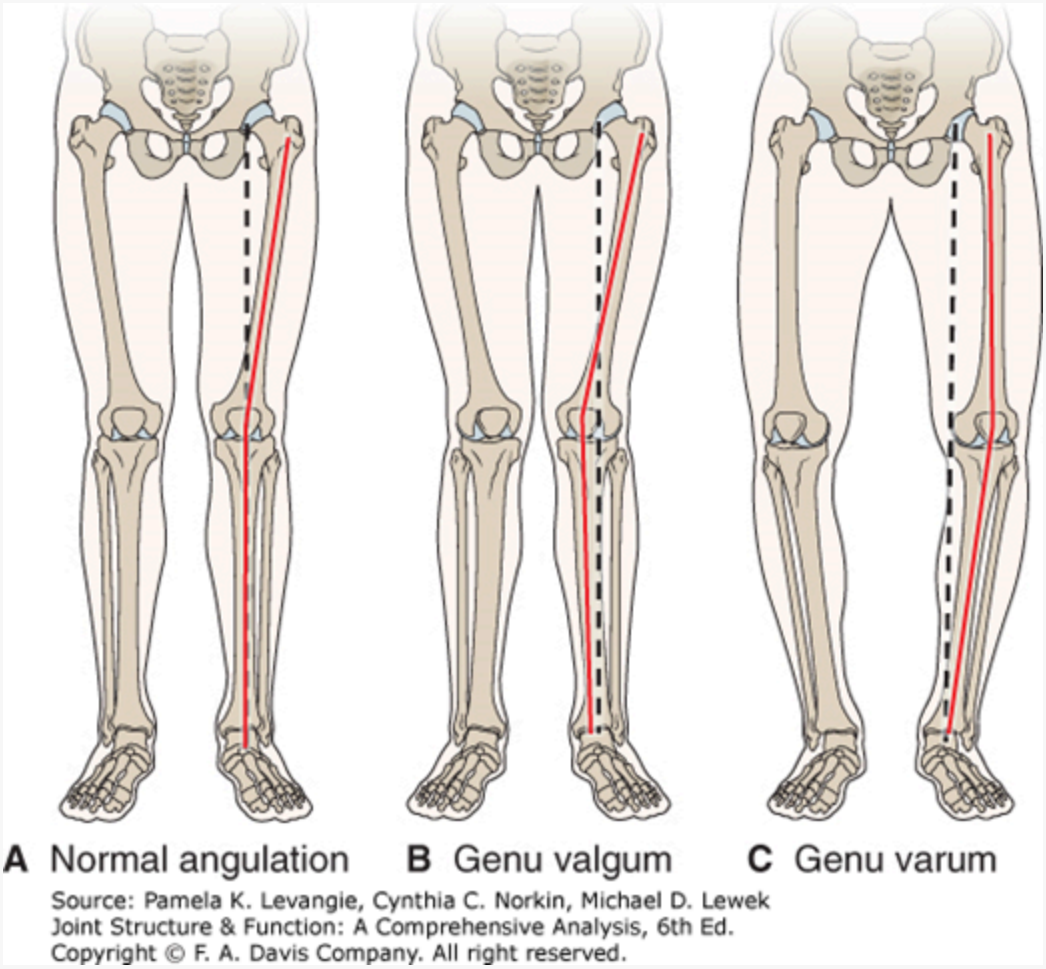
Tibiofemoral Alignment and Weightbearing Forces

The **anatomical (longitudinal) axis** of the femur, as previously discussed, is oblique, and directed inferiorly and medially from its proximal to distal end; the longitudinal axis of the tibia is directed almost vertically (**Fig. 11–5**). Consequently, the femoral and tibial longitudinal axes normally form an angle medially at the knee joint of 180° to 185°; that is, the femur is angled up to 5° off vertical, creating a slight physiological (normal) valgus angle at the knee (**Fig. 11–5A**).[10] If the medial tibiofemoral angle is greater than 185°, an abnormal condition called **genu valgum** (“knock knees”) exists (**Fig. 11–5B**). If

the medial tibiofemoral angle is 175° or less, the resulting abnormality is called **genu varum** (“bow legs”; Fig. 11–5C). Each condition alters the compressive and tensile stresses on the medial and lateral compartments of the knee joint.

FIGURE 11-5

A. The anatomical axes of the femur and tibia result in a normal physiological valgus angulation of approximately 185°. **B.** An increase in the normal tibiofemoral angle results in genu valgum, or “knock knees.” Compression forces are increased on the lateral aspect of the tibiofemoral joint. **C.** A decrease in the normal tibiofemoral angle results in genu varum, or “bow legs.” Here, compression forces are increased medially.



Frontal plane knee malalignment has significant clinical implications due to its strong association to the incidence and progression of knee osteoarthritis.[11,12] The impact of knee malalignment can be realized by measuring tibiofemoral alignment as the **mechanical axis,** or **weightbearing line,** of the lower extremity. The weightbearing line is computed from long leg radiographs of the lower extremity and can be used as a simplification of the ground reaction force as it travels up the lower extremity. The weightbearing line is represented as a line drawn from the middle of the calcaneus to the head of the femur (Fig. 11–5A–C).[13] In a neutrally aligned knee, the weightbearing line will pass through the center of the knee joint, distributing load equally between the

medial and lateral compartments.

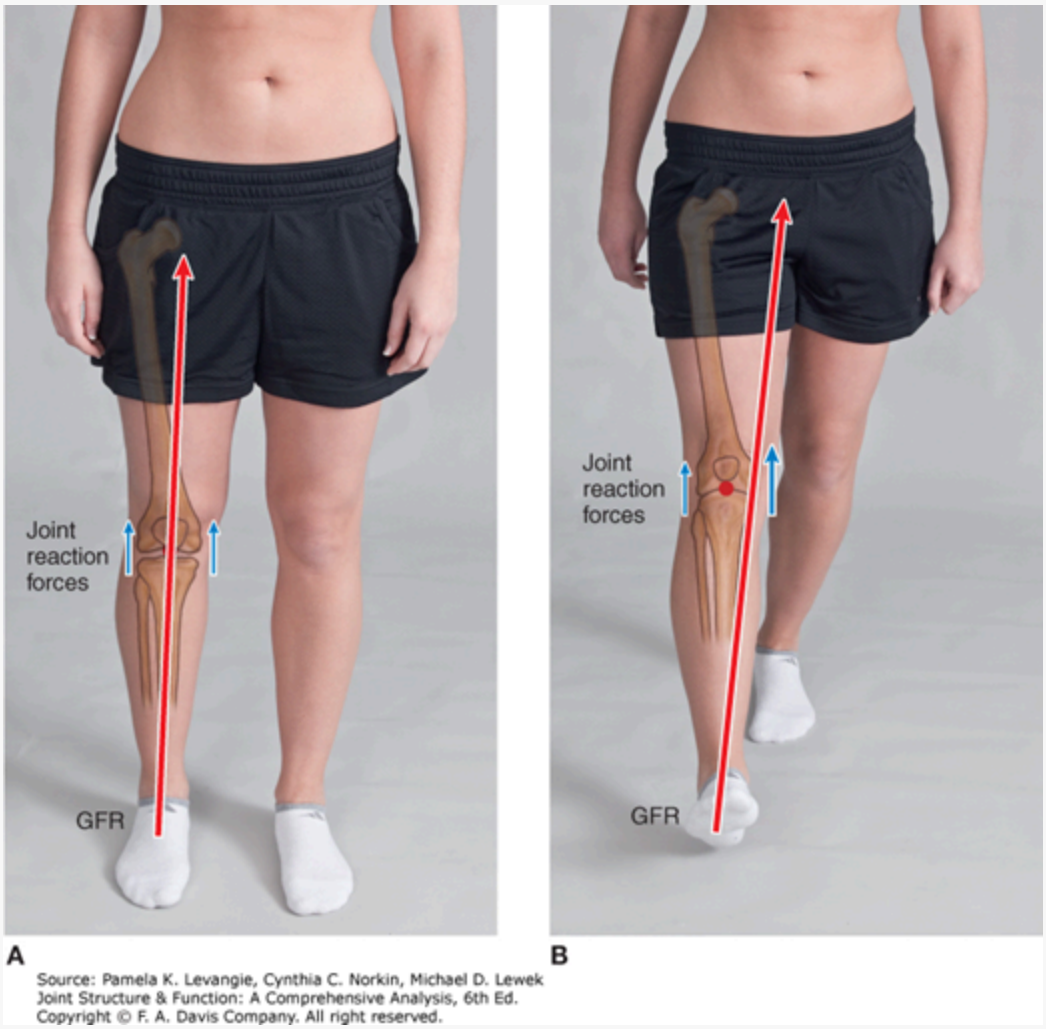
In the presence of genu valgum the weightbearing line is shifted onto the lateral compartment, increasing the lateral compressive force (Fig. 11–5B) compared with a neutrally aligned knee. In the case of genu varum, the weightbearing line is shifted medially, further increasing the compressive force on the medial condyle (Fig. 11–5C). The presence of genu valgum or genu varum creates a constant overload of the lateral or medial articular cartilage, respectively, which may result in damage to the articular cartilage and the development of frontal plane knee laxity.[14,15] Genu varum, which contributes to the progression of medial compartment knee osteoarthritis, yields a corresponding medial joint laxity as the attachment sites of ligamentous and capsular structures on the medial side of the joint are gradually approximated through the narrowing of the medial compartment's articular cartilage ("pseudo-laxity").[16]

The weightbearing stresses on the knee joint differ in bilateral and unilateral stance (**Fig. 11–6**). In bilateral stance, the weightbearing stresses on the knee joint are equally distributed between the medial and lateral condyles (or medial and lateral compartments) in an individual with a neutral mechanical axis (**Fig. 11–6A**).[17] However, if unilateral stance is adopted or dynamic forces are applied to the joint, compartmental loading is altered. In the case of unilateral stance (e.g., during the stance phase of gait), the weightbearing line shifts toward the medial compartment to account for the now smaller base of support below the center of mass (**Fig 11–6B**). This shift increases the compressive forces on the medial compartment and reduces compression across the lateral compartment.[18] Although the medial compartment typically experiences greater joint compression than the lateral compartment, the larger articular surface of the

medial compartment helps maintain a moderate level of joint stress.

FIGURE 11-6

A. The mechanical axis (weightbearing line) of the lower extremity passes from the center of the hip to the center of the ankle joint and, in a neutrally aligned limb, results in weightbearing forces that are distributed about equally between the medial and lateral condyles of the knee joint. **B.** During dynamic activities, such as gait, the line of force shifts medially to the knee joint center. This medial shift increases the compressive stresses medially.



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Advances in technology allow both direct and indirect measurements of the mechanical loading across the knee's medial and lateral compartments. In vivo knee loading characteristics have been measured by instrumenting the prostheses used for total knee arthroplasty. Such direct measurements indicate that most activities of daily living place a greater load on the medial compartment compared with the lateral compartment, which may correspond to the higher rate of medial compartment osteoarthritis when compared with the lateral compartment.[19,20] Less invasive measures can be used to estimate the compressive loads within the knee joint.

Specifically, external forces through the leg can be measured from a force plate embedded in a walkway, allowing researchers to measure ground reaction forces during normal daily and sports activities (e.g., walking, running, jumping; Fig. 11–6B). These external forces can be used as surrogates for internal forces incurred by the knee joint and may be used to calculate torques (moments of force) about the joint. For example, if the force from the floor extends medial to the knee joint center, an external adduction moment is created around the knee joint, which acts to rotate the knee into greater varum (adduction). Thus, the magnitude of the knee adduction moment is often used as a surrogate measure for medial compartment loading during gait and other activities of daily living.[21] This is important because higher knee adduction moments are associated with the presence and severity of medial knee osteoarthritis.[22]



Expanded Concepts 11–1

Effects and Corrections of Malalignment

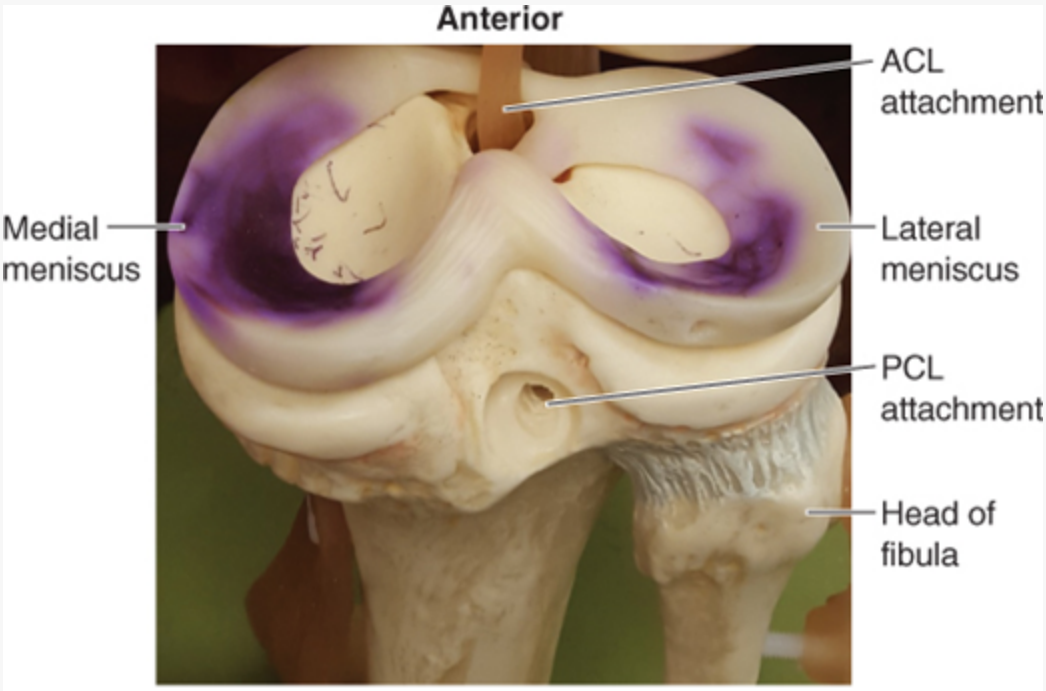
In the presence of frontal plane malalignment and unicompartmental osteoarthritis, orthopedic surgeons may perform a realignment procedure at the knee, called a high tibial osteotomy.[23] This procedure is intended to decrease the compressive force on the damaged, painful tibiofemoral compartment by realigning the limb and moving the weightbearing line toward the opposite, unaffected tibiofemoral compartment.[24] High tibial osteotomies are also sometimes used in combination with meniscus and articular cartilage procedures when offloading of the surgically repaired structures is needed.[23] In the case of either significant genu varum or genu valgum, the surgery creates a surgical fracture in the tibia (or sometimes in the femur) to realign the limb to a more neutral position. Other less invasive methods of attempting to diminish compartmental loads in the presence of malalignment include lateral/medial heel wedges or a knee brace that shifts weightbearing to the uninvolved compartment (so-called unloading braces).

Menisci

The relative tibiofemoral incongruence is improved by the presence of **medial** and **lateral menisci**, which act to transform the convex tibial plateaus into concavities for the femoral condyles (**Fig. 11–7**). In addition to enhancing joint congruence, these accessory joint structures play an important role in transmitting and distributing weightbearing loads, reducing friction between the tibia and the femur, providing shock absorption, and enhancing joint stability.[25] The menisci are fibrocartilaginous discs with a semicircular shape. The medial meniscus is C-shaped, whereas the lateral meniscus forms four fifths of a circle.[26] Lying within the tibiofemoral joint, the menisci are located on top of the tibial condyles, covering one-half to two-thirds of the articular surface of the tibial plateaus (**Fig. 11–8**).[27] Both menisci are open toward the intercondylar tubercles and are thick peripherally and thin centrally. The lateral meniscus covers a greater percentage of the smaller lateral tibial surface than the surface covered by the medial meniscus.[28] As a result of its larger exposed surface, the articular cartilage of the medial tibial plateau not covered by meniscus carries a greater load during routine daily activities than that of the lateral tibial plateau, making it more susceptible to injury.[29]

FIGURE 11-7

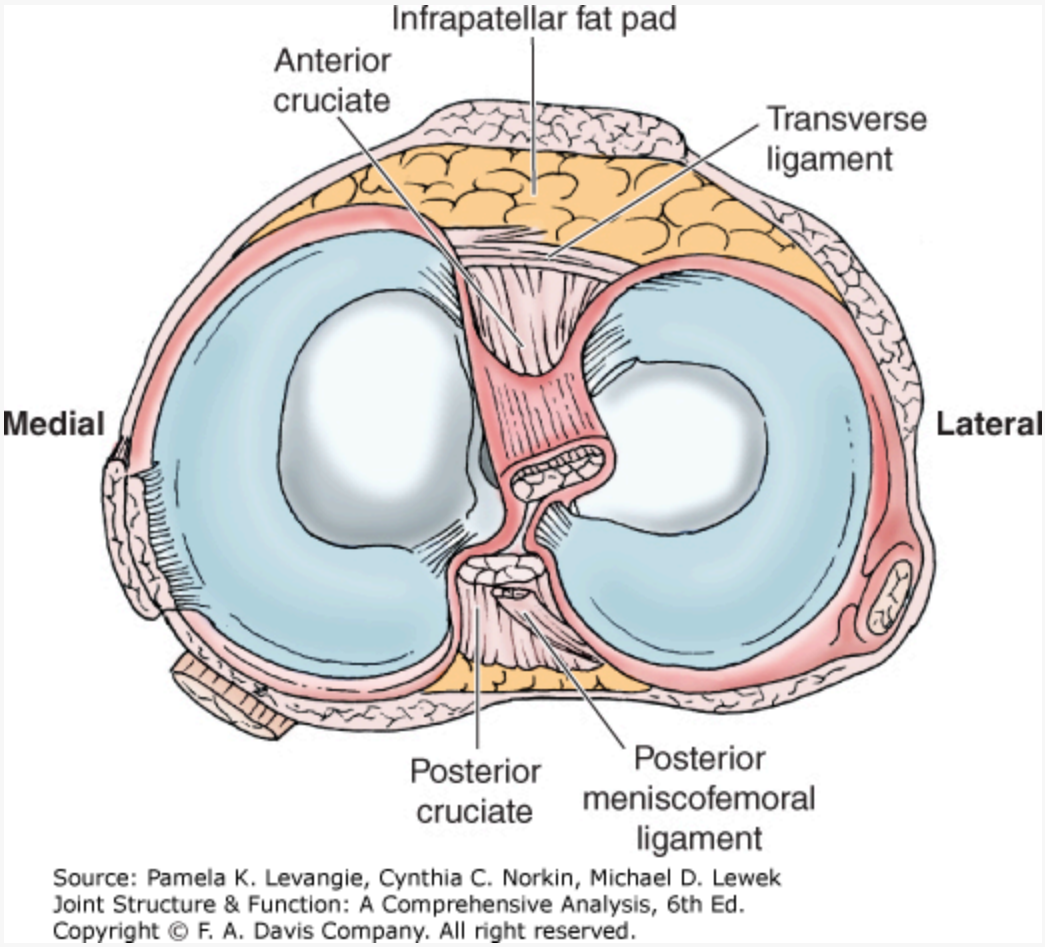
A posterosuperior view of the tibia with the menisci. The wedge shape of the menisci is evident and the purple coloration emphasizes how the menisci deepen and contour the tibial articulating surface to accommodate the femoral condyles.



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FIGURE 11-8

Structure of the menisci. A superior view of the menisci illustrates differences in size and configuration between the medial and lateral menisci. The medial meniscus is C-shaped, whereas the lateral meniscus is shaped like a nearly complete ring or circle. The location of the attachments of the anterior cruciate ligament and posterior cruciate ligament on the tibial plateau is also shown.



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The circumferential arrangement of meniscal fibers allows axial loads to be dispersed in a radial direction, thereby reducing wear on the hyaline articular cartilage of the femur and tibia.[30] The menisci assume 50% to 70% of the loads imposed through the knee, which can be considerable given that compressive forces in the knee may reach one to four times body weight during gait and stair climbing, three to four times body weight during running, and seven to eight times body weight during jumping and landing.[26,31–35] These loads can be further influenced by the presence of frontal plane malalignment. The greater the degree of genu varum, for instance, the greater the compression on the medial meniscus.

MENISCAL ATTACHMENTS

Meniscal motion on the tibia is limited by multiple attachments to surrounding structures. The medial meniscus has more ligamentous and capsular restraints than the lateral meniscus, resulting in more restricted translational movements.[36] The relative lack of mobility of the medial meniscus may contribute to its greater incidence of injury.[28,37]

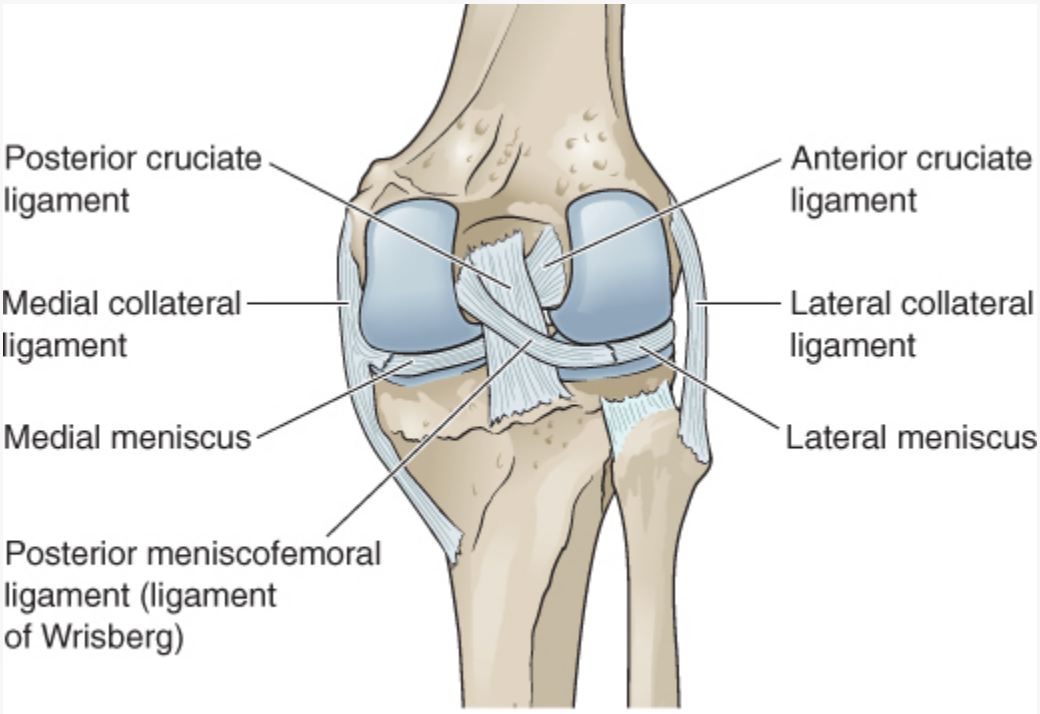
The anterior and posterior ends of the menisci are called the **anterior** and **posterior horns,** each of which are firmly attached to the tibia.[36,38] Anteriorly, the menisci are connected to each other by the **transverse ligament** (Fig. 11–8).[27,28] Both menisci are also attached directly or indirectly to the patella via the **patellomeniscal ligaments,** which are anterior capsular thickenings.[39] At the periphery, the menisci are connected to the tibial condyle by the **coronary ligaments,** which are composed of fibers from the knee joint capsule. The medial meniscus is firmly attached to the medial joint capsule through a capsular thickening extending from the femur to the tibia, referred to as the deep portion of the **medial collateral ligament (MCL).** This connection to the MCL contributes to the restricted motion of the medial meniscus.[36] The anterior and posterior horns of the medial meniscus are attached to the **anterior cruciate ligament (ACL)** and **posterior cruciate ligament (PCL),** respectively. Through capsular connections, the **semimembranosus muscle** connects to the medial meniscus.[40]

The anterior horn of the lateral meniscus and the anterior cruciate ligament share a common tibial insertion site.[41] Posteriorly, the lateral meniscus attaches to the posterior cruciate ligament and the medial femoral condyle through the **meniscofemoral ligament** (Figs. 11–8 and **11–9**).[42,43] In much the same way that the

semimembranosus tendon is attached to the medial meniscus, the tendon of the **popliteus muscle** attaches to the lateral meniscus.[27,43] The attachment to the popliteus tendon helps restrain or control the motion of the lateral meniscus.[44]

FIGURE 11-9

The medial meniscus is attached to the medial collateral, anterior cruciate, and posterior cruciate ligaments. The lateral meniscus is also attached to the anterior and posterior cruciate ligament (the joint capsule has been removed for visualization). Note the lack of meniscal attachment to the lateral collateral ligament.



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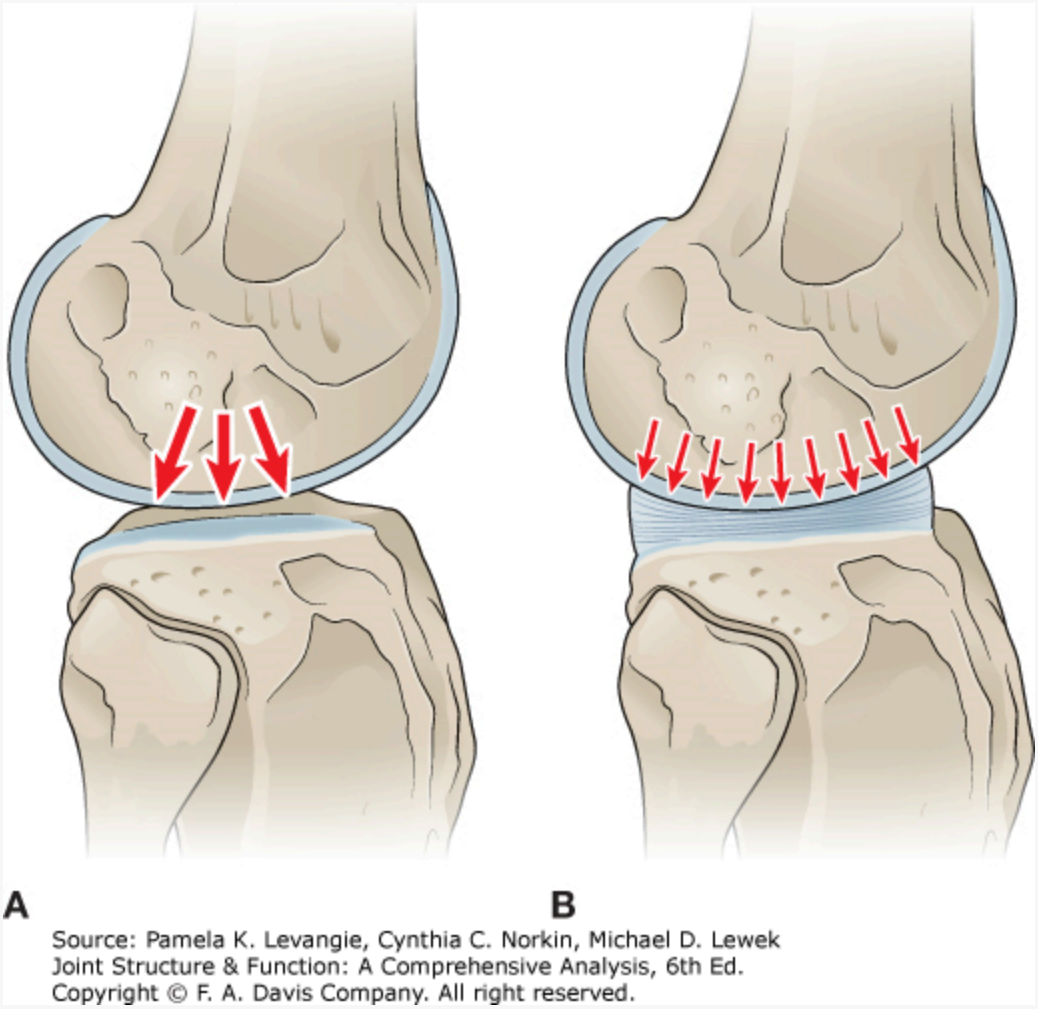
ROLE OF THE MENISCI

The strong attachments of the menisci prevent extrusion during compression of the

tibiofemoral joint, allowing for greater contact area between the menisci and the femur. If the femoral condyles sat directly on the relatively flat tibial plateau, a small contact area would exist between the bony surfaces. With the addition of the menisci, the contact area at the tibiofemoral joint is increased and thus joint stress (force per unit area) is reduced (Fig. 11–10).[26] After the removal of part or of all of a meniscus (meniscectomy), the contact area in the tibiofemoral joint is reduced. This results in increased joint stress and may lead to damage of the articular cartilage and other degenerative changes within the tibiofemoral joint. Removal of the menisci nearly doubles the articular cartilage stress on the femur and multiplies the stress by six or seven times on the tibial plateau.[45] For this reason, total meniscectomies are rarely performed after a meniscal tear; instead, care is taken to preserve as much of the meniscus as possible, either through debridement (removal of damaged tissue) or repair.[46] Meniscal transplants are sometimes completed in young patients when previous meniscal procedures have been performed but persistent pain continues. Although short-term results indicate improved function and relief of pain, long-term clinical outcomes are limited and variable.[47–50]

FIGURE 11-10

A. If the femoral condyles sit directly on the flat tibial plateau, the stress (force per unit area) is high because of the limited contact area. **B.** With the addition of the wedge-shaped menisci, the contact area is increased, and the stress between the bony surfaces is reduced.



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Patient Application 11–1

Tina is a 43-year-old woman who presents with complaints of knee pain with increased activity. Tina's chief complaint is pain in and around her right knee (pain at best: 2/10) that worsens when she plays tennis and during stair ascents and descents (pain at worst: 8/10). She has palpable pain along the medial aspect of her tibiofemoral joint line and subjectively describes pain deep on the inside of her knee. She is currently unable to jog, play tennis, and walk for more than 1 mile without discomfort. Tina's past medical history includes tearing her medial meniscus on the right side when she was 24 years old, which was surgically treated with a partial medial meniscectomy. Clinical testing of her right knee revealed increased laxity with a valgus stress test with no pain at the end of range, full tibiofemoral range of motion (ROM), and 80% quadriceps strength compared with her left side. Diagnostic images

were obtained of Tina's lower extremities. Weightbearing radiographs of the right limb revealed genu varum with moderate joint space narrowing in the right medial tibiofemoral compartment. What structural abnormalities do you think are contributing to the limitations in function and pain at her right knee?

Tina's prior history of medial meniscal damage and joint space narrowing predispose her knee to static malalignment.[51] Her genu varum, according to measurements taken from her radiographs, may alter the distribution of compartmental joint forces and increase the magnitude of compression force through the medial compartment during activities of daily living, resulting in subsequent articular cartilage degradation and the joint space narrowing evident on her radiograph. In the presence of the greater medial compartment compressive force caused by genu varum, the partial removal of the medial meniscus during her initial surgery becomes more detrimental. The partial removal of Tina's medial meniscus diminishes the contact area within the medial tibiofemoral compartment. A greater force is now being focused through a smaller contact area, creating significantly higher joint stress and raising the potential for degenerative articular cartilage changes within the joint. Furthermore, Tina's quadriceps weakness and knee joint laxity may be influenced by her knee malalignment.[52] A history of compensatory strategies could lead to medial compartment irritation and may partially explain her medial knee pain.

MENISCAL NUTRITION AND INNERVATION

The location of a meniscal lesion, along with the age and health of the patient, influences the treatment options available after injury due to the capacity of the meniscus to heal. During the first year of life, blood vessels are contained throughout the meniscal body. Once weightbearing is initiated, vascularity begins to diminish until only the outer 25% to 33% is vascularized by capillaries from the joint capsule and the synovial membrane.[53] After 50 years of age, only the periphery of the meniscal body is vascularized. Therefore, the peripheral portion obtains its nutrition through blood vessels, but the central portion must rely on the diffusion of synovial fluid.[25,37] The process of fluid diffusion to support nutrition requires intermittent loading of the

meniscus by either weightbearing or muscular contractions.[53] Subsequently, during prolonged non-weightbearing conditions or periods of immobilization, the meniscus may not receive adequate nutrition. In addition, the avascular nature of the central portion of the meniscus reduces the potential for healing after an injury.[54] In adults, only the peripheral vascularized region of the meniscus is capable of inflammation, repair, and remodeling after a tearing injury. Therefore, a meniscal repair may be indicated only if the lesion is isolated to the peripheral margins of the meniscus.[46]

The horns of the menisci and the peripheral vascularized portion of the meniscal bodies are well innervated with free nerve endings (nociceptors) and three different mechanoreceptors (Ruffini corpuscles, Pacinian corpuscles, and Golgi tendon organs), whereas the central portions are aneural.[25,53] The presence of nociceptors in the meniscus may explain some of the pain felt by patients after a meniscal tear if the lesion is located peripherally.[55] The potential for proprioceptive deficits exists after meniscal injury as a result of injury to the mechanoreceptors within the meniscus.

Joint Capsule

Despite the additional support provided by the menisci, the incongruent knee joint is heavily dependent on the surrounding joint structures for joint stability. The delicate balance between stability and mobility varies as the knee is flexed from full extension toward flexion. Bony congruence and overall ligament tautness are maximal in full extension, representing the close-packed position of the knee joint. In mid-range knee flexion, the periarticular passive structures tend to be more lax, and the relative bony incongruence of the joint permits greater anterior and posterior translations, as well as rotation of the tibia beneath the femur.[56]

A single, large joint capsule encloses both the tibiofemoral and patellofemoral joints. It is grossly composed of a superficial fibrous layer and a thinner, deep synovial membrane. In general, the outer fibrous portion of the capsule is firmly attached to the inferior aspect of the femur and the superior portion of the tibia.[57] Posteriorly, the capsule is attached proximally to the posterior margins of the femoral condyles and intercondylar notch, and distally to the posterior tibial condyle.[58] The anterior borders of the joint capsule consist of the tendon of the quadriceps muscles, the patella, and the **patellar tendon.** The anteromedial and anterolateral portions of the capsule are often separately identified as the **medial** and **lateral patellar retinaculae,** or together as the **extensor retinaculum.**[59] The joint capsule is reinforced medially, laterally, and posteriorly by capsular ligaments, which will be discussed in detail later.

The knee joint capsule and its associated ligaments are critical in restricting excessive joint motions to maintain joint integrity and normal function. Although muscles clearly play a dominant role in stabilization (as we shall examine more closely later in the chapter), it is difficult to stabilize the knee with active muscular forces alone in the presence of a disruption of the knee's capsule and ligaments. Beyond knee joint stability, the joint capsule plays important sensory and proprioceptive roles. The joint capsule is strongly innervated by both nociceptors as well as Pacinian and Ruffini corpuscles.[57] These mechanoreceptors may contribute to muscular stabilization of the knee joint by initiating reflex-mediated muscular responses. In addition, the joint capsule provides a tight seal to keep the lubricating synovial fluid within the joint space.[57]

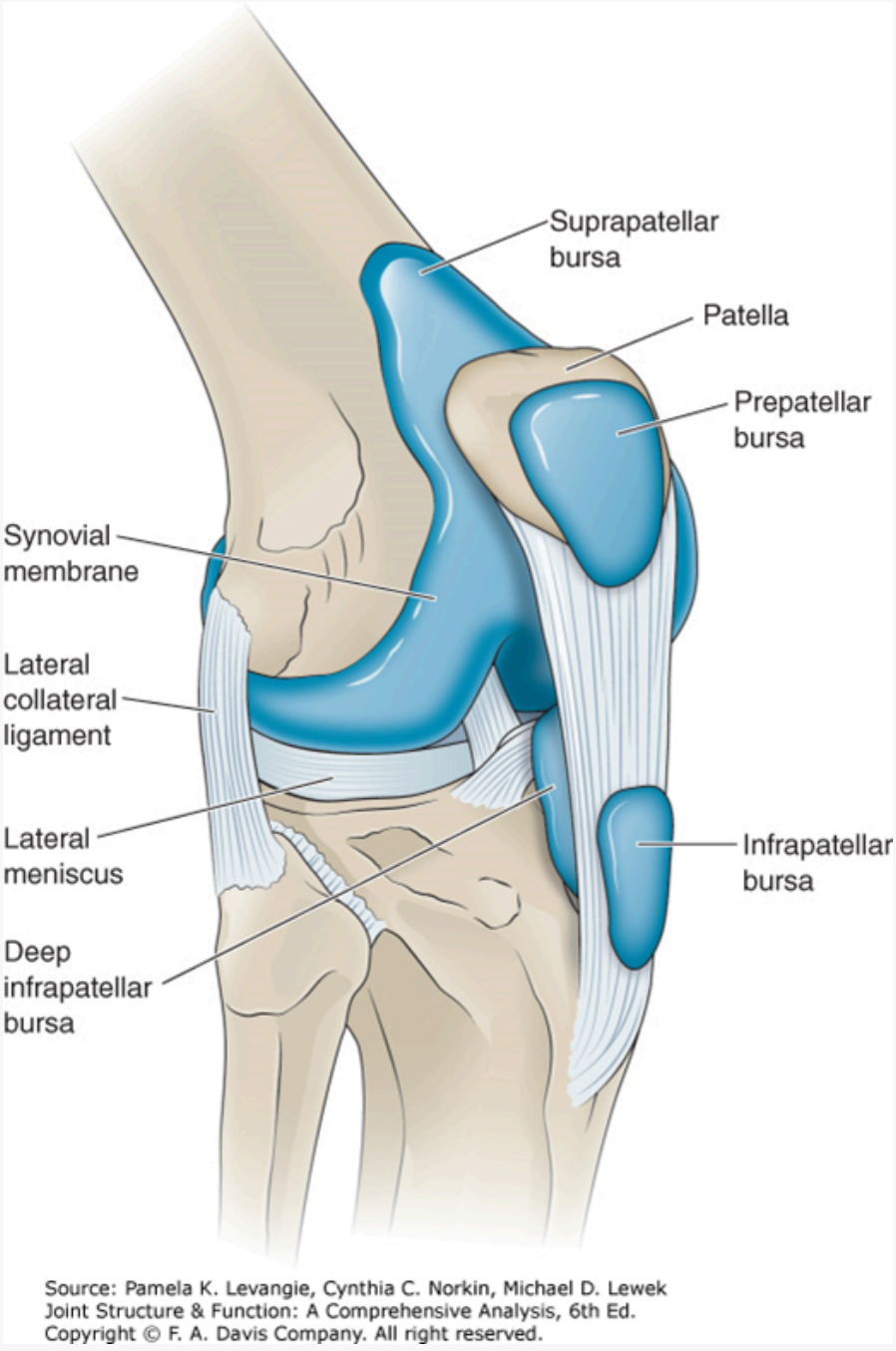
SYNOVIAL LAYER OF THE JOINT CAPSULE

A synovial membrane forms the inner lining in much of the knee joint capsule.[60,61] The roles of the synovial tissue are to secrete and absorb synovial fluid into the joint for lubrication and to provide nutrition to avascular structures, such as portions of the menisci.[62] The synovial lining of the joint capsule is quite complex and is among the most extensive and involved in the body (**Fig. 11–11**). Posteriorly, the synovium breaks away from the inner wall of the fibrous joint capsule and invaginates anteriorly between the femoral condyles, where it adheres to the anterior aspect and sides of the anterior and the posterior cruciate ligaments.[61,63,64] Therefore, both the anterior cruciate ligament and the posterior cruciate ligament are contained within the fibrous capsule (intracapsular), but lie outside of the synovial sheath (extrasynovial).[1,63,64] Posterolaterally, the synovial lining delves between the popliteus muscle and lateral femoral condyle, whereas posteromedially it may invaginate between the semimembranosus tendon, the medial head of the **gastrocnemius** muscle, and the medial femoral condyle. The intricate folds of the synovium exclude several fat pads that lie within the fibrous capsule, making them intracapsular but extrasynovial, similar to the cruciate ligaments.[65] The anterior and posterior suprapatellar fat pads lie posterior to the **quadriceps tendon** and anterior to the distal femoral epiphysis, respectively. The **infrapatellar (Hoffa's) fat pad** lies deep to the patellar tendon (see Fig. 11–8).

FIGURE 11-11

An anterolateral view of the knee complex (with the fibrous outer layer of the capsule removed) shows the complex course of the

synovial layer of the knee joint capsule, including the related bursae.



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Formation of the knee joint's synovial membrane occurs early in embryonic development, when it separates the medial and lateral articular surfaces into separate joint cavities.[66,67] By the 12th week of gestation, the synovial septae are somewhat

resorbed, resulting in a single joint cavity but with retention of the posterior invagination of the synovium between the femoral condyles.[61] The failure of the synovial membrane to become fully resorbed can result in persistent folds in specific regions of the membrane. These folds are called **patellar plicae.**[67] There are four potential locations relative to the patella where plicae may be located: inferior (infrapatellar plica), superior (suprapatellar plica), medial (mediopatellar plica), and lateral (lateral patellar plica).[66,67] The size, shape, and frequency of these plicae vary among individuals, although infrapatellar plica are most common and lateral patellar plica are least common.[66] Synovial plicae, when they exist, are generally composed of loose, pliant, and elastic fibrous connective tissue that easily passes back and forth over the femoral condyles as the knee flexes and extends.[68] On occasion, a plica may become irritated and inflamed, called **plica syndrome,** which can lead to pain, effusion, and changes in joint structure and function.[68]



Expanded Concepts 11–2

Patellar Plicae

The infrapatellar plica, also called the **ligamentum mucosum,** originates in the intercondylar area of the femur and courses parallel to the anterior border of the anterior cruciate ligament, passes through the infrapatellar fat pad (of Hoffa), and attaches distally to the inferior pole of the patella.[67] It is the most common plica present in the knee.[67] The suprapatellar plica is generally located superior to the patella, forming from the undersurface of the quadriceps tendon, often extending to the suprapatellar synovial pouch at the anterior aspect of the distal femoral shaft.[67] Despite its location above the patella, the superior plica rarely gets impinged between the patella and the femur.[68] In contrast, the medial plica is more often implicated as a source of pain, despite being found less frequently (in only 25% to 30% of knees) than either the superior or inferior plica (found in 50% to 65% of knees).[67,68] The medial plica arises from the synovium lining the medial wall of the pouch of the extensor retinaculum and courses parallel to the medial edge of the

patella to attach to the synovium of the infrapatellar fat pad and sometimes the inferior plicae.[67,68] Discomfort from the medial plica often occurs during knee movements that change the plical dimension and orientation stemming from their attachments to synovial lined structures.[67] For example, a normally thin and elastic medial plica can become impinged, beginning around 30° to 50° of knee flexion between the medial aspect of the patella and the medial femoral condyle, eventually leading to inflammation and thickening of the plica tissue.[67] The great deal of pain that occurs from the irritated synovial membrane can be attributed to its rich supply of Pacinian corpuscles and free nerve endings.[60]

FIBROUS LAYER OF THE JOINT CAPSULE

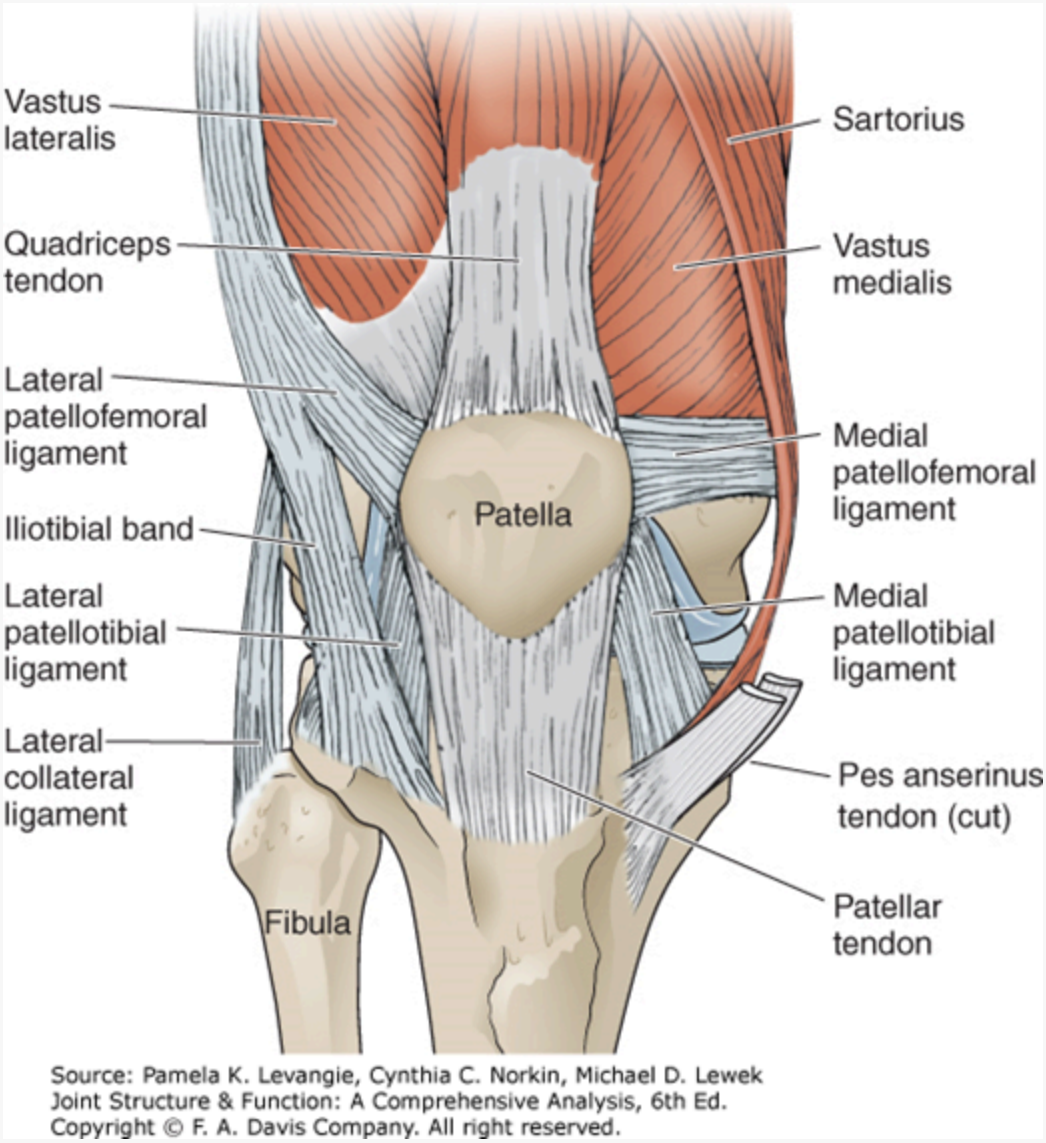
The fibrous joint capsule lies superficial to the synovial lining of the knee joint and provides passive support for the joint. The fibrous joint capsule itself is composed of two or three layers, depending on location. Several capsular thickenings (or capsular ligaments), as well as both intracapsular and extracapsular ligaments, provide additional structural support to the incongruent knee joint.

The anterior portion of the knee joint capsule is called the extensor retinaculum. Superficially it consists of a fascial layer that covers the distal **quadriceps muscles** and extends inferiorly. Deep to this layer, the anterior capsule consists of the medial and lateral retinacula, a series of transverse and longitudinal fibrous bands connecting the patella to the surrounding medial and lateral knee structures (**Fig. 11–12**).[59] The thickest band within the medial retinaculum is the **medial patellofemoral ligament (MPFL)**.[69] Its fibers, oriented in a transverse manner, course anteriorly from the adductor tubercle of the femur to blend with the distal fibers of the **vastus medialis** and eventually insert onto the medial border of the patella.[69,70] The medial patellofemoral ligament is a clinically important stabilizer to maintain the patella in the femoral sulcus, and can rupture during lateral patellar subluxation or dislocation.[71] The lateral

retinaculum consists of less discrete fibrous bands compared with the medial retinaculum.[59] The transversely oriented fibers within the lateral retinaculum, called the **lateral patellofemoral ligament,** travel from the **iliotibial (IT) band** to the lateral border of the patella.[72]

FIGURE 11-12

The extensor retinaculum is reinforced medially by the transversely oriented medial patellofemoral ligament and the longitudinally oriented medial patellotibial ligament. Laterally, the lateral patellofemoral ligament and lateral patellotibial ligament help resist an excessive medial glide of the patella.



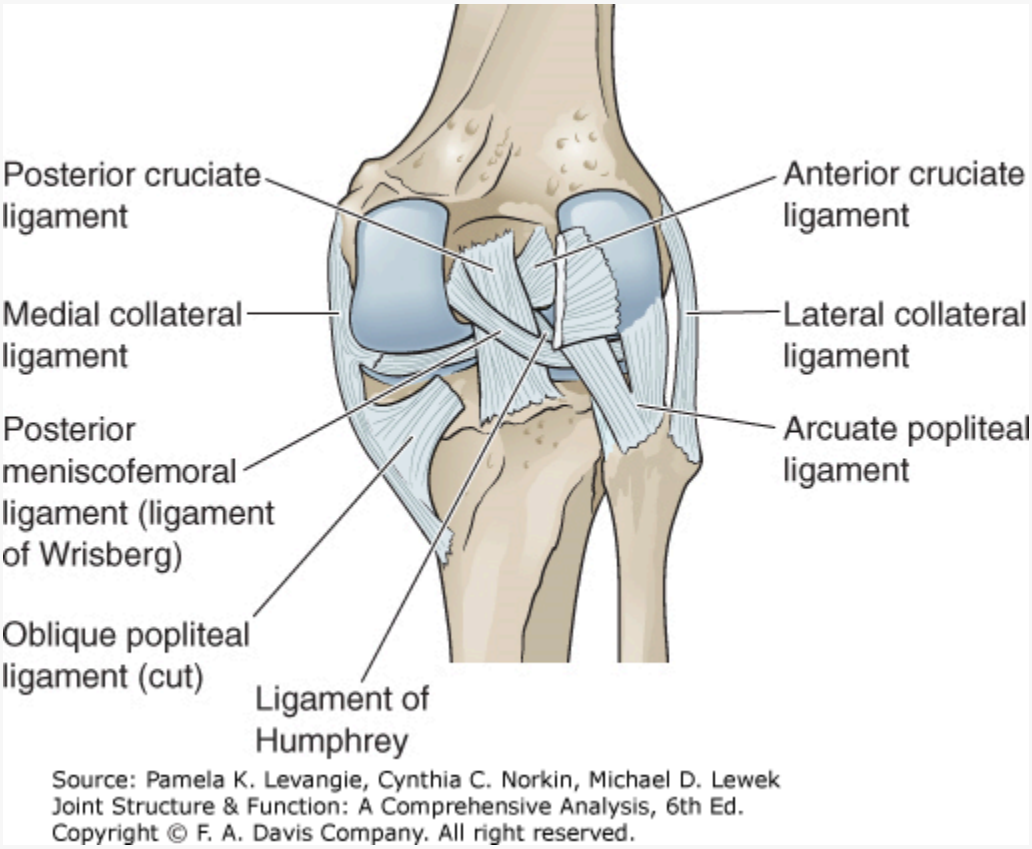
The remainder of the retinacular bands include the obliquely oriented **medial patellomeniscal ligament,** the **lateral patellomeniscal ligament,** and the longitudinally positioned **medial** and **lateral patellotibial ligaments** (Fig. 11–12).[59,72]

The medial portion of the joint capsule is composed of the deep and superficial portions of the medial collateral ligament. The superficial layer of the medial joint capsule blends with the fascial layers that cover the vastus medialis muscle anteriorly

and the **sartorius** muscle posteriorly.[73] Laterally, the joint capsule is reinforced superficially by the iliotibial band and its thick **fascia lata.**[74] The capsule is reinforced posteromedially by the **posterior oblique ligament (POL)** and posterolaterally by the **arcuate ligament** (Fig. 11–13).[69,73,75,76]

FIGURE 11-13

A view of the posterior capsule of the knee shows reinforcement from the oblique popliteal ligament, arcuate ligament, and posterior oblique ligament.



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Ligaments

The roles of the various knee ligaments have received extensive attention in the

literature, which reflects their importance for knee joint stability and the frequency with which function is disrupted through injury. Given the lack of bony restraint to virtually any of the knee motions, the knee joint ligaments are variously credited with resisting or controlling the following:

1. Excessive knee extension
2. Varus and valgus stresses at the knee (adduction or abduction of the tibia, respectively)
3. Anterior or posterior displacement of the tibia relative to the femur
4. Medial or lateral rotation of the tibia relative to the femur
5. Combinations of anteroposterior displacements and rotations of the tibia, together known as **rotary stabilization** of the tibia



Foundational Concepts 11–1

Weightbearing/Non-Weightbearing Versus Closed/Open Kinetic Chain

Although the ligamentous checks previously described were defined by the tibia's motions, stresses may also occur on the femur while the tibia is fixed (as in weightbearing). Such weightbearing activities (often called *closed-chain* activities) involve motion of the femur moving on a relatively fixed tibia. In contrast, non-weightbearing activities (often termed *open-chain* activities) involve a moving tibia on a relatively fixed femur. As noted in earlier chapters, weightbearing activities result in true closed-chain effects only when the position of the head (or trunk) is relatively fixed in space, generally to maintain the line of gravity within the base of support. Consequently, we will refer to activities as weightbearing or non-weightbearing, rather than as "closed chain" or "open chain." The difference between weightbearing and non-weightbearing motions is important because the

displacements and rotations of the tibia and the femur are expressed differently for each condition. For example, anterior displacement of the tibia on the femur (during non-weightbearing) is equivalent to posterior displacement of the femur on the tibia (during weightbearing).

The large body of literature available on ligamentous function of the knee joint can be confusing and sometimes appears contradictory. This may be due to vague descriptions regarding whether the tibia or the femur is being referenced, or the testing of ligaments in dissimilar conditions. For example, it is clear that ligamentous function can change depending on the position of the knee joint, how the stresses are applied, and what active or passive structures are concomitantly intact. Our knowledge of the stresses (force per unit area) and strains (relative deformation) of ligamentous tissues has been significantly advanced using cadaveric experiments. Two primary approaches have been used to reveal the relative roles of the knee ligaments. The first approach is to measure the amount of knee displacement in response to an applied load. Following the sectioning (cutting) of a particular ligament, joint displacement is measured again with the same applied load. The relative change in displacement (strain) provides evidence for the ligament's role in restricting motion against the magnitude and direction of the applied force. An alternative approach uses a predetermined joint displacement and measures the force (stress) necessary to achieve that displacement. After a ligament is cut, the same pre-established displacement is applied while force values are measured. The change in the force required to displace the joint provides evidence of that ligament's role in resisting a given amount of joint displacement.

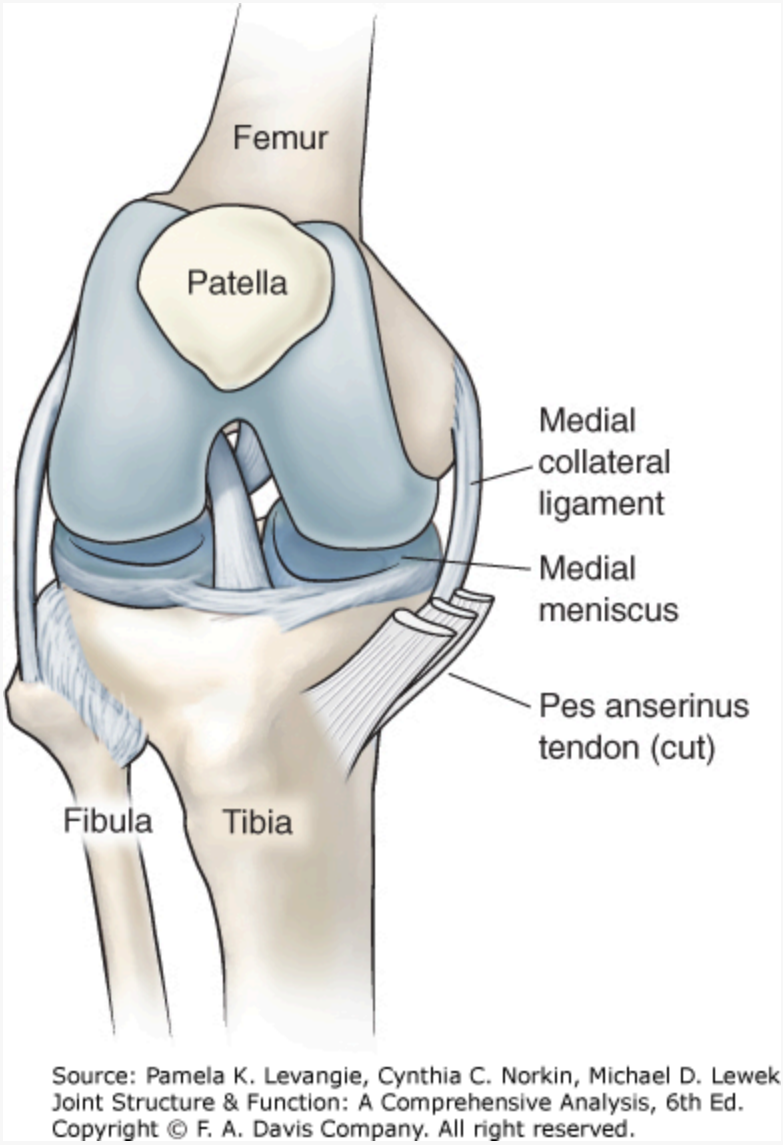
MEDIAL COLLATERAL LIGAMENT

The medial collateral ligament (MCL) can be divided into a superficial portion and a

deep portion that are separated by a bursa. The superficial portion of the MCL arises proximally from the medial femoral epicondyle, anterior to the origin of the posterior oblique ligament and just distal to the femoral insertion of the medial patellofemoral ligament.[58,77] It travels distally to insert into the medial aspect of the proximal tibia, distal to the **pes anserinus** (**Fig. 11–14**).[58,77] The deep portion of the MCL is continuous with the joint capsule, originating from the inferior aspect of the medial femoral epicondyle and inserting on the proximal aspect of the medial tibial plateau.[58] Throughout its course of travel, the deep portion of the MCL is rigidly affixed to the medial border of the medial meniscus (see Fig. 11–9).[58]

FIGURE 11-14

The superficial portion of the medial collateral ligament runs inferiorly from the medial femoral condyle to the anteromedial tibial condyle. Anterior and inferior to the distal portion of the medial collateral ligament is the attachment of the pes anserinus.



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The medial collateral ligament, specifically the superficial portion, is the primary restraint to excessive valgus (abduction) and lateral tibial rotation stresses at the knee.[78] The MCL is also important in restraining medial tibial rotation stresses at the knee.[78] The knee joint is better able to resist a valgus stress at full extension compared with a position of knee flexion because the MCL is taut in extension (e.g., close-packed position). As joint flexion is increased, the MCL becomes more lax and bony congruence decreases, resulting in greater possible medial joint space opening (medially

gapping).[79] With the knee flexed, however, the MCL plays a more critical role in resisting valgus stress despite the permitted joint gapping. Grood and colleagues explained that the MCL accounted for 57% of the restraining force against valgus opening near full extension, but accounted for 78% of the load at 25° of knee flexion.[80] This difference is likely due to the greater bony congruence and inclusion of other soft tissue structures (e.g., posteromedial capsule, anterior cruciate ligament) that can more effectively assist with resisting a valgus stress at full extension.

The medial collateral ligament also plays a secondary role in resisting anterior translation of the tibia on the femur in the absence of the primary restraints against anterior tibial translation.[81] Because of its secondary role as a restraint to anterior tibial translation, both partial and complete MCL tears will significantly increase the load on the anterior cruciate ligament.[82] Individuals with an MCL injury may also need protection from valgus and lateral rotation forces. For example, when concomitant anterior cruciate ligament tears or newly reconstructed anterior cruciate ligament grafts are present, rehabilitation exercises must be completed with the tibia neutrally positioned to avoid excessive strain on the medial collateral ligament until sufficient healing is completed. Surgical stabilization of an isolated MCL injury is rarely necessary and also frequently not completed with concomitant multiligament knee injuries because of its self-healing capacity.[83] The MCL has a rich blood supply providing it with the capacity to heal when ruptured or damaged. Nevertheless, the remodeling process can take up to a year.[83,84]

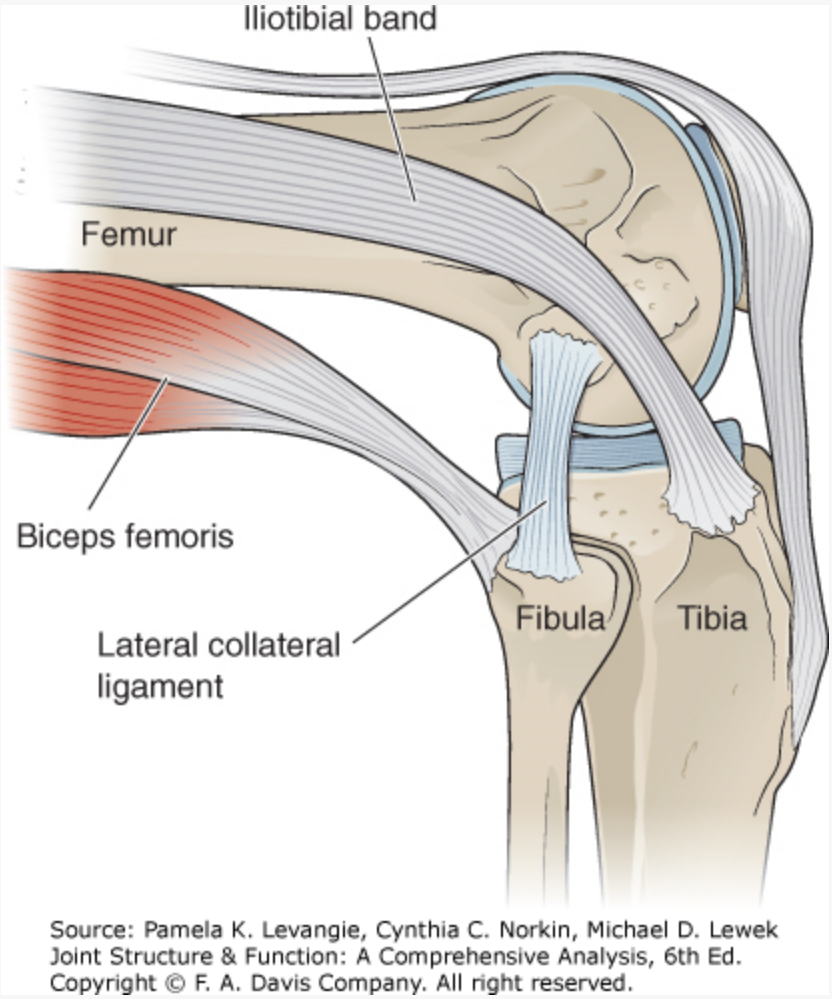
LATERAL COLLATERAL LIGAMENT

The **lateral collateral ligament (LCL)** attaches proximally at the lateral femoral

epicondyle and distally to the fibular head (**Fig. 11–15**), where it joins with the tendon of the **biceps femoris muscle** to form the conjoined tendon.[85,86] Unlike the medial collateral ligament, the LCL is not a thickening of the capsule but is separate throughout much of its length, therefore considered to be an extracapsular ligament (see Fig. 11–13). The LCL is primarily responsible for resisting varus stresses, and like the medial collateral ligament, limits frontal plane motion most successfully at full extension.[79,80,85] Also similar to the medial collateral ligament, the LCL's role in resisting frontal plane stress is greater in knee flexion than in full extension. Grood and colleagues reported that the LCL accounted for 55% of the restraining force against varus stress at 5° of knee flexion, but increases to 69% with the knee flexed to 25°.[80] Although the LCL's primary role is to resist varus stresses, its orientation enables the LCL to also limit excessive lateral rotation of the tibia in positions of 60° to 90° of knee flexion.[87] As with other knee ligaments, the LCL assumes additional stabilization roles in the presence of injury to other knee ligaments. For example, the LCL acts as a weak secondary restraint to anterior tibial translation and internal rotation in an anterior cruciate ligament deficient knee.[88]

FIGURE 11-15

The lateral collateral ligament joins the biceps femoris muscle in a common attachment to the fibular head, whereas the iliotibial band is attached distally to the anterolateral tibia.

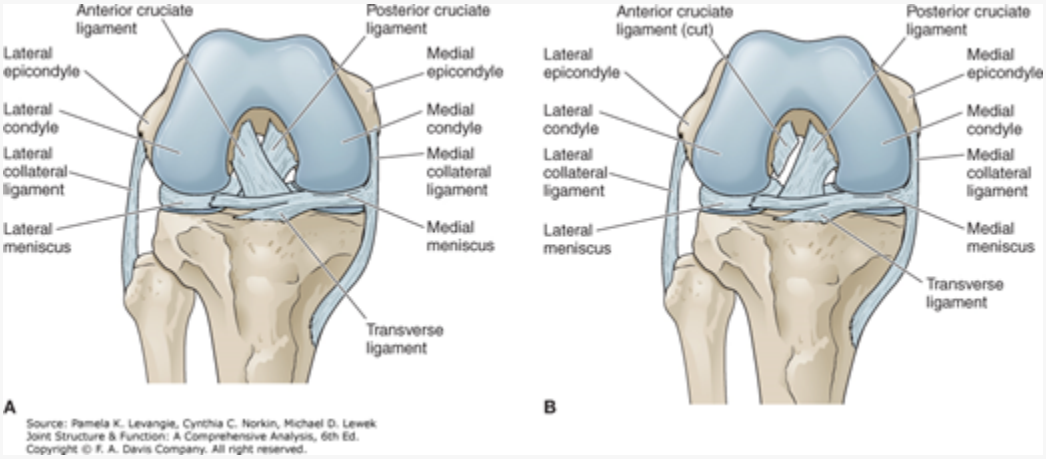


ANTERIOR CRUCIATE LIGAMENT

The high anterior cruciate ligament (ACL) injury rate among athletes and other active individuals has resulted in a substantial amount of research related to the ligament's structure and function (Fig. 11–16). The ACL originates at the posteromedial aspect of the lateral femoral condyle (see Fig. 11–9) and extends inferiorly, medially, and anteriorly to attach distally to the tibia on the lateral and anterior aspect of the medial intercondylar tibial spine (Fig. 11–16).[89]

FIGURE 11-16

A. An anterior view of a flexed knee joint shows the course of the anterior cruciate ligament from the lateral femoral condyle to the anterior portion of the tibial plateau. **B.** With the anterior cruciate ligament resected the posterior cruciate ligament can be viewed from the medial femoral condyle to the posterior tibial plateau.

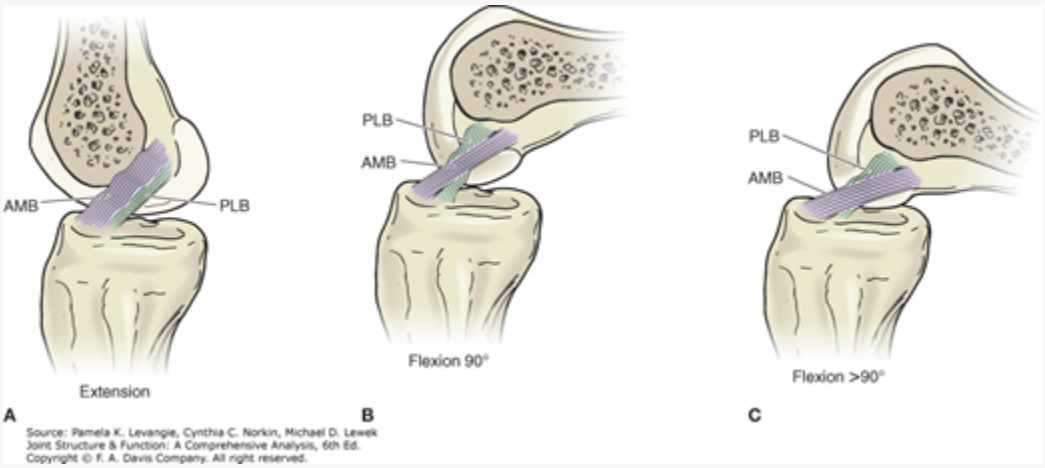


In addition, the ACL twists outwardly (laterally) as it travels distally.[63] The ACL consists of two separate bundles that wrap around each other as the knee flexes (Fig. 11–17).[89,90] The **anteromedial bundle (AMB)** and **posterolateral bundle (PLB)** are named for their tibial insertions.[91] The primary blood supply to both bundles of the ACL comes from the middle genicular artery.[91]

FIGURE 11-17

Although the anteromedial bundle (AMB) of the anterior cruciate ligament (ACL) is slack in extension and the posterolateral bundle (PLB) is slack in flexion, there is a continuum between the two, so that

some portion of the anterior cruciate ligament remains fairly taut throughout the range of motion.



The ACL functions as the primary restraint against anterior translation (anterior shear) of the tibia on the femur.[91] The anteromedial bundle and posterolateral bundle contribute differently to restraining anterior tibial translation depending on the knee flexion angle. The two bundles shift tension between themselves to allow some portion of the ACL to remain relatively taut throughout the knee's ROM. For example, with the knee near full extension, the posterolateral bundle is taut so that anterior tibial translation forces are greater within it compared with the anteromedial bundle. As the knee flexes beyond 15°, however, the posterolateral bundle loosens and the anteromedial bundle becomes more taut, resulting in greater force within the anteromedial bundle.[92,93] This sharing of the load by the anteromedial and posterolateral bundles allows the ACL to resist anterior translation of the tibia on the femur through a larger range of knee motion than either bundle could do on its own. In the intact joint, forces producing an anterior translation of the tibia on the femur will result in maximal tibial excursion at about 30° of flexion when neither of the ACL

bundles is maximally tensed.[94] In contrast, the least amount of anterior translation of the tibia occurs in full extension when many of the supporting passive structures of the knee are taut (including the posterolateral bundle of the ACL).[93] Because the posterolateral bundle is taut in full extension, the ACL is also responsible for resisting hyperextension of the knee.[95]

In addition to its primary restraint against anterior shear forces on the tibia, the ACL provides rotary stability of the knee during medial/lateral rotation, varus/valgus angulations, and combinations thereof.[69,90,93,95,96] The posterolateral bundle provides more rotational stability than the anteromedial bundle, especially when the knee joint is extended and the posterolateral bundle is more taut.[89,90,93] The ACL provides secondary stabilization to the medial collateral ligament in resisting valgus loads. In the presence of a partial or complete rupture of the medial collateral ligament of the knee, the strain in the ACL has been reported to increase 55% and 185%, respectively, during a valgus load.[82] The ACL can also play a secondary role in limiting medial rotation of the tibia. Medial tibial rotation on the femur can increase the strain on the anteromedial bundle of the ACL, with the peak strain occurring between 10° and 15° of knee flexion.[97–99] This is likely due to the orientation of the ACL winding its way medially around the posterior cruciate ligament, becoming more taut with medial rotation. Furthermore, Li and colleagues reported that quadriceps loading of an ACL-deficient knee results in increased medial tibial translation.[96] The medial translation might shift the contact location during knee joint loading and contribute to the development of degenerative changes in the joint, as new regions of articular cartilage are loaded that are not conditioned to frequent weightbearing forces.[100]

Although the ACL primarily resists anterior tibial translation, injuries to this ligament are not thought to occur during isolated instances of excessive anterior tibial translation. Rather, injury to the ACL occurs most commonly when the knee is slightly flexed and in a valgus position combined with anterior tibial translation or medial tibial rotation, or lateral tibial rotation during weightbearing activities (see Expanded Concepts 11–3).[103] In flexion and medial tibial rotation, the ACL is tensed as it winds around the posterior cruciate ligament, while in flexion and lateral tibial rotation the ACL is tensed as it is stretched over the lateral femoral condyle.[104]



Expanded Concepts 11–3

Loading the Anterior Cruciate Ligament Through Combined Motions

Quatman and colleagues demonstrated that combined translational and rotational movements can generate greater ACL strain than each movement individually. Importantly, the cadaveric testing was performed at 25° knee flexion to simulate landing from a jump, a common position for anterior cruciate ligament injury.[101] The authors determined that a combination of a valgus force with anterior translation and medial rotation of the tibia increases the strain on the ACL over three times as much as with either motion alone. Further, these three combined motions resulted in significantly greater strain in the ACL compared with the medial collateral ligament. These findings suggest that the medial collateral ligament requires greater loads than the ACL to cause failure and may explain the higher rate of reported isolated ACL injuries compared with concomitant anterior cruciate and medial collateral ligament injuries.[102]

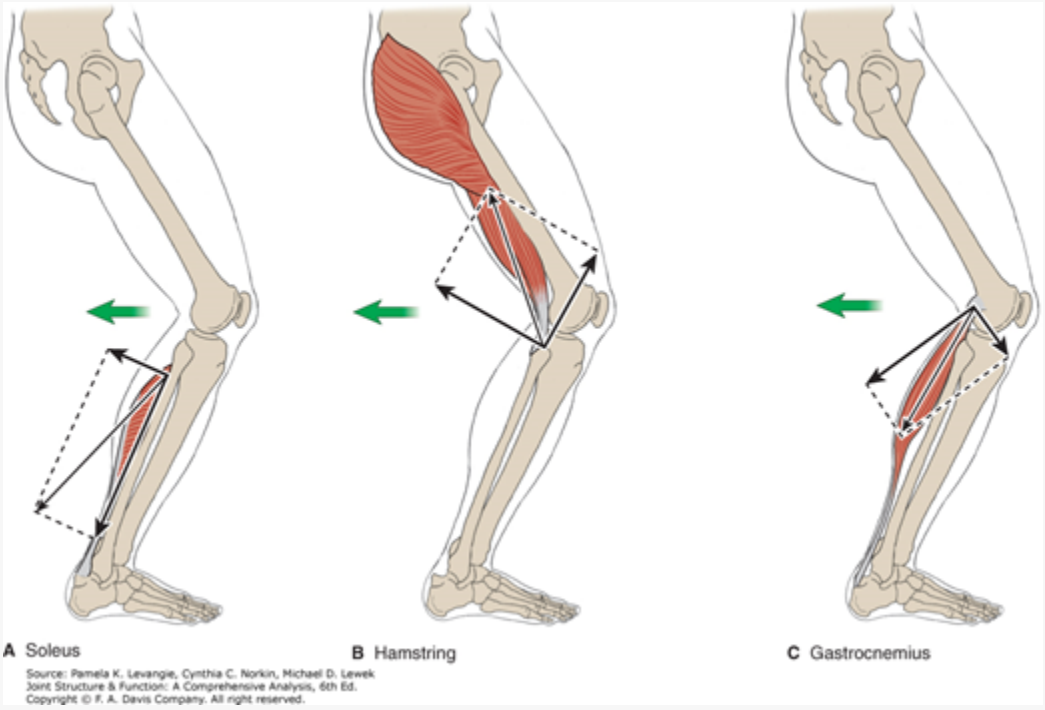
The muscles surrounding the knee joint are capable of altering (either increasing or decreasing) strain in the anterior cruciate ligament (**Fig. 11–18**). Because the ACL's primary role is to resist an anterior translation of the tibia on the femur, any muscle action that produces a shear force at the tibiofemoral joint will influence the strain on the ACL. For example, a muscle action that acts to translate the tibia anteriorly on the

femur will increase strain on the resisting ACL. In contrast, a muscle action that induces a posterior tibial translation force will put the ACL on slack and thus reduce the strain on the ligament. Specifically, with the tibiofemoral joint near full extension, an isolated quadriceps muscle contraction is capable of generating an anterior shear force on the tibia, thereby increasing strain in the ACL.[97,105–108] Fleming and colleagues reported that the gastrocnemius muscle similarly has the potential to translate the tibia anteriorly and increase strain on the ACL because the proximal tendon of the gastrocnemius wraps around the posterior tibia, effectively pushing the tibia forward when the muscle becomes tense through active contraction or passive stretch.[109] The **hamstring muscles** are capable of inducing a posterior shear force on the tibia throughout the range of knee flexion, becoming more effective in this role at greater knee flexion angles.[110–113] The hamstrings, therefore, have the potential to relieve the ACL of some of the stress of checking anterior shear of the tibia on the femur. With the foot on the ground, the soleus muscle may also have the ability to posteriorly translate the tibia and assist the ACL in restraining anterior tibial translation.[114]

FIGURE 11-18

Muscles that surround the knee joint are capable of producing tibial translation at the knee. **A.** A contraction of the soleus muscle acting on the tibia in weightbearing has a component that will produce posterior tibial translation at the knee. **B.** Similarly, a contraction of the hamstrings can produce posterior tibial translation. **C.** The gastrocnemius produces a posterior femoral translation (or anterior tibial translation) because of its proximal tendon wrapping around the

posterior tibia.



Given the potential of individual muscles to either increase or decrease loads on the ACL, it is not surprising that co-contraction of multiple muscles across the knee can influence the strain on the ACL. For example, co-contraction of the hamstrings and quadriceps muscles allows the hamstrings to counter the anterior translation imposed by the quadriceps to reduce the strain on the ACL at knee flexion angles of 60° and greater.[108] In contrast, activation of both the gastrocnemius and the quadriceps muscles results in greater strain on the ACL than either muscle would produce alone, unless hamstring co-contraction also occurs to mitigate the anterior translation imposed by the quadriceps and gastrocnemius.[109] Although muscular co-contraction will limit the strain imposed on the ligaments of the knee, it comes at a price. Specifically, greater muscle co-contraction will also increase joint compressive loads that could induce degenerative changes in the articular cartilage and other structures of

the knee.[110,115]

Patient Application 11–2

James is a 20-year-old man who injured his left knee when he attempted to make a cut while playing soccer. The patient was diagnosed with a complete ACL rupture and partial medial collateral ligament rupture following physical examination and magnetic resonance imaging (MRI) testing. In the weeks following his injury, James reported frequent episodes of his knee giving way, including buckling, shifting, or slipping of the knee during weightbearing activities. He also demonstrated decreased knee extension and flexion ROM, quadriceps muscle weakness, and knee joint effusion early after injury. After rehabilitation to resolve these initial impairments, James underwent ACL reconstruction using a hamstring-gracilis autograft. No surgical procedures were performed on the partially torn medial collateral ligament due to its rich blood supply and self-healing capacity. Three days after surgery, James returned to rehabilitation with the goal of returning to soccer. What considerations must be given when choosing therapeutic exercises to protect the healing ACL graft early after reconstruction?

The ultimate strength of the native ACL is approximately 2000 N with an ultimate strain of approximately 15%; however, during the first 2 to 4 weeks after reconstruction the ACL graft and its fixation is at its weakest and may be injured by lesser forces.[116–118] Therapeutic exercises within a rehabilitation program after ACL reconstruction must not produce forces that exceed the tensile properties of the new graft to avoid graft stretching or rupture. Various activities are routinely prescribed to improve muscle strength and joint function after anterior cruciate ligament tear or reconstruction. It should be noted, however, that it is currently unclear as to how much strain can be detrimental to an already damaged anterior cruciate ligament or a healing graft. Anterior cruciate ligament strain is typically greatest between 10° and 30° of knee flexion for both weightbearing and non-weightbearing exercises, and 0% strain in positions of greater than 60° of knee flexion.[118] Therefore, recommended exercises early after reconstruction typically avoid more extended knee joint positions.[119] However, strain values for both weightbearing and non-weightbearing exercises are relatively small in magnitude, and thus safe to incorporate later in rehabilitation as graft strength improves.[118,119] In addition, because the source of the graft was from the hamstring muscles, resisted knee flexion exercises should be avoided to allow adequate healing at the donor site.[119]

Expanded Concepts 11–4

Coping With Anterior Cruciate Ligament Deficiency

Certain individuals are capable of compensating successfully with a dynamic knee stabilization strategy in the presence of a complete anterior cruciate ligament rupture.[120–123] Quadriceps strength training and neuromuscular re-education may help facilitate proper dynamic control and normal knee motion after anterior cruciate ligament injury.[124–126] A percentage of athletes who cope well can even return to high-demand sports that require cutting and pivoting without reconstructing the passive restraint to anterior tibial translation.[121,127,128] However, not all patients return to pre-injury activity levels after sustaining an anterior cruciate ligament rupture regardless of operative and rehabilitative interventions.[129] In addition, evidence indicates that patients who choose a nonoperative approach are at no greater risk for developing knee osteoarthritis compared with those who undergo anterior cruciate ligament reconstructions.[130,131]

POSTERIOR CRUCIATE LIGAMENT

The posterior cruciate ligament (PCL) originates at the lateral aspect of the medial femoral condyle (see Fig. 11–16B) and travels inferiorly and somewhat posteriorly to attach distally to the posterior tibial surface, between the posterior horns of the two menisci (see Fig. 11–9).[132] Like the anterior cruciate ligament, the PCL is intracapsular but extrasynovial.[132] The PCL is a shorter and less oblique structure than the ACL, with a cross-sectional area 120% to 150% greater than that of the ACL.[133] The large size and shape of the PCL along with its extensive femoral attachment help it to resist greater loads before failing than the anterior cruciate ligament.[134] The PCL blends with the posterior capsule and periosteum as it crosses to its tibial attachment.[135] The vascular supply is provided by the middle genicular artery, like that of the anterior cruciate ligament.[136] Also like the anterior cruciate ligament, the PCL is typically divided into two bands that are named for their tibial origins: the **posteromedial bundle (PMB)** and

the **anterolateral bundle (ALB).**[137] When the knee is near full extension, the larger and stronger anterolateral bundle is lax, whereas the posteromedial bundle becomes taut. At 80° to 90° of flexion, the anterolateral bundle is maximally taut and the posteromedial bundle is relaxed.[138] However, in deep knee flexion, the femoral attachment of the posteromedial bundle translates anteriorly relative to its tibial attachment, giving the posteromedial bundle a more horizontal orientation.[132,134] This translation creates greater tension and the necessary orientation for the posteromedial bundle to increase its ability to resist posterior displacement of the tibia.[132] Concurrently, a more vertical orientation of the anterolateral bundle decreases its ability to resist posterior tibial displacement in full knee flexion.[132]

The PCL serves as the primary restraint to posterior displacement, or posterior shear, of the tibia beneath the femur.[132] The PCL restrains approximately 90% of the posterior load directed along the tibia across the majority of the knee flexion ROM; thus, sectioning of the PCL increases posterior translation at all angles of knee flexion.[139,140] In the fully extended knee, only minimal posterior displacements of the tibia can occur because of the multitude of ligaments that aid the PCL in resisting posterior tibial translation, including the posteromedial and posterolateral capsule, medial collateral ligament, lateral collateral ligament, arcuate, meniscofemoral, and fabellofibular ligaments.[136,141] The PCL is more adept at restraining posterior tibial motion with the knee flexed, unlike the anterior cruciate ligament, which resists force better at full extension.[95,136] Although maximal posterior displacement of the tibia occurs at 75° to 90° of flexion, the secondary restraints against posterior translation become ineffective in full flexion, enhancing the role of the PCL.[136] Like the anterior cruciate ligament, the PCL has a role in restraining varus and valgus stresses at the knee and appears to play a

role in both restraining and guiding rotation of the tibia.[141–143] The orientation of the PCL may result in a concomitant lateral rotation of the tibia when a posterior translational force is applied to the tibia. In the presence of PCL deficiency, lateral tibial translation is observed, which may alter the contact locations of the knee joint and serve as a mechanism for articular cartilage degeneration as similarly postulated in anterior cruciate ligament injury.[142]

In the absence of the PCL, muscles must be recruited to actively stabilize against excessive posterior tibial translation. The popliteus muscle shares the PCL's role in resisting posteriorly directed forces on the tibia, and can thus contribute to knee stability following PCL rupture.[133] In contrast, hamstring contraction can destabilize a flexed knee joint in the absence of the PCL because of the hamstring's induced posterior shear force on the tibia.[140] Contraction of the gastrocnemius muscle also significantly strains the PCL at flexion angles greater than 40°, whereas quadriceps contraction reduces the strain in the PCL at knee flexion angles between 20° and 60°.[105,140]

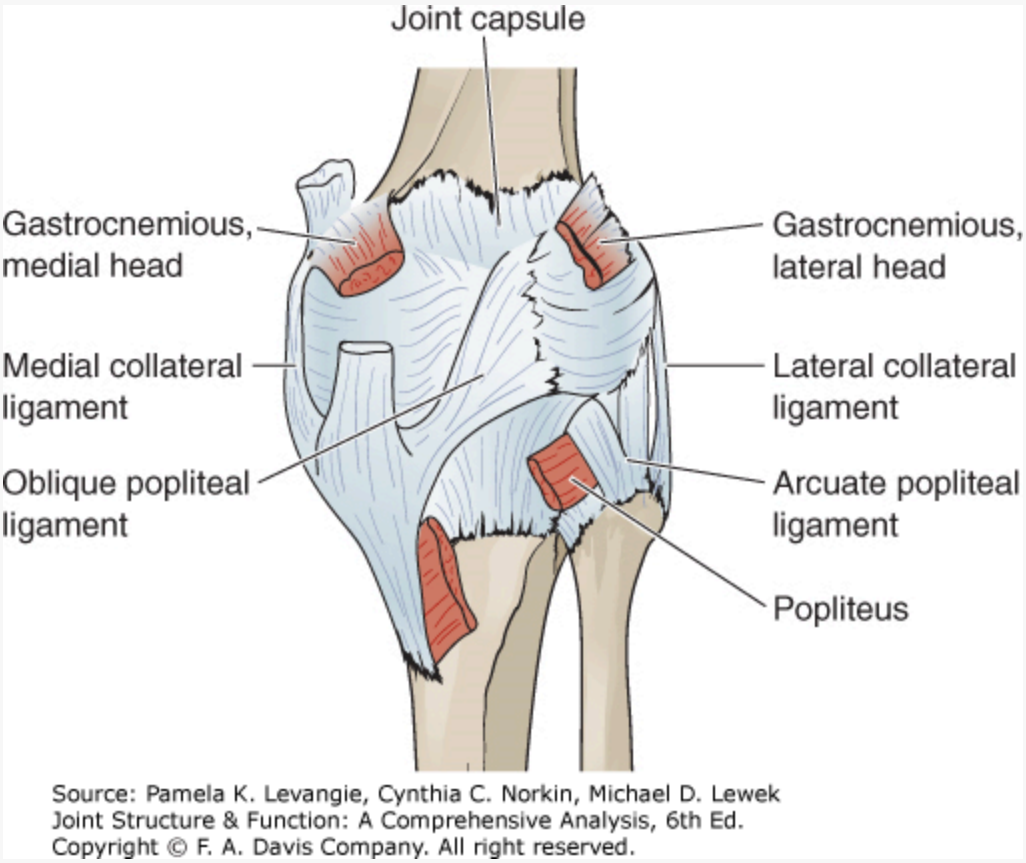
LIGAMENTS OF THE POSTERIOR CAPSULE

Several structures reinforce the "corners" of the posterior knee joint capsule (Fig. 11–19). The posteromedial corner of the capsule is reinforced by the semimembranosus muscle, by its tendinous expansion called the **oblique popliteal ligament,** and by the stronger and more superficial posterior oblique ligament.[144] The semimembranosus has an expansive attachment, consisting of up to eight portions distal to the main tendon, one of which is the oblique popliteal ligament.[145] The oblique popliteal ligament is the largest posterior knee structures and extends laterally across the

posterior aspect of the knee joint.[145] The posterior oblique ligament originates posterior to the adductor tubercle and anterior to the gastrocnemius tubercle on the femur.[69] Distally, the posterior oblique ligament attaches to the posterior capsule and medial meniscus, helping to reinforce the deep portion of the medial collateral ligament.[69] The posteromedial complex, specifically the posterior oblique ligament, is taut in full extension and assists in checking posterior tibial translation and valgus forces.[146] Thus, after trauma involving the posterior cruciate ligament and medial collateral ligament, the posterior oblique ligament and posteromedial capsule become important stabilizers against posterior tibial translation and valgus loads, respectively.[147]

FIGURE 11-19

A view of the posterior capsule of the knee joint shows the reinforcing oblique popliteal ligament. Also seen are the collateral ligaments (medial collateral ligament [MCL] and lateral collateral ligament [LCL]), the arcuate ligament, and some of the reinforcing posterior musculature (semimembranosus, biceps femoris, medial and lateral heads of the gastrocnemius, and the upper and lower sections of the popliteus muscles). The medially located posterior oblique ligament is not shown because it lies superficial to the other medial capsular structures.



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Joint Structure & Function: A Comprehensive Analysis, 6th Ed.
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The posterolateral corner of the capsule is primarily stabilized by the lateral collateral ligament, the popliteus tendon, and the popliteofibular ligament.[148] The popliteus tendon originates just anterior to the lateral collateral ligament, courses underneath the lateral collateral ligament, and runs obliquely to the posteromedial tibia.[148] The popliteofibular ligament originates at the musculotendinous junction of the popliteus muscle and attaches to the posteromedial aspect of the fibular head.[148] Other structures that comprise the posterolateral corner include the iliotibial band, biceps femoris tendon, lateral gastrocnemius tendon, lateral joint capsule, arcuate ligament, oblique popliteal ligament, and the fabellofibular ligament.[149] Together, these structures protect the posterolateral corner of the knee by restraining tibial lateral rotation and providing varus stabilization, which is provided primarily by the lateral

collateral ligament.[148] The posterolateral corner and posterior cruciate ligament work synergistically, as the posterolateral complex provides secondary stabilization to posterior tibial translation while the posterior cruciate ligament provides secondary stabilization to tibial lateral rotation. Thus, after injury to the posterior cruciate ligament the posterolateral complex becomes an important stabilizer against posterior tibial translation, whereas an injury to both can result in significant posterior tibial instability.[148]



Expanded Concepts 11–5

Meniscofemoral Ligaments

The meniscofemoral ligaments originate from the posterior horn of the lateral meniscus and insert on the lateral aspect of the medial femoral condyle either anterior to the posterior cruciate ligament **(ligament of Humphry)** or posterior to the posterior cruciate ligament (**ligament of Wrisberg;** see Fig. 11–9).[26,42,132] Because of their attachment to the lateral meniscus, they are not true ligaments. In addition, their presence within the knee is variable. In a review of the literature, Amis and colleagues reported that at least one of the meniscofemoral ligaments is present in 93% of knees, with only 50% of knees having both of the meniscofemoral ligaments.[134] The prevalence of having the posterior meniscofemoral ligament appears to be greater than the occurrence of the anterior meniscofemoral ligament.[134] Because the meniscofemoral ligaments assist the posterior cruciate ligament in restraining posterior translation of the tibia on the femur, they are sometimes considered to be part of the posterior cruciate ligament complex.[134] Nagasaki and colleagues have reported that, when present, the cross-sectional area of the mensicofemoral ligaments is 17.2% of the posterior cruciate ligament.[150]



Expanded Concepts 11–6

Anterolateral Ligament

The anterolateral ligament has recently received a considerable increase in attention and research. Its presence, anatomy, biomechanical role, and need for surgical

reconstruction have become topics of controversy and debate. Kennedy and colleagues defined the anterolateral ligament as a thickening of the lateral joint capsule attaching to the lateral epicondyle of the femur posterior and proximal to the attachment of the lateral collateral ligament and coursing anterodistally to insert onto the tibia midway between the insertion of the iliotibial band at Gerdy's tubercle and the fibular head.[151] Although the incidence of the anterolateral ligament is reported to be between 80% and 100%, there is a lack of consensus regarding its anatomical attachments.[152] Nevertheless, the anterolateral ligament is thought to provide anterior and rotational stability to the knee.[152] Thus, some have suggested reconstruction of the anterolateral ligament concurrently with the anterior cruciate ligament reconstruction to fully restore knee joint arthrokinematics.[153–155] However, other reports indicate the anterolateral ligament provides no additional tibiofemoral translation or rotation stabilization in the presence of anterior cruciate ligament deficiency or reconstruction, and thus challenge the necessity of anterolateral ligament reconstruction.[156,157]

Iliotibial Band

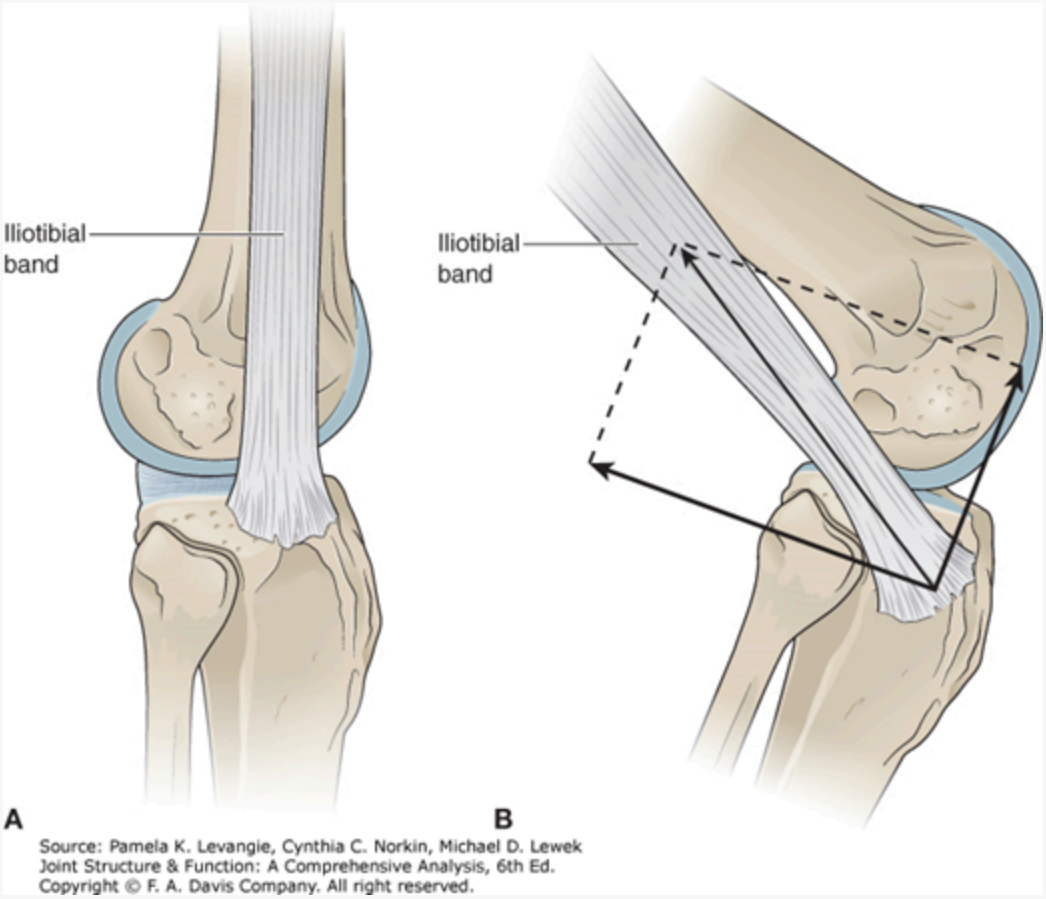
The iliotibial (IT) band or tract is an extension of fascia covering the tensor fascia latae (TFL) and gluteus maximus muscles that originates at the anterior superior iliac spine and external lip of the iliac crest.[149] The iliotibial band continues distally with a primary attachment on the anterolateral tibia at Gerdy's tubercle, but also attaches distally to the distal femur, lateral border of the patella, short head of the biceps femoris, and lateral head of the gastrocnemius, reinforcing the anterolateral aspect of the knee joint (see Fig. 11–15).[85] The iliotibial band can be viewed as (1) a tendinous portion between its proximal origin and its attachment to the lateral femoral epicondyle, and (2) a ligamentous portion between the lateral femoral epicondyle and Gerdy's tubercle on the tibia.

Despite the muscular attachments to the proximal end of the iliotibial band, it functions as a passive structure at the knee joint. For example, a contraction of the tensor fascia

lata or the gluteus maximus muscles that attach to the proximal end of the iliotibial band produces only minimal longitudinal excursion of the band distally. Despite limited longitudinal movements, the iliotibial band is thought to translate anterior to the knee joint axis as the knee is extended and posterior as the knee is flexed (Fig. 11–20).[149,158] However, cadaveric studies have challenged this concept that the iliotibial band moves or "rolls" over the lateral femoral epicondyle during knee flexion/extension because of its observed fibrous connections to the femoral epicondyle.[159] These studies have also failed to identify a bursa between the iliotibial band and the epicondyle of the femur that might become irritated, although adipose tissue is present between the iliotibial band and bony interface.[159–161] As the knee moves from full extension to 30° of flexion, compression of this highly vascularized and richly innervated adipose tissue increases between the iliotibial band and the lateral epicondyle. The adipose tissue is less compressed in full extension, which may account for the complaints of pain at the distal insertion of the iliotibial band around 30° of flexion and not at full extension in patients with iliotibial band symptoms.[159,162]

FIGURE 11-20

The iliotibial band provides lateral support to the knee joint. In the flexed knee, the iliotibial band tends to migrate posteriorly, increasing its ability to restrict excessive anterior translation of the tibia under the femur.



Additional fibrous connections from the iliotibial band to the biceps femoris, lateral gastrocnemius, and **vastus lateralis** muscles form a sling behind the lateral femoral condyle, which assists the anterior cruciate ligament in restraining posterior femoral (or anterior tibial) translation with the knee near full extension.[86,163] With the knee flexed, the combination of the iliotibial band, the lateral collateral ligament, and the popliteal tendon can provide even greater assistance in resisting anterior displacement of the tibia on the femur as well as increased stability to the lateral side of the joint.[160] Despite its lateral location, the iliotibial band alone provides only minimal resistance to varus loads and lateral joint space opening.[80,149] The iliotibial band also attaches to the patella via the lateral patellofemoral ligament of the lateral retinaculum. This portion of

the iliotibial band becomes tighter as the knee moves into a flexed position.[159] As we shall see later in this chapter, the attachment of the iliotibial band to the lateral border of the patella may have important implications for patellofemoral function.

Bursae

The extensive array of ligaments and muscles crossing the tibiofemoral joint, in combination with the large excursions of bony segments, creates the potential for substantial frictional forces among muscular, ligamentous, and bony structures. Numerous bursae, or fluid-filled sacs, however, prevent or limit such damaging forces. Three of the knee joint's bursae, the suprapatellar bursa, the **subpopliteal bursa,** and the **gastrocnemius bursa,** are not separate entities but rather are extensions of the capsule's synovium or communicate with the synovial lining of the joint capsule through small openings (see Fig. 11–11). The anteriorly located suprapatellar bursa lies between the quadriceps tendon and the anterior femur, superior to the patella. The posteriorly located subpopliteal bursa lies between the tendon of the popliteus muscle and the lateral femoral condyle. The gastrocnemius bursa lies between the tendon of the medial head of the gastrocnemius muscle and the medial femoral condyle. The gastrocnemius bursa continues beneath the tendon of the semimembranosus muscle to protect it from the medial femoral condyle.

The three aforementioned bursae that are connected to the synovial lining of the joint capsule allow the lubricating synovial fluid to move from recess to recess during flexion and extension of the knee. In extension, the posterior capsule and ligaments are taut, and the gastrocnemius and subpopliteal bursae are compressed. This shifts the synovial fluid anteriorly.[164,165] In flexion, the suprapatellar bursa is compressed and the

fluid is forced posteriorly. When the knee joint is in the semiflexed position (e.g., loose-packed position), the synovial fluid is under the least amount of pressure. When there is excess fluid within the joint cavity as a result of injury or disease **(joint effusion),** the semiflexed knee position helps to relieve tension in the capsule and, therefore, can minimize discomfort.

Besides the bursae that communicate with the synovial capsule, there are several other bursae associated with the knee joint (Fig. 11–11). The **prepatellar bursa,** located between the skin and the anterior surface of the patella, allows free movement of the skin over the patella during flexion and extension. The **infrapatellar bursa** lies inferior to the patella, between the patellar tendon and the overlying skin. Both the infrapatellar bursa and the prepatellar bursa may become inflamed as a result of direct trauma to the front of the knee or through activities such as prolonged kneeling. The **deep infrapatellar bursa,** located between the patellar tendon and the tibial tuberosity, helps to reduce friction between the patellar tendon and the tibial tuberosity.[166] This bursa is separated from the synovial cavity of the joint by the infrapatellar (Hoffa's) fat pad.[165,166] If the infrapatellar fat pad is removed, patellofemoral contact area decreases, which increases joint stress and the potential for contact surface irritation.[167] There are also several small bursae that are associated with the ligaments of the knee joint. A bursa is often present between the lateral collateral ligament and the biceps femoris tendon.[168] On the medial side of the joint, small bursae can be found both superficial and deep to the superficial portion of the medial collateral ligament to protect it from the deep portion of the medial collateral ligament and the tendons of the **semitendinosus** and **gracilis** muscles, respectively.[40]

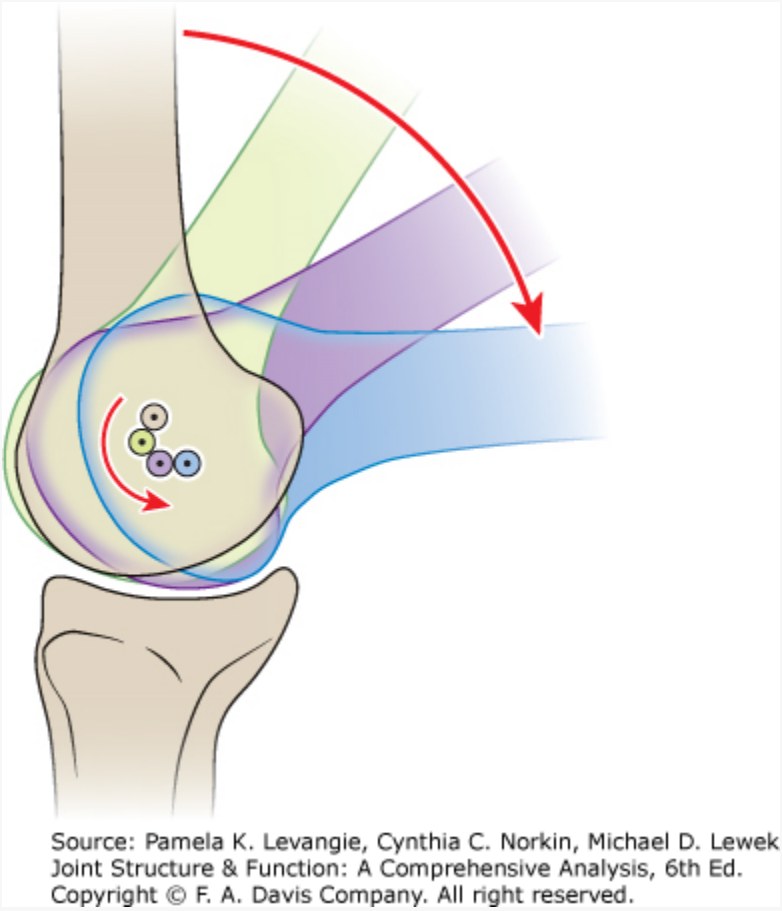
Function

Joint Kinematics

The primary angular (or rotary) motion of the tibiofemoral joint is flexion/extension, although small amounts of both medial/lateral (internal/external) rotation and varus/valgus (adduction/abduction) motions also take place. Angular motion is defined by movement of the distal segment (tibia) relative to the proximal segment (femur). Each of these motions occurs about changing, but definable, axes. The complex bony architecture between the femoral condyles and the tibial plateau results in a constantly changing center of rotation at the knee joint (Fig. 11–21). Current advances in motion analysis tracking systems, musculoskeletal modeling techniques, and imaging modalities have permitted accurate representation of three-dimensional descriptions of motion, including all six degrees of freedom (three rotational, three translational) at the tibiofemoral joint. Understanding the three primary axes of motion will provide the necessary framework to comprehend normal knee kinematics and to diagnose and treat abnormal movement patterns. In addition to angular motions at the knee, translation between the tibia and femur in an anteroposterior direction is common within the tibiofemoral compartments. Medial and lateral translations may also occur in response to varus and valgus forces, although to a significantly lesser extent. The small amounts of anteroposterior displacements that occur in the normal knee are the result of joint incongruence and variations in ligamentous elasticity. Importantly, these small anteroposterior translations are necessary for normal joint motions to occur. Excessive translational motions, however, should be considered abnormal and generally indicate some degree of ligamentous incompetence. We will focus on normal knee joint motions, including both osteokinematics and arthrokinematics.

FIGURE 11-21

A lateral view of the knee demonstrating the changing knee center of rotation along a medial-lateral axis during knee flexion and extension.



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
Joint Structure & Function: A Comprehensive Analysis, 6th Ed.
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FLEXION/EXTENSION

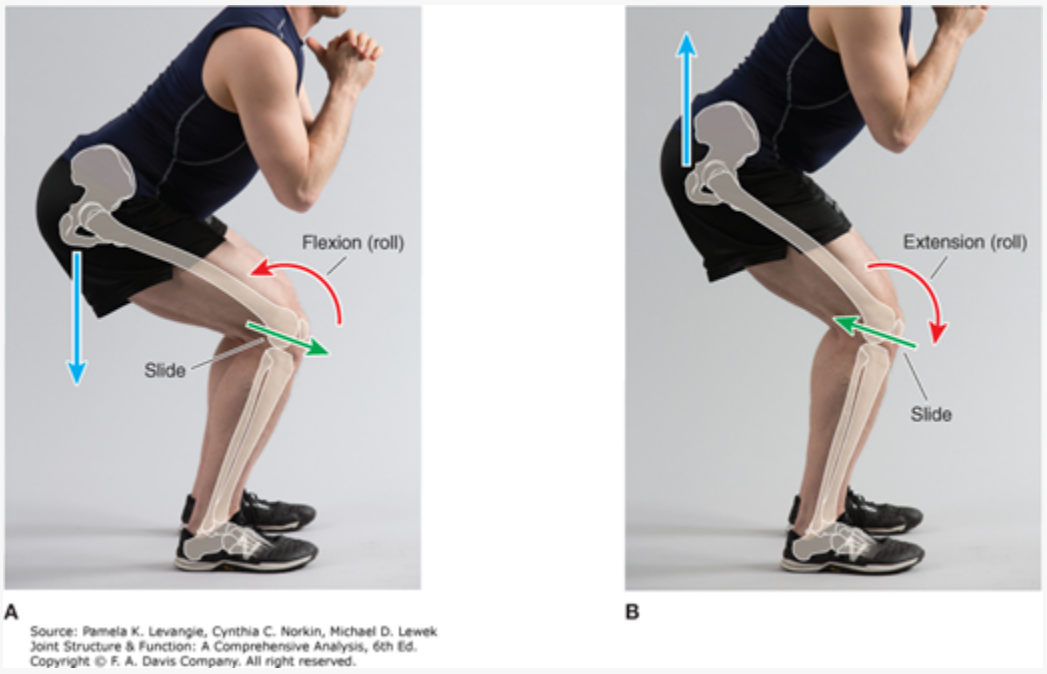
The axis for tibiofemoral flexion and extension can be simplified as a horizontal line passing through the femoral epicondyles.[2,169] Although this transepicondylar axis represents an accurate estimate of the axis for flexion and extension, it should be appreciated that this axis is not truly fixed but rather shifts throughout the ROM (Fig. 11–21). The shift in rotational axes of the knee can be largely attributed to the changing radius of curvature of the distal end of the femur.

Gliding motions must occur between the large articular surfaces of the femur and the relatively small tibial condyles as one joint surface is rolling over the other to maintain adequate joint contact. If the convex femoral condyles were permitted to roll posteriorly on a fixed, concave tibial plateau, the femur would “run out of” tibia to roll on, and limit flexion. For the femoral condyles to continue to roll posteriorly as flexion increases, the femoral condyles must simultaneously glide anteriorly on the tibial plateau (Fig. 11–22A). The initiation of knee flexion (0° to 25°), therefore, occurs primarily as posterior rolling of the femoral condyles on the tibia that moves the contact of the femoral condyles posteriorly on the tibial plateau. As flexion continues, the rolling of the femoral condyles is accompanied by a simultaneous anterior glide that creates a nearly pure spin of the femur on the posterior tibia with little linear displacement of the contact point on the tibial plateau after 25° of flexion. Extension of the knee from a flexed position is essentially a reversal of this motion (Fig. 11–22B). Tibiofemoral extension occurs initially as an anterior rolling of the femoral condyles on the tibial plateau, displacing the femoral condyles back to a neutral position on the tibial plateau. After the initial forward rolling, the femoral condyles glide posteriorly just enough to continue extension of the femur as an almost pure spin of the femoral condyles on the tibial plateau. These interdependent osteokinematic and arthrokinematic motions describe how the femur moves on a fixed tibia (e.g., during a squat). The tibia, of course, is also capable of moving on a fixed femur (e.g., during a seated knee extension or during the swing phase of gait; Fig. 11–23). In this case, the movements would be described somewhat differently, but represent the same relative movements. When the tibia is flexing on a fixed femur, the tibia both rolls and glides posteriorly on the relatively fixed femoral condyles (Fig. 11–23A). Extension of the tibia

on a fixed femur incorporates an anterior roll and glide of the tibial plateaus on the fixed femur (Fig. 11–23B). It is important to recognize that an anterior glide of the tibia on the femur represents the same movement as a posterior glide of the femur on the tibia. In the first case, we are simply describing the movement of the tibia on a fixed femur (i.e., non-weightbearing), whereas the second case describes the movement of the femur on a fixed tibia (i.e., weightbearing).

FIGURE 11-22

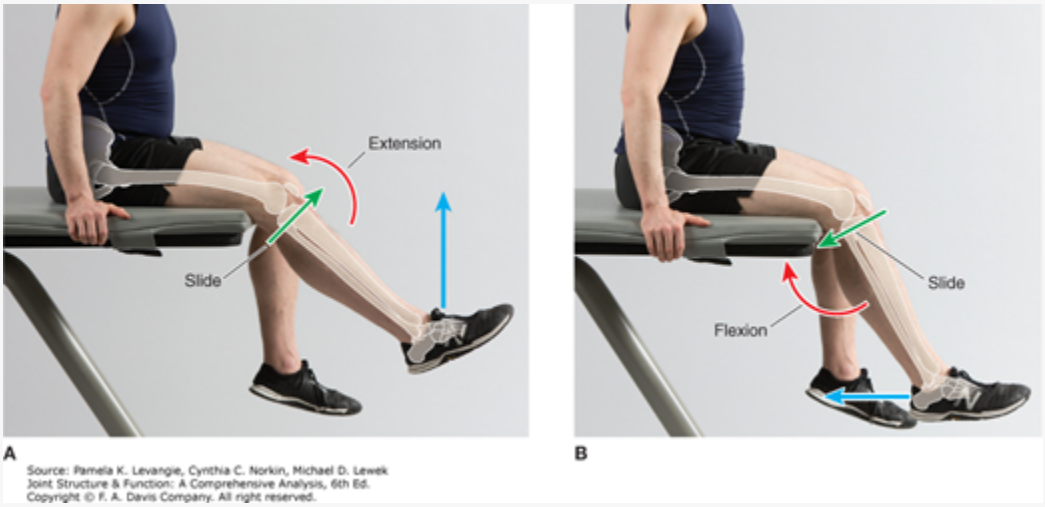
A. A schematic representation of rolling and gliding of the femoral condyles on a fixed tibia during knee flexion in a weight-bearing position. The femoral condyles roll posteriorly while simultaneously gliding anteriorly. **B.** Motion of the femoral condyles during extension. The femoral condyles roll anteriorly while simultaneously gliding posteriorly.



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FIGURE 11-23

A. A schematic representation of rolling and gliding of the tibial plateaus on a fixed femur during knee extension in a non-weightbearing position. The tibial plateaus roll anteriorly while simultaneously gliding anteriorly. **B.** Motion of the tibial plateaus during flexion. The tibial plateaus roll posteriorly while simultaneously gliding posteriorly.



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Expanded Concepts 11–7

Tibiofemoral Contact Points

Advancements in diagnostic imaging techniques and biomedical modeling techniques have improved the ability to accurately predict the contact points of cartilage in the knee. Li and colleagues used advanced diagnostic imaging techniques to model surface contact locations between the articular cartilage of the femur and tibia.[170] Three-dimensional motions were assessed to measure the contact points of the cartilage within the medial and lateral compartments of the knee. This investigation concluded that the contact points of the lateral tibial cartilage translated posteriorly with increased flexion, whereas the contact points of the medial tibia did not translate significantly during knee flexion. These results

suggest that a greater amount of rolling motion occurs between the lateral femoral condyle and the lateral tibial plateau compared with that of the medial compartment, whereas more sliding motion occurs between the medial femoral condyle and the medial tibial plateau.[170]

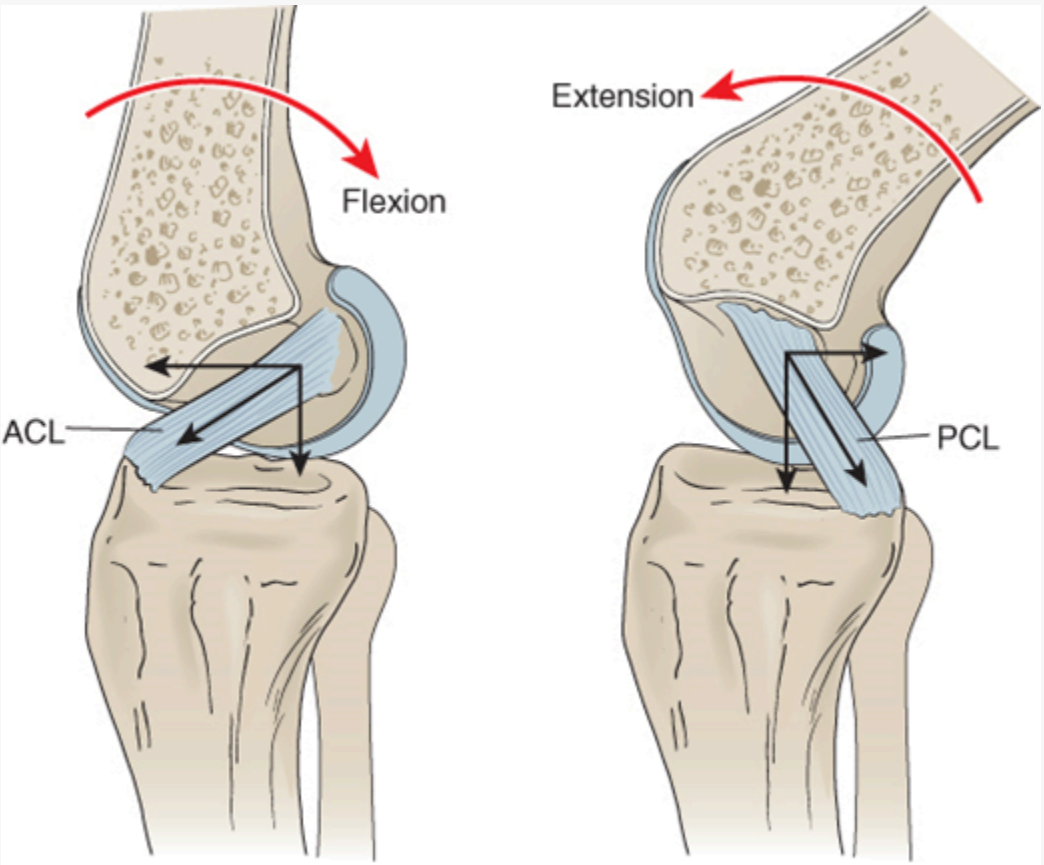
ROLE OF THE CRUCIATE LIGAMENTS AND MENISCI IN FLEXION/EXTENSION

The arthrokinematics associated with tibiofemoral flexion and extension are somewhat dictated by the presence of the cruciate ligaments (Fig. 11–24). If the cruciate ligaments are assumed to be rigid segments with a constant length, posterior rolling of the femur during weightbearing knee flexion would cause the "rigid" anterior cruciate ligament to tighten (or serve as a check rein).[171] Continued rolling of the femur would result in the taut anterior cruciate ligament simultaneously creating an anterior translational force on the femoral condyles (Fig. 11–24A).The posterior cruciate ligament would function in a similar manner during weightbearing knee extension. The femoral condyles roll anteriorly on the tibial plateau until the "rigid" posterior cruciate ligament checks further anterior progression of the femur, creating a posterior translational force on the femoral condyles (Fig. 11–24B).

FIGURE 11-24

Cross-section, sagittal view, of the knee. **A.** In flexion of the femur, posterior rolling of the femoral condyles creates tension in the "rigid" anterior cruciate ligament (ACL) that results in an anterior translational force imposed by the anterior cruciate ligament on the femur. **B.** In extension of the femur, anterior rolling of the femoral condyles creates tension in the "rigid" posterior cruciate ligament

(PCL), which results in a posterior translational force imposed by the posterior cruciate ligament on the femur.



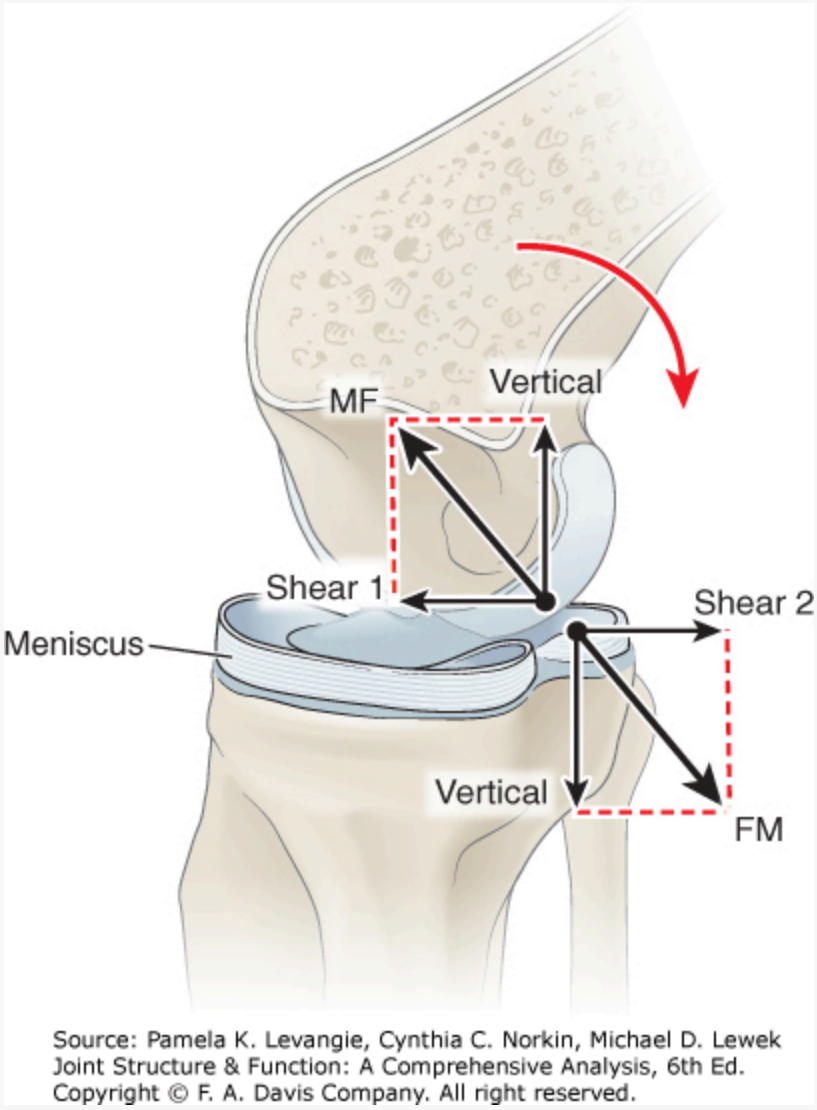
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The anterior glide of the femur during weightbearing flexion may be further facilitated by the shape of the menisci. As the femoral condyles roll posteriorly on the tibial plateaus, they must roll "uphill" with weightbearing knee flexion because of the circular "wedge" created by the menisci around the circumference of the tibial plateaus. The oblique contact force of the menisci on the femur helps guide the femur anteriorly during flexion, whereas the reaction force of the femur on the menisci deforms the menisci posteriorly on the tibial plateau (Fig. 11–25).[172] Posterior deformation of the

menisci occurs because the rigid attachments at the meniscal horns limit the ability of the menisci to move in its entirety.[172] Posterior deformation also allows the menisci to remain beneath the rounded femoral condyles during the rolling phase at the initiation of weightbearing knee flexion as the condyles move on the relatively flat tibial plateau. As the knee joint begins to return to extension from full flexion, the posterior margins of the menisci return to their neutral position. As extension continues, the anterior margins of the menisci are deformed anteriorly by the femoral condyles.

FIGURE 11-25

The oblique contact of the femur with the wedge-shaped meniscus results in the forces of meniscus-on-femur (MF) and femur-on-meniscus (FM). These can be resolved into vertical and shear (shear 1 and shear 2) components. Shear 1 assists the femur in its forward glide during flexion, and shear 2 assists in the posterior migration of the menisci that occurs with knee flexion.



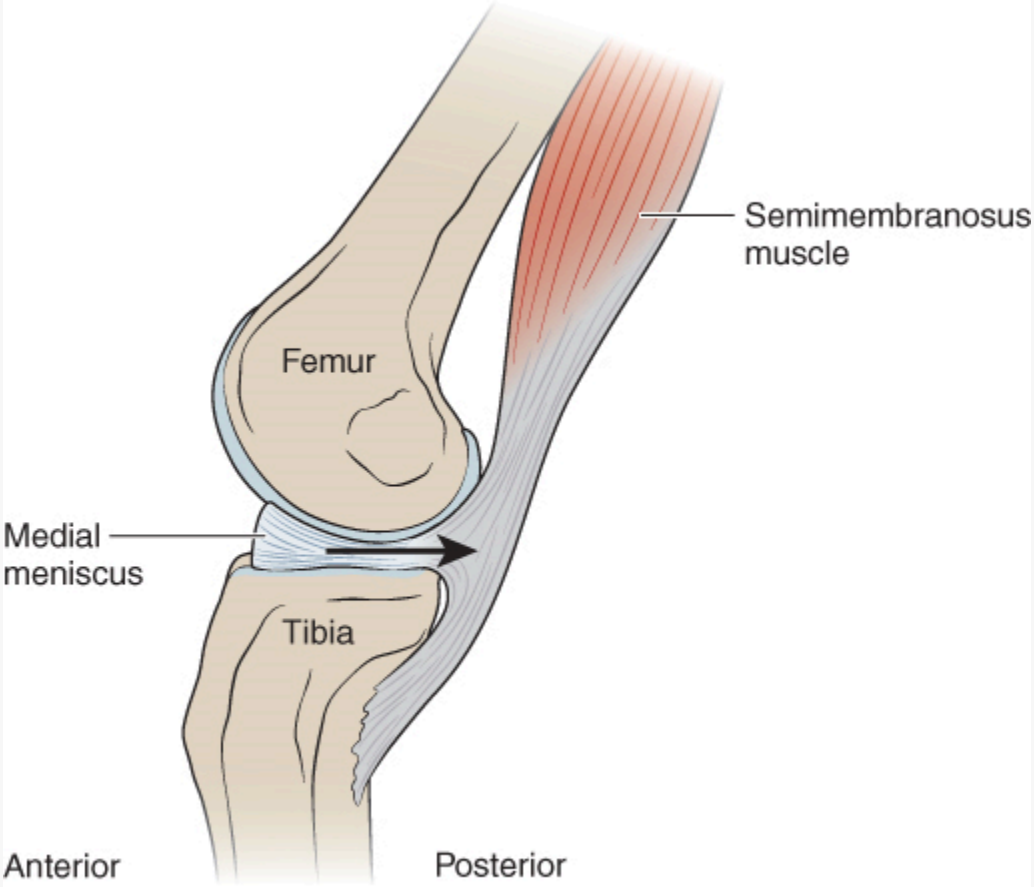
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The motion (or distortion) of the menisci is an important component of tibiofemoral flexion and extension. Given the need of the menisci to reduce friction and absorb the forces of the femoral condyles that are imposed on the relatively small tibial plateau, the menisci must remain beneath the femoral condyles to continue these functions. The posterior deformation of the menisci during flexion is assisted by muscular mechanisms to ensure that appropriate meniscal motion occurs. During knee flexion, for example, the semimembranosus muscle exerts a posterior pull on the medial

meniscus (Fig. 11–26), whereas the popliteus muscle assists with posterior deformation of the lateral meniscus.[40,44]

FIGURE 11-26

A schematic representation of the semimembranosus muscle and its attachment to the medial meniscus is shown. The arrow represents the direction of pull of the muscle on the medial meniscus during flexion.



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Expanded Concepts 11–8

Meniscal Motion

Failure of the menisci to distort in the proper direction can result in impairments in joint motion or damage to the menisci. If the femur literally rolls up the wedge-shaped menisci in flexion (without either the anterior glide of the femur or the posterior distortion of the menisci), the increasing thickness of the menisci and the threat of rolling off the posterior margin will cause flexion to be limited. Alternatively, the stress on the meniscus (especially the less mobile medial meniscus) may cause the meniscus to tear. Similarly, failure of the menisci to distort anteriorly during extension causes the thick anterior margins to become wedged between the femur and tibia as the segments are drawn together in the final stages of extension, thus limiting extension.

FLEXION/EXTENSION RANGE OF MOTION

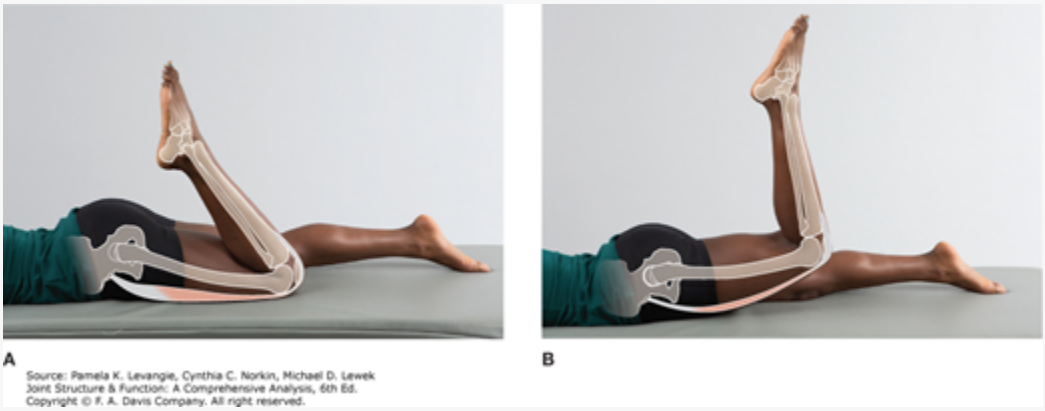
Passive range of knee flexion is generally considered to be 130° to 140°.[173] During an activity such as deep knee squatting, however, knee flexion may reach as much as 160° as the hip and knee are both flexed and the body weight is superimposed on the joint.[174] Normal gait on level ground requires approximately 60° to 70° of knee flexion, whereas ascending stairs requires about 80°, and sitting down into and arising from a chair requires 90° of flexion or more.[173] Over 90% of people demonstra te knee joint extension (or hyperextension) beyond 0°, and up to 5° to 10° is common.[175] Excessive knee hyperextension is termed **genu recurvaium.**[176]

Many of the muscles acting at the knee are two-joint (biarticulate) muscles crossing not only the knee but also the hip or ankle. Therefore, the hip or ankle joint position can influence the knee joint's ROM. For example, knee flexion can be limited to 120° or less if the hip joint is simultaneously extended due to passive insufficiency of the rectus femoris (Fig. 11–27). As a reminder, passive insufficiency is the inability of a biarticular muscle to lengthen sufficiently to complete full ROM at both joints simultaneously.

When the lower extremity is in weightbearing, ROM limitations at other joints, such as the ankle, may cause restrictions in knee flexion or extension.

FIGURE 11-27

A. With the hip in a neutral position the rectus femoris is able to lengthen sufficiently to allow knee flexion range of motion. **B.** With the hip in an extended position, passive insufficiency in the rectus femoris limits flexion at the knee joint.



A

B

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Expanded Concepts 11–9

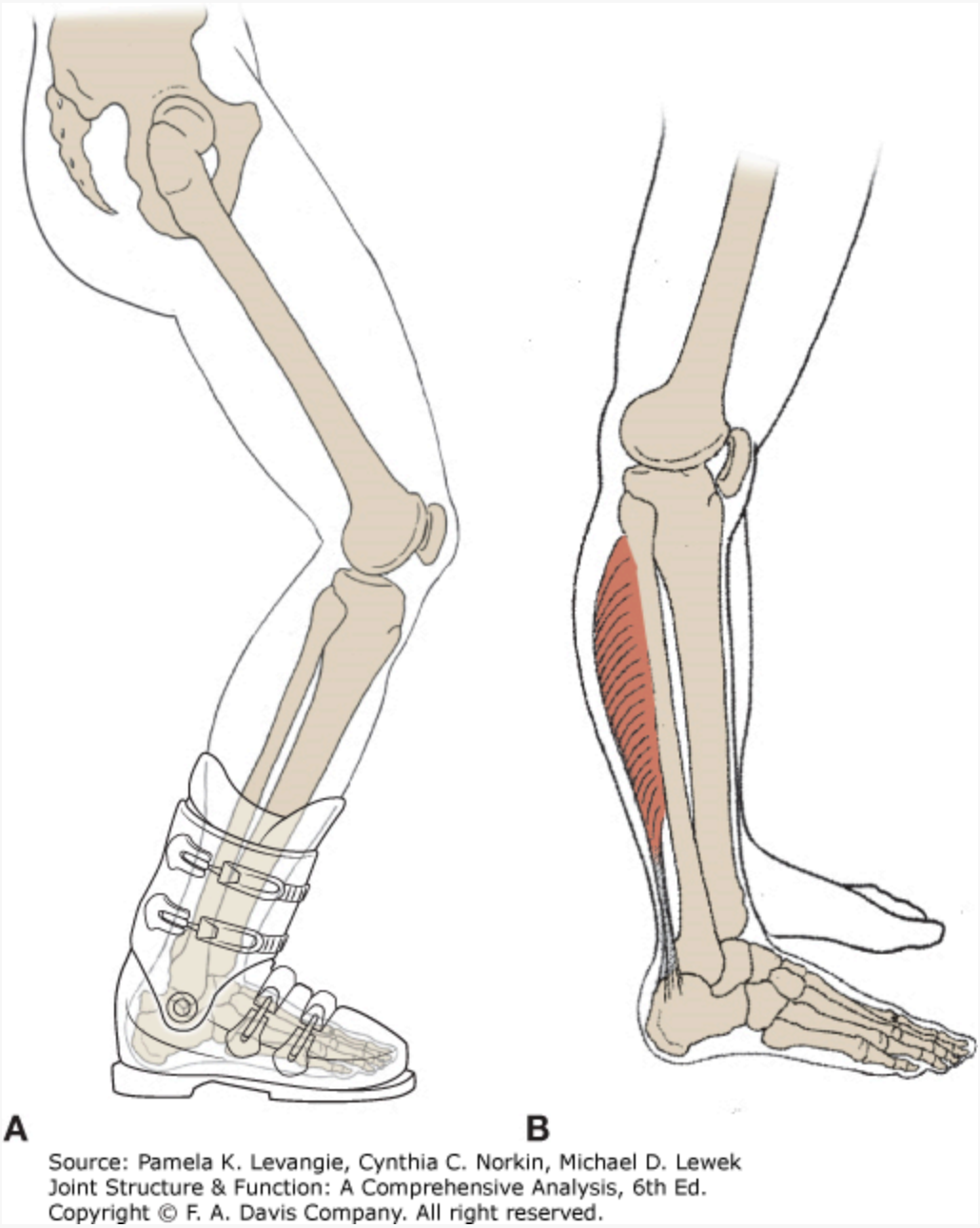
Ankle-Knee Interaction in Weightbearing

Ski boots generally hold the ankle in dorsiflexion, preventing full knee extension when the foot is on the ground (Fig. 11–28). This results in a flexed knee gait pattern or walking on one's heels (Fig. 11–28A). A similar problem can be created by a fixed dorsiflexion deformity in the ankle–foot complex. The opposite situation occurs with a limitation to dorsiflexion. A lack of ankle dorsiflexion (e.g., caused by tight plantarflexors) may limit the amount of knee flexion that can be performed before lifting the heel off the ground. For example, tight plantarflexors at the ankle can lead to the inability to bring the tibia forward in weightbearing and result in a hyperextension deformity (genu recurvatum) at the knee in standing (Fig. 11–28B). The relationship between ankle and knee position when the foot is on the ground can be exploited by intentionally altering ankle joint motion (e.g., through a heel lift or an

ankle–foot orthosis) to prevent or control knee motion.

FIGURE 11-28

A. With the ankle fixed in dorsiflexion by the ski boot, the knee cannot be fully extended without the forefoot being lifted from the ground. **B.** With a fixed plantarflexion deformity of the ankle/foot, the knee is forced into hyperextension when the foot is flat on the ground.



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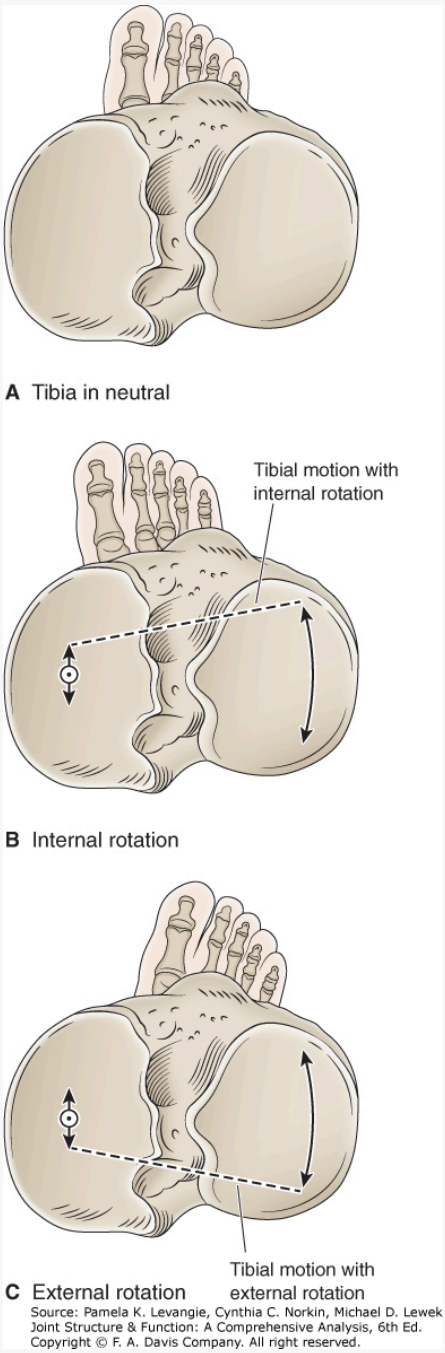
MEDIAL/LATERAL ROTATION

Medial and lateral (axial) rotations of the knee joint are angular motions that occur about a longitudinal axis that runs through or close to the medial tibial intercondylar tubercle.[3] Consequently, the medial condyle acts as the pivot point, whereas the lateral condyles move through a greater arc of motion, regardless of the direction of rotation

(Fig. 11–29). As the tibia laterally rotates on the femur, the medial tibial condyle moves only slightly on the relatively fixed medial femoral condyle, although the lateral tibial condyle moves a larger distance posteriorly on the relatively fixed lateral femoral condyle. During tibial medial rotation, the medial tibial condyle again rotates only slightly, whereas the lateral condyle moves anteriorly through a larger arc of motion. With the center of rotation located within the medial tibial plateau, the contact forces are focused on a smaller area on the medial femoral and tibial condyles, however the contact forces are distributed over a larger portion of the lateral femoral and tibial condyles. During both medial and lateral rotation, the knee joint's menisci will distort in the direction of movement of the corresponding femoral condyle and, therefore, will maintain their relationship to the femoral condyles just as they did in flexion and extension. For example, as the tibia medially rotates on the femur (or femur laterally rotates on the tibia in weightbearing), the medial meniscus will distort posteriorly on the tibial condyle to remain beneath the relatively posteriorly moving medial femoral condyle, and the lateral meniscus will distort anteriorly to remain beneath the anteriorly moving lateral femoral condyle. In this way, the menisci continue to reduce friction and distribute forces without restricting motion of the femur.

FIGURE 11-29

During medial/lateral rotation of the tibia, there is more motion of the lateral tibial condyle than of the medial tibial condyle in both directions; that is, the longitudinal axis for medial/lateral rotation appears to be located on the medial tibial plateau.



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Axial rotation is permitted by ligamentous laxity and the knee's unique bony anatomy, and its magnitude depends on the flexion/extension position of the knee. When the knee is in full extension, the ligaments are taut, the tibial tubercles are lodged in the intercondylar notch, and the menisci are tightly interposed between the articulating

surfaces; consequently, very little axial rotation is possible. As the knee flexes toward 90°, capsular and ligamentous laxity increases, the tibial tubercles are no longer within the intercondylar notch, and the condyles of the tibia and femur are free to move on each other. The greatest amount of axial rotation is therefore available at 90° of knee flexion. At 90°, the total medial/lateral rotation available is approximately 35°, with the range for lateral rotation being slightly greater (0° to 20°) than the range for medial rotation (0° to 15°).[177] The magnitude of axial rotation again decreases as the knee approaches full flexion.

VALGUS (ABDUCTION)/VARUS (ADDUCTION)

Frontal plane motion at the knee, although minimal, does exist and contributes to normal functioning of the tibiofemoral joint. Frontal plane ROM is typically only 8° at full extension, and 13° at 20° of knee flexion.[56,178] Excessive frontal plane motion can indicate ligamentous insufficiency. Muscles that cross the knee joint have the capacity to both generate and control substantial valgus and varus torques.[179–181] When there is ligamentous laxity, the excessive varus/valgus motion could increase peak stresses across one compartment (e.g., the lateral compartment would experience greater stress during valgus motion). Consequently, there could be an increase in activity of the muscles around the knee joint to control this excessive motion; however, greater muscle activity creates greater joint compression and thus may also precipitate greater peak stresses across the joint.[18,181]

COUPLED MOTIONS

Typical tibiofemoral motions are, unfortunately, not as straightforward as we have described. In fact, biplanar intra-articular motions can occur because of the oblique orientation of the axes of motion with respect to the longitudinal axes of the femur and

tibia. The true flexion/extension axis is not perpendicular to the shafts of the femur and tibia.[182] Therefore, flexion and extension do not occur as pure sagittal plane motions but include frontal and transverse plane components termed **coupled motions** (similar to coupling that occurs with lateral flexion and axial rotation in the vertebral column). As previously noted, the medial femoral condyle lies slightly distal to the lateral femoral condyle, which results in a physiological valgus angle in the extended knee that is similar to the physiological valgus angle that exists at the elbow. With knee flexion around the obliquely oriented axis, the tibia moves from a position oriented slightly lateral to the femur, to a position slightly medial to the femur in full flexion; that is, the foot approaches the midline of the body with knee flexion just as the hand may approach the midline of the body with elbow flexion. Therefore, extension is considered to be coupled to a valgus motion, whereas flexion is coupled with a varus motion.

There is an obligatory lateral rotation of the tibia that accompanies the final stages of knee extension that is not voluntary or produced by muscular forces. This coupled motion (lateral rotation with extension) is referred to as **automatic** or **terminal rotation.** We have already noted that the medial articular surface of the knee is longer (has more articular surface) than does the lateral articular surface (see Fig. 11–5). Consequently, during the last 30° of non-weightbearing knee extension (30° to 0°), the shorter lateral femoral condyle/tibial plateau pair completes its rolling-gliding motion before the longer medial articular surfaces.[183] As extension continues, the longer medial plateau continues to roll and glide anteriorly after the lateral side of the plateau has halted. This continued anterior motion of the medial tibial plateau on the medial femoral condyle results in lateral rotation of the tibia on the femur, with the motion most evident in the final 5° of extension.[182] Increasing tension in the knee joint ligaments, in particular the

cruciate ligaments, as the knee approaches full extension may also contribute to the obligatory rotational motion, bringing the knee joint into its close-packed or locked position with the tibial tubercles lodged in the intercondylar notch, the menisci tightly interposed between the tibial and femoral condyles, and the ligaments taut.[136,184] Consequently, automatic rotation is also known as the **locking** or **screw home mechanism** of the knee. To initiate knee flexion from full extension, the knee must first be "unlocked"; that is, the laterally rotated tibia cannot simply flex but must medially rotate simultaneously as flexion is initiated. A flexion force will automatically result in medial rotation of the tibia because the longer medial side will move before the shorter lateral compartment. Automatic rotation or locking of the knee is thought to occur during both weightbearing and non-weightbearing knee joint function. In weightbearing, this would require the freely moving femur to medially rotate on the relatively fixed tibia during the last 30° of extension. Unlocking would require lateral rotation of the femur on the tibia before flexion can proceed.

The motions of the knee joint, exclusive of automatic rotation, are produced to a great extent by the muscles that cross the joint. We will complete our examination of the tibiofemoral joint by first examining the individual contribution of the muscles, emphasizing their role in producing and controlling knee joint motion. We will then reexamine the muscles in combination with the passive joint structures of the knee as stabilizers of this very complicated joint.

Muscles

The muscles that cross the knee are typically thought of as either flexors or extensors, because flexion and extension are the primary motions occurring at the tibiofemoral

joint. Each of the muscles that flex and extend the knee also has a moment arm that is capable of generating both frontal and transverse plane motions, although the moment arms for these latter motions are generally small. Therefore, each of the muscles, although grouped as flexors and extensors, will also be discussed with regard to its role in controlling frontal and transverse plane motions.

KNEE FLEXOR GROUP

There are seven muscles that cross the knee joint posteriorly, and thus have the ability to flex the knee. These are the semimembranosus, semitendinosus, biceps femoris (long and short heads), sartorius, gracilis, popliteus, and gastrocnemius muscles. The **plantaris muscle** may be considered an eighth knee flexor, but it is commonly absent. With the exception of the short head of the biceps femoris and the popliteus, all of the knee flexors are two-joint muscles. As biarticulate muscles, the ability to produce effective force at the knee is influenced by the relative position of the other joint that the muscle crosses. Five of the flexors (the semimembranosus, semitendinosus, sartorius, gracilis, and popliteus muscles) have the potential to medially rotate the tibia on a fixed femur, whereas the biceps femoris has a moment arm capable of laterally rotating the tibia.[185] The lateral muscles (biceps femoris, popliteus, and the lateral head of the gastrocnemius) are capable of producing valgus (abduction) moments at the knee, whereas those on the medial side of the joint (semimembranosus, semitendinosus, sartorius, gracilis, and the medial head of the gastrocnemius) can generate varus (adduction) moments.[180]

The semitendinosus, semimembranosus, and the long and short heads of the biceps femoris muscles are collectively known as the hamstrings. Each of these muscles

attaches proximally to the ischial tuberosity of the pelvis, except the short head of the biceps, which originates on the posterior femur. The semitendinosus muscle attaches distally to the anteromedial aspect of the tibia by way of a common tendon with the sartorius and the gracilis muscles. The common tendon is called the pes anserinus because of its shape (pes anserinus means “goose’s foot”; see Fig. 11–14). The semimembranosus muscle inserts posteromedially on the tibia (and, as noted earlier, has fibers that attach to the medial meniscus that can facilitate posterior distortion of the medial meniscus during knee flexion).[40] Both heads of the biceps femoris muscle attach distally to the head of the fibula, with a slip to the lateral tibia. The short head of the biceps femoris muscle does not cross the hip joint and, therefore, acts only at the knee joint. The rest of the hamstring muscles cross both the hip (as extensors) and the knee (as flexors); therefore, their force-producing capability at the knee is affected by the angle of the hip joint. Greater hamstring force is produced with the hip flexed because the hamstrings are lengthened across the hip.[148] When the two-joint hamstrings are required to contract with the hip extended and the knee flexed to 90° or more, the hamstrings are shortened across both the hip and the knee. The hamstrings produce less force as knee flexion approaches the hamstring’s maximal shortened position (active insufficiency), and the hamstrings must also overcome the increasing tension in the lengthened rectus femoris muscle (passive insufficiency).[186] In non-weightbearing activities, the hamstrings generate a posterior shearing force of the tibia on the femur that increases as knee flexion increases, peaking between 75° and 90° of knee flexion.[187] This posterior translational force can reduce strain on the anterior cruciate ligament, although it increases the strain on the posterior cruciate ligament.[140]

The two heads of the gastrocnemius muscle originate from the posterior aspects of the

medial and lateral condyles of the femur and jointly attach distally to the calcaneal (or Achilles) tendon. Except for the small and often absent plantaris muscle, the gastrocnemius muscle is the only muscle that crosses both the knee and ankle joints. Much like the hamstrings' interaction with the hip joint, the gastrocnemius muscle's ability to produce knee flexor torque is dependent on the position of the ankle joint. The gastrocnemius muscle makes a relatively small contribution to knee flexion torque when compared with the large ankle plantarflexor torque it can produce. It generates the greatest knee flexion torque when the knee is in full extension.[188] As the knee is flexed, the ability of the gastrocnemius muscle to produce a knee flexion torque significantly decreases.[188] The gastrocnemius muscle often functions as more of a stabilizing muscle at the knee than a mobilizing muscle. For example, during gait, the gastrocnemius muscle works synergistically with the quadriceps to increase joint stiffness to stabilize the knee joint as the knee accepts the weight of the body during early stance phase.[114]

The sartorius muscle arises anteriorly from the anterior superior iliac spine (ASIS) and crosses the femur to insert into the anteromedial surface of the tibial shaft (most often as part of the common pes anserinus tendon; see Fig. 11–14). Variations in the distal attachment of the sartorius muscle are not uncommon and may be functionally relevant. When attached just anterior to its typical location, the sartorius muscle's line of action may fall anterior to the knee joint axis, serving as a mild knee joint extensor rather than as a knee flexor. Typically, however, the sartorius muscle functions as a flexor and medial rotator of the tibia. Despite its potential actions at the knee, activity within the sartorius muscle is more common during hip motion rather than with knee motion. During gait, the sartorius muscle is typically active only during the swing

phase.[189] The gracilis muscle arises from the symphysis pubis and attaches distally to the common pes anserinus tendon (see Fig. 11–14). The gracilis muscle functions primarily as a hip joint flexor and adductor, but also has the capability to produce weak flexion at the knee joint and produce slight medial rotation of the tibia. The three muscles of the pes anserine appear to function effectively as a group to resist valgus forces and provide dynamic stability to the anteromedial aspect of the knee joint.

The popliteus muscle is a relatively small single-joint muscle that attaches to the posterolateral aspect of the lateral femoral condyle and courses inferiorly and medially to attach to the posteromedial surface of the proximal tibia. The primary function of the popliteus muscle is to medially rotate the tibia on the femur.[185] The role of medially rotating the knee to unlock it during early knee flexion has been attributed to the popliteus muscle. However, it should be noted that unlocking is part of automatic rotation and is due in part to the obliquity of the joint axis and the anatomy of the articular surfaces. The obligatory medial rotation of the knee joint during early flexion is a coupled motion that would likely occur even with paralysis of the popliteus muscle. The popliteus muscle is also attached to the lateral meniscus and plays a role in deforming the lateral meniscus posteriorly during active knee flexion.[27] Together, the activity of both the semimembranosus and the popliteus muscles contribute to the posterior movement and deformation of their respective menisci on the tibial plateau while also generating a flexion torque at the knee. However, the menisci will move posteriorly on the tibial condyle even during passive flexion. Active assistance of the semimembranosus and popliteus muscles ensures that tibiofemoral congruence is maximized throughout the range of knee flexion assisting to keep both menisci beneath the femoral condyles. This actively assisted motion of the menisci minimizes the

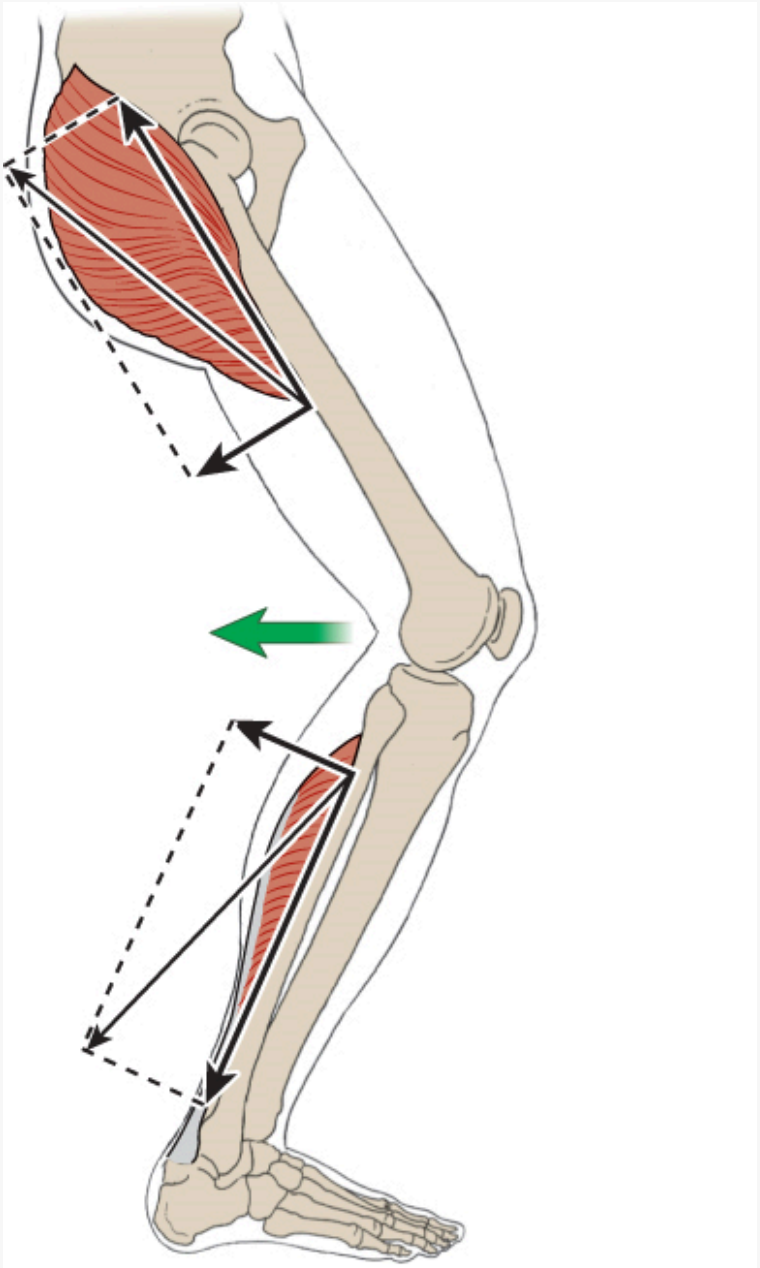
potential for meniscal entrapment, which could limit knee flexion and increase the risk of meniscal injury.

The soleus and gluteus maximus muscles do not cross the knee joint. Despite this, both can still influence function at the knee during weightbearing activities. The soleus muscle originates on the posterior aspect of the proximal tibia and fibula and attaches distally to the calcaneal tendon. With the foot fixed on the ground during weightbearing activity, a soleus muscle contraction can assist with knee extension by pulling the tibia posteriorly (Fig. 11–30). As noted earlier, the posterior pull of the soleus on the weightbearing leg can also assist the hamstrings in restraining excessive anterior displacement of the tibia.[114] The gluteus maximus muscle, like the soleus muscle, is capable of assisting with knee extension during weightbearing activities. It is well-known that the large muscle mass of the gluteus maximus functions well as a hip extensor. With the foot flat on the ground and the knee bent, a contraction of the gluteus maximus can also influence more distal joints by generating knee extension and ankle plantarflexion. The gluteus maximus, however, could also produce a posterior shear of the femur on the tibia (or a relative anterior shear of the tibia on the femur), which would increase tension in the anterior cruciate ligament without an offsetting co-contraction of other muscles.

FIGURE 11-30

The actions of the gluteus maximus and soleus muscles can influence knee motion in weightbearing. Although they do not cross the knee joint, these muscles are capable of assisting with knee extension with

the foot on the ground.



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KNEE EXTENSOR GROUP

The four extensors of the knee (rectus femoris, vastus lateralis, vastus medialis, and **vastus intermedius**) are known collectively as the quadriceps femoris muscle. The only

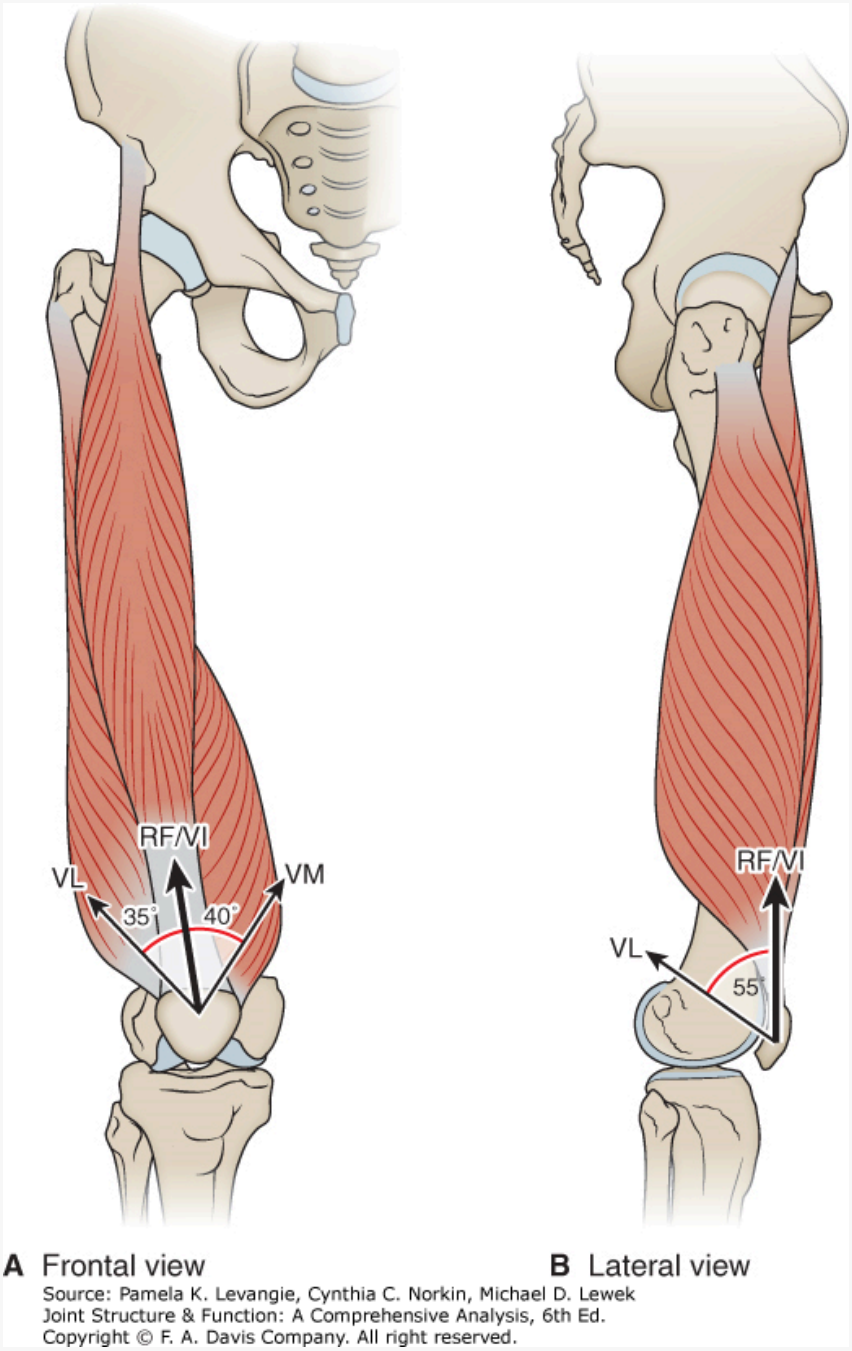
portion of the quadriceps that crosses two joints is the rectus femoris muscle, which crosses the hip and knee from its proximal origin on the anterior inferior iliac spine. The vastus intermedius, vastus lateralis, and vastus medialis muscles originate on the femur and merge with the rectus femoris muscle into the common quadriceps tendon. The quadriceps tendon inserts into the proximal aspect of the patella and then continues distally past the patella, where it is known as the patellar tendon (or **patellar ligament).** The patellar tendon runs from the apex of the patella to the proximal portion of the tibial tuberosity. The vastus medialis and vastus lateralis also insert directly into the medial and lateral aspects of the patella by way of the retinacular fibers of the joint capsule (see Fig. 11–12).

Together, the four muscles of the quadriceps femoris function to extend the knee. Each muscle is oriented differently from the others, resulting in different lines of pull relative to the femoral shaft (**Fig. 11–31**). Biomechanical testing has shown that the resultant pull of the vastus lateralis muscle on the long axis of the femur is 35° laterally, whereas the resultant pull of the vastus medialis muscle is 40° medially (**Fig. 11–31A**).[190] The pull of the vastus intermedius muscle is parallel to the shaft of the femur, making it the purest knee extensor of the group.[191] Because of the pennate nature of the quadriceps muscles, not all fibers within each muscle are oriented in the same direction. For example, the upper fibers of the vastus medialis are angled 15° to 18° medially to the femoral shaft, whereas the distal fibers are angled as much as 50° to 55° medially.[191,192] Because of the drastically different orientation of the upper and lower fibers of the vastus medialis muscle, the upper fibers are sometimes referred to as the **vastus medialis longus (VML),** and the lower fibers are referred to as the **vastus medialis oblique (VMO).** The obliquity of the distal portion of the vastus medialis muscle has

become the focus of attention in patients with patellofemoral pain as clinicians and researchers have attempted to try to preferentially recruit the vastus medialis oblique to maximize its medial pull on the patella. It should be noted, however, that despite the different orientation of the fibers of the vastus medialis oblique and the vastus medialis longus, these fibers are simply portions of the same muscle innervated commonly by the femoral nerve.[192,193] Isolated atrophy of the vastus medialis oblique does not occur in patients with patellofemoral pain, nor is selective activation of these fibers possible.[194] Lieb and Perry found the resultant pull of the combined four portions of the quadriceps femoris muscle to be 7° to 10° in the lateral direction, and 3° to 5° anteriorly in relation to the long axis of the femur.[191] Powers and colleagues have reported a posterior attachment site of the vastus lateralis and vastus medialis muscles, resulting in a net posterior or compressive force that averages 55° in the extended knee (Fig. 11–31B).[190] The compressive force from these muscles on the patella is present throughout ROM, but is minimized at full extension and increases as knee flexion continues.

FIGURE 11-31

A. With the data from Powers and colleagues, the orientation of the four components of the quadriceps muscle are shown, including the vastus lateralis (VL), vastus intermedius (VI), rectus femoris (RF), and vastus medialis (VM).[190] **B.** The posteriorly directed vectors of the VL and VM (VL shown) were found to result in net compression of the patella against the tibia, even in full extension.



Patellar Influence on Quadriceps Muscle Function

Function of the quadriceps muscle is strongly influenced by the patella (which, in turn, is strongly influenced by the quadriceps, as will be discussed later). From the perspective of mechanical advantage, the patella lengthens the moment arm of the

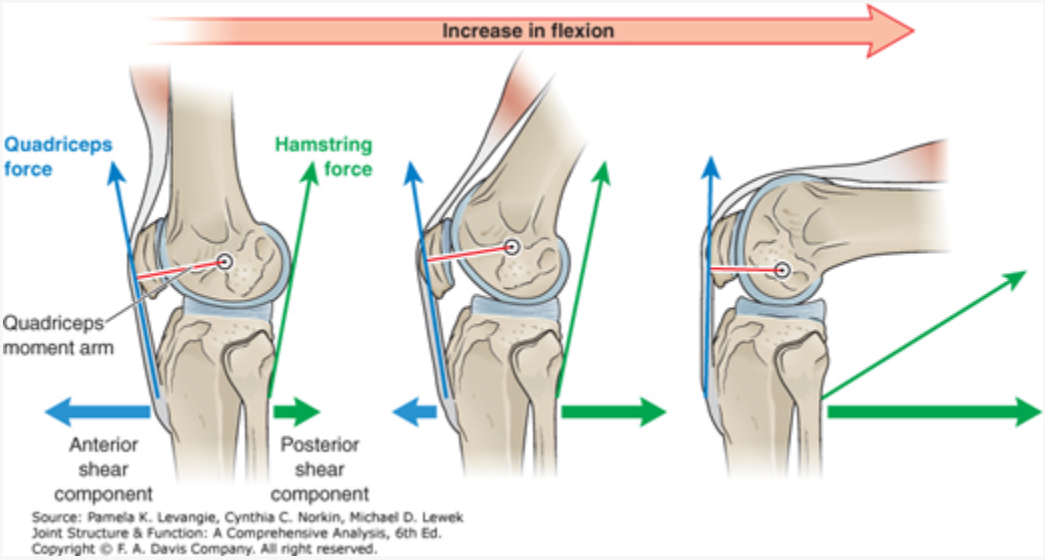
quadriceps by increasing the distance of the quadriceps tendon and patellar tendon from the axis of the knee joint. The patella functions as an anatomical pulley, deflecting the action line of the quadriceps femoris muscle away from the joint center and increasing the angle of pull on the tibia to enhance the ability of the quadriceps to generate extension torque. The patella does not, however, function as a simple pulley. In a simple pulley the tension is equal on either side of the pulley; in contrast, the tension in the patellar tendon on the inferior aspect of the patella is rarely equal to the tension in the quadriceps tendon at the superior aspect of the patella.[195]

The knee joint's geometry and the patella together dictate the quadriceps' angle of pull on the tibia as the knee flexes and extends (**Fig. 11–32**). The patella plays a primary role in increasing the quadriceps' angle of pull when the knee is in extended positions. In full knee flexion, the patella's function as a pulley is significantly reduced because the patella is fixed firmly inside the intercondylar notch of the femur. Despite this, the quadriceps maintains the ability to produce considerable torque in full knee flexion because of a large moment arm created by the rounded contour of the femoral condyles. The condyles deflect the muscle's line of action, and with the posteriorly shifted axis of rotation a relatively large moment arm is present with little contribution from the patella (Fig. 11–21). As the knee extends from full flexion, the moment arm of the quadriceps muscle lengthens as the patella leaves the intercondylar notch and begins to travel up and over the rounded femoral condyles. At about 50° of knee flexion, the femoral condyles have pushed the patella to its farthest distance from the knee's axis of rotation. With continued extension, the moment arm will once again diminish.[196] The influence of the changing moment arm on quadriceps torque production is readily apparent when knee extension strength is measured throughout the ROM. Peak torques

are often observed at approximately 45° to 60° of knee flexion, a region in which both the moment arm and the length-tension relationship of the muscle are maximized.[197]

FIGURE 11-32

The patella acts as a pulley for quadriceps muscle force production. Changes in the knee joint center of rotation and position of the patella within the femoral sulcus influences the magnitude of the moment arm of the quadriceps muscle.



Although the patella's effect on the quadriceps' moment arm is diminished in the final stages of knee extension, the small improvement in joint torque provided by the patella may be most important here. The quadriceps is in a shortened position near end range extension, which reduces its ability to generate active tension. The decreased ability of the quadriceps to produce active force makes the relative size of the moment arm critical to torque production in the last 15° of knee extension.

Expanded Concepts 11–10

Quadriceps Lag

The quadriceps may not be able to produce adequate torque to complete the last 15° of non-weightbearing knee extension if there is substantial quadriceps weakness or if the patella has been removed because of trauma (a procedure known as a patellectomy). The inability of a patient to fully extend the knee actively is referred to clinically as “quad lag” or “extension lag.” For example, the patient may have difficulty maintaining full knee extension while performing a straight leg raise (**Fig. 11–33**). With the tibiofemoral joint in greater flexion, removal of the patella or quadriceps weakness will have less effect on the ability of the quadriceps to generate extension torque because (1) the femoral condyles also serve as a pulley to increase the quadriceps’ moment arm and (2) the relatively lengthened muscle can produce greater force because of its position on the length-tension curve. The patient likely will not have a “quad lag” during weightbearing activities because with the foot fixed the soleus and gluteus maximus muscles can assist the quadriceps with knee extension.

FIGURE 11-33

Severe quadriceps weakness can result in a quadriceps lag (“quad lag”) during a straight-leg-raise exercise. Near full extension, the patella is only capable of increasing the moment arm slightly, and the decreased length-tension relationship of the already weakened quadriceps renders the quadriceps incapable of generating sufficient torque to complete the extension range of motion.



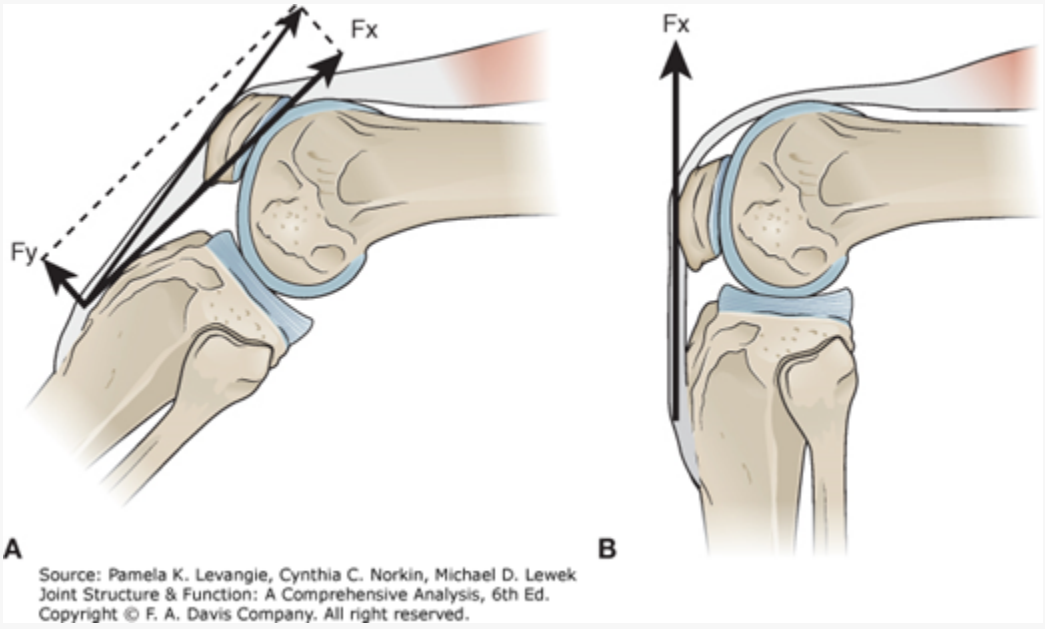
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The patella's role in increasing the quadriceps' angle of pull on the tibia enhances torque production but can also lead to negative side effects (Fig. 11–34). The quadriceps muscle group not only produces an extension torque but also creates an anterior shear of the tibia on the femur at various knee flexion angles, depending on the task (Fig. 11–34A).[118] This anterior translational force imposed by the quadriceps must be resisted by either actively producing a posterior tibial shear force, or passively resisting the anterior tibial translation with a passive restraint. The anterior cruciate ligament represents the most prominent passive restraint to the imposed anterior tibial translation of the quadriceps. Increases and decreases in the angle of pull of the quadriceps are accompanied by concomitant increases and decreases in stress in the anterior cruciate ligament.[118] The strain on both bundles of the anterior cruciate ligament ordinarily increases as the knee joint approaches full extension. In the

absence of passive stabilizers such as the anterior cruciate ligament, a quadriceps contraction near full extension has the potential to generate a large anterior tibial translation, which the patient may describe as the knee “giving way” or “buckling”.[107] The strain on the anterior cruciate ligament evoked by a quadriceps contraction is substantially diminished as the knee is flexed beyond 60° because the shear component of the quadriceps diminishes from its maximum value (Fig. 11–34B).

FIGURE 11-34

A. With the knee close to full extension, a forceful quadriceps contraction is capable of inducing an anterior tibial translation. **B.** Once the knee is flexed to greater than 60°, little to no anterior translation occurs.



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During weightbearing activities, the quadriceps’ activity is influenced by a number of other factors. Muscles such as the soleus and gluteus maximus muscles are capable of

assisting with knee joint extension. During erect standing postures, activity of the quadriceps is minimal because the line of gravity passes just anterior to the knee axis for flexion/extension, creating an extension torque that maintains the joint in extension. The posterior joint capsule, ligaments, and posterior muscles offset the small external extension torque, preventing further knee joint extension. In weightbearing with the knee somewhat flexed, as during a squat or when someone cannot fully extend the knee (e.g., flexion contraction), the line of gravity will pass posterior to the knee joint axis. The gravitational torque will promote knee flexion, and activity of the quadriceps is necessary to oppose the gravitational torque and maintain the knee joint in equilibrium. Because the quadriceps femoris muscle is responsible for supporting the body weight and resisting the force of gravity, it is often over 50% stronger than the hamstring muscles.[197] Although the hamstrings perform a similar function in supporting the body weight when there is a gravitational flexion moment at the hip, the hamstrings are assisted in this function by the large gluteus maximus. In contrast, the quadriceps are the primary muscle for supporting the knee joint against large external torques imposed by gravity.

The quadriceps muscle functions differently because of different biomechanical demands of various activity and exercise conditions. During seated (non-weightbearing) knee extension, the moment arm of the resistance (e.g., weight of the leg plus external resistance) is minimal when the knee is flexed to 90°, but increases as knee extension progresses (**Fig. 11–35**). Therefore, greater quadriceps force is required as the knee approaches full extension. The opposite occurs during weightbearing activities. In a standing squat, the moment arm of the resistance (e.g., the effect of gravity on the body's center of mass) is minimal when the knee is extended, and increases as knee

flexion increases. Therefore, during weightbearing activities such as a squat, the quadriceps muscle must produce greater force in positions of greater knee flexion (Fig. 11–36).[198]

FIGURE 11-35

During non-weightbearing exercises, the quadriceps muscle must generate more torque (and more force) as the knee approaches full extension to overcome the increasing moment arm (and torque) of the resistance.


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FIGURE 11-36

In a weightbearing exercise, the quadriceps must generate more torque (and more force) as knee flexion increases to control the increasing moment arm (and torque) of the superimposed body weight at the knee joint.



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Expanded Concepts 11–11

Quadriceps Strengthening: Weightbearing Versus Non-Weightbearing

Weightbearing exercises are often prescribed after anterior cruciate ligament, posterior cruciate ligament, or patellofemoral injury on the premise that these exercises are less stressful to the joints, more like functional movements, and safer than non-weightbearing exercises. W ilk and colleagues computationally estimated anteroposterior shear force, compression force, and extensor torque at the knee during weightbearing and non-weightbearing exercises that are commonly used for quadriceps muscle strengthening).[199] Weightbearing quadriceps exercises of a squat and leg press resulted In a (posterior tibial shear force at the knee throughout the entire ROM, peaking at between 83° and 105 ° of knee flexion.[199,200] The posterior shear would presumably increase the stress on the posterior cruciate ligament. In contrast, there was an anterior shear force in a non-weightbearing knee extension exercise when the quadriceps actively extended the knee from 40° to 10°, with the maximal anterior shear force occurring between 20° and 10° of knee flexion. The anterior shear forces would presumably increase the stress on the anterior cruciate ligament. Stresses on the posterior and anterior cruciate ligament during certain

types of weightbearing and non-weightbearing exercises may actually be detrimental to the healing process if these ligaments are damaged or recently surgically reconstructed.[199,200]

Beynnon and colleagues measured anterior cruciate ligament strain values in vivo to compare strain values during commonly performed therapeutic exercises.[201,202] Similar anterior cruciate ligament strain values were found regardless of whether the exercises were performed during weightbearing or non-weightbearing activities.[201,202] For example, weightbearing one-legged sit-to-stands, lunges, step ups and step downs produced anterior cruciate ligament strain values similar to active non-weightbearing knee extension exercises. Anterior cruciate ligament strains were higher when the quadriceps contracted between 50° to 0° of knee flexion regardless of whether the exercise was performed closed or open chain.[201,202]

The findings of these computationally derived forces and experimentally measured strains in the cruciate ligaments cast doubt on the rationale of basing rehabilitation exercises on the premise that weightbearing or non-weightbearing protocols are inherently better, safer, or more effective. Instead, a rehabilitation program incorporating both types of exercises and the functional requirements of the patient will likely maximize quadriceps strength gains.

Stabilizers of the Knee

Throughout the chapter, we have identified the role of both passive (capsuloligamentous) and active (muscular) forces in contributing to stability of the tibiofemoral joint. The contributions of both capsuloligamentous and muscular structures to maintaining appropriate joint stability are dependent on the position of the knee joint as well as the surrounding joints, the magnitude and direction of the applied force, and the availability of secondary restraints. There can also be considerable variation among individuals (as well as between knees in the same individual) that contributes to the diversity of findings observed by both clinicians and researchers. Although admittedly an oversimplification, Table 11–1 summarizes the potential contribution of the different structures that limit anteroposterior translation,

knee joint hyperextension, varus/valgus rotation, and medial/lateral rotation of the knee joint.

Table 11-1

Summary of Knee Joint Stabilizers*

Table 11–1 describes stability in terms of single plane movements. In reality, as shown in Table 11–2, more complicated, combined motions normally occur, requiring coupled stability, or rotary stability (a combination of uniplanar motions). For example, injury to the posterolateral corner (e.g., posterolateral joint capsule, popliteus muscle, arcuate ligament) can yield posterior instability and excessive lateral tibial rotation. This is termed **posterolateral instability.** In contrast, damage to the posterior oblique ligament, medial hamstrings, medial collateral ligament, and posteromedial joint capsule contributes to **posteromedial instability.** Anteromedial and anterolateral stability is provided by the respective portions of the joint capsule, which fuses with extensions of the quadriceps femoris muscle that forms the extensor retinaculum on each side of the patella.

Table 11-2

Components to Rotary Stability

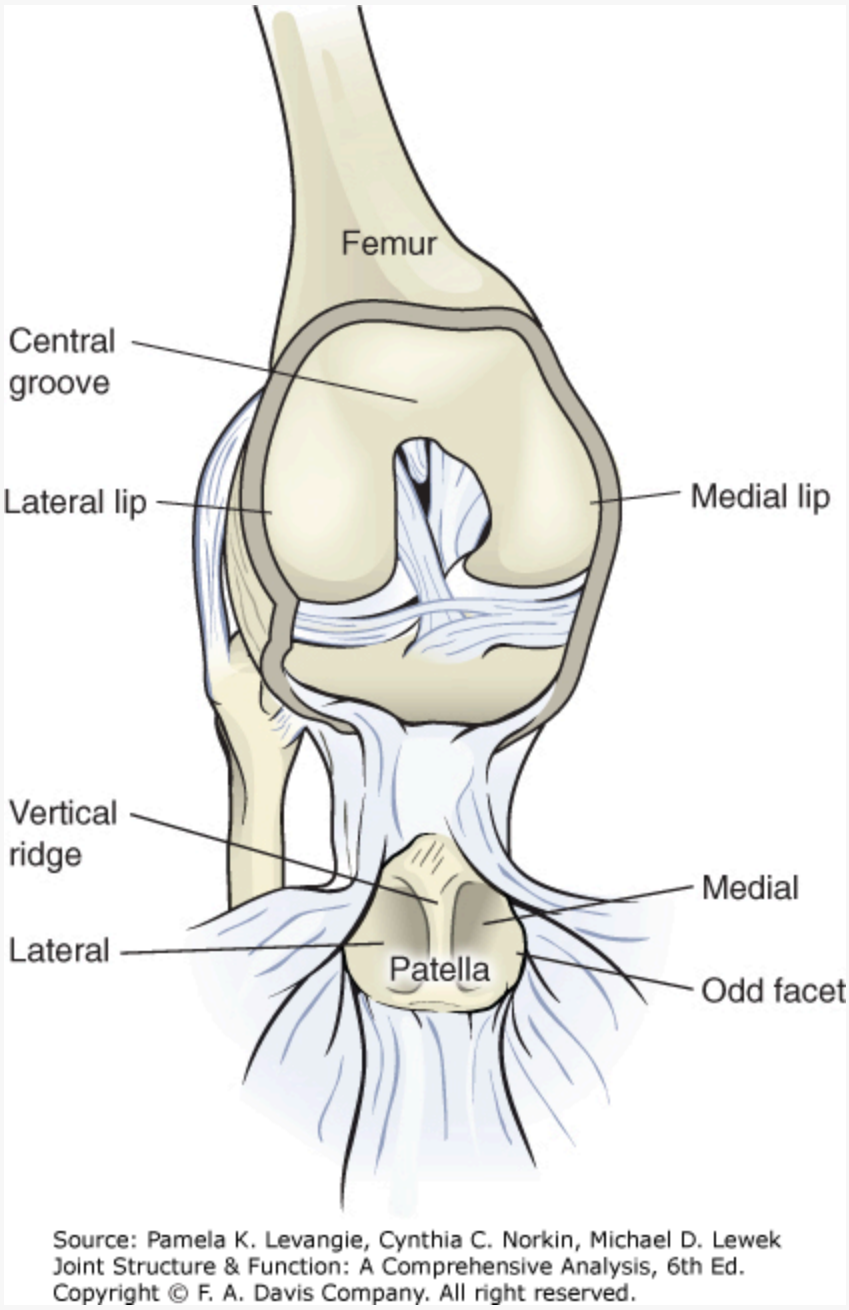
Patellofemoral Joint

The patella is a flat, triangularly shaped bone embedded within the quadriceps tendon, making it the largest sesamoid bone in the body (Fig. 11–37).[203] The patella is an inverted triangle with its apex directed inferiorly. The posterior surface is divided by a vertical ridge and covered by articular cartilage. This vertical ridge is situated in the center of the patella and divides the articular surface into approximately equally sized medial and lateral facets. Both the medial and lateral facets are flat to slightly convex side to side and superior to inferior. Most patellae also have a second vertical ridge toward the medial border that separates the medial facet from an extreme medial edge, known as the **odd facet** of the patella.[203] In the extended knee, the posterior surface of the patella sits on the femoral sulcus (or patellar surface) of the anterior aspect of the distal femur. The femoral sulcus has a central trochlear (intercondylar) groove that corresponds to the median ridge on the posterior patella. The femur's trochlear groove divides the sulcus into medial and lateral facets for articulation with the medial and lateral facets of the patella, respectively. Importantly, the lateral facet of the femoral sulcus is slightly more convex and has a more highly developed lip on its lateral edge than the medial facet.[203] This larger relative size of the lateral facet is important for patellar mechanics and pathomechanics. The patella is attached inferiorly to the tibial tuberosity by the patellar tendon. The patellofemoral joint is one of the most

incongruent joints in the body because of the shape of its articular surfaces and the much smaller articular surface area of the patella compared with the femur.

FIGURE 11-37

Articulating surfaces on the posterior aspect of the patella and on the femoral sulcus. Note the well-developed lateral lip on the lateral aspect of the articulating femoral surface.



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The patella's primary function is as an anatomical pulley for the quadriceps muscle.[203] Interposing the patella between the quadriceps tendon and the femoral condyles also reduces friction, because the femoral condyles will contact the smooth hyaline cartilage–covered posterior surface of the patella rather than rubbing against the

quadriceps tendon. The ability of the patella to perform its functions without restricting knee motion depends on its mobility. The patella is dependent on static and dynamic structures for its stability because of the bony incongruence of the patellofemoral joint. The oddly shaped patella and the uneven surface on which it sits help determine the motions of the patella that accompany tibiofemoral motion and make the patella and patellofemoral surfaces susceptible to tremendous forces. The joint's complex surfaces can also lead to many potential problems while performing what appears to be a relatively simple function. A comprehension of the structures and forces that influence patellofemoral function leads readily to an understanding of the common clinical problems found at the patellofemoral joint as it attempts to meet its contradictory demands for both mobility and stability.

Patellofemoral Articular Surfaces and Joint Congruence

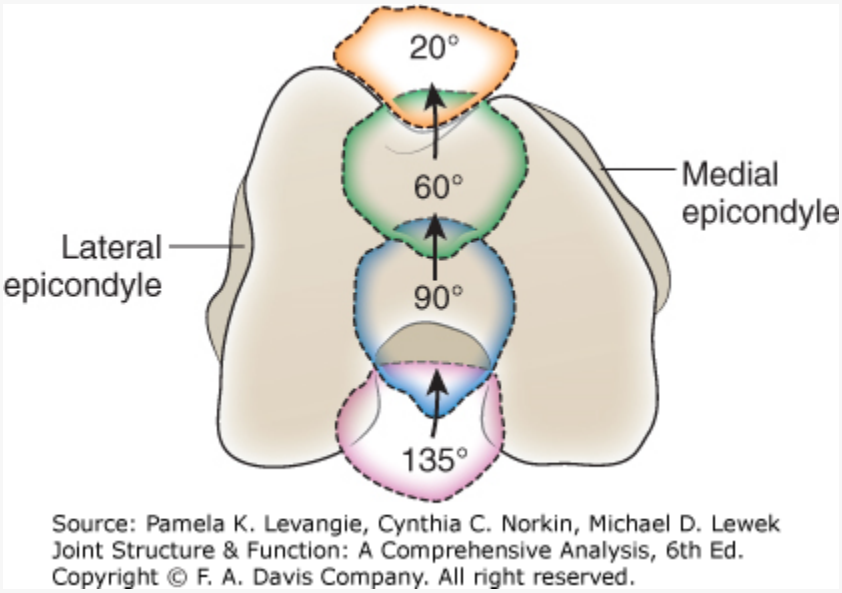
In the fully extended knee, the patella lies on the femoral sulcus.[203] Because the patella has not yet entered the intercondylar groove, bony support in this position is minimal, suggesting a greater potential for patellar instability compared with more flexed knee positions. In such extended knee positions the medial and lateral retinaculum and joint capsule provide passive stability to the patella.[203] The vertical position of the patella on the femoral sulcus is related to the length of the patellar tendon. Ordinarily, the ratio of the length of the patellar tendon to the diagonal length of the patella is referred to as the **Insall-Salvati index** and typically approximates 1:1.[204] A markedly short tendon produces an abnormally low position of the patella on the femoral sulcus known as **patella baja.** In contrast, a high-riding patella is termed **patella alta,** which increases the risk for patellar instability.[203] In this condition, the lateral lip of the femoral sulcus is not necessarily underdeveloped (although it may be), but the relatively more superior

position of the patella places the patella proximal to the high lateral femoral facet, rendering the patella less stable and easier to sublux. In patients with patella alta, the tibiofemoral joint must be more flexed before the patella translates inferiorly and engages the intercondylar groove, leaving a larger knee ROM within which the patella is relatively unstable.

The contact between the patella and the femur changes throughout the knee's ROM because of incongruence of the patellofemoral joint (Fig. 11–38). When the patella sits in the femoral sulcus in the extended knee, only the inferior pole of the patella is making contact with the femur.[203,205,206] As the knee (i.e., tibiofemoral joint) begins to flex, the patella slides down the femur, increasing the surface contact area. The first consistent contact between the patella and the femur occurs along the inferior margin of both the medial and lateral facets of the patella at 15° to 20° of knee flexion. As tibiofemoral flexion progresses, the contact area increases and shifts from the initial inferior location on the patella to a more superior position (Fig. 11–39).[206] As the contact area shifts superiorly along the posterior aspect of the patella, it also spreads transversely to cover the medial and lateral facet. By 90° of knee flexion, all portions of the patella have experienced some (although inconsistent) contact, with the exception of the odd facet. As flexion continues beyond 90°, the area of contact begins to migrate inferiorly once again as the smaller odd facet makes contact with the medial femoral condyle for the first time.[206] At full flexion, the patella is lodged in the intercondylar groove, and contact is on the lateral and odd facets and no longer on the medial facet.[203]

FIGURE 11-38

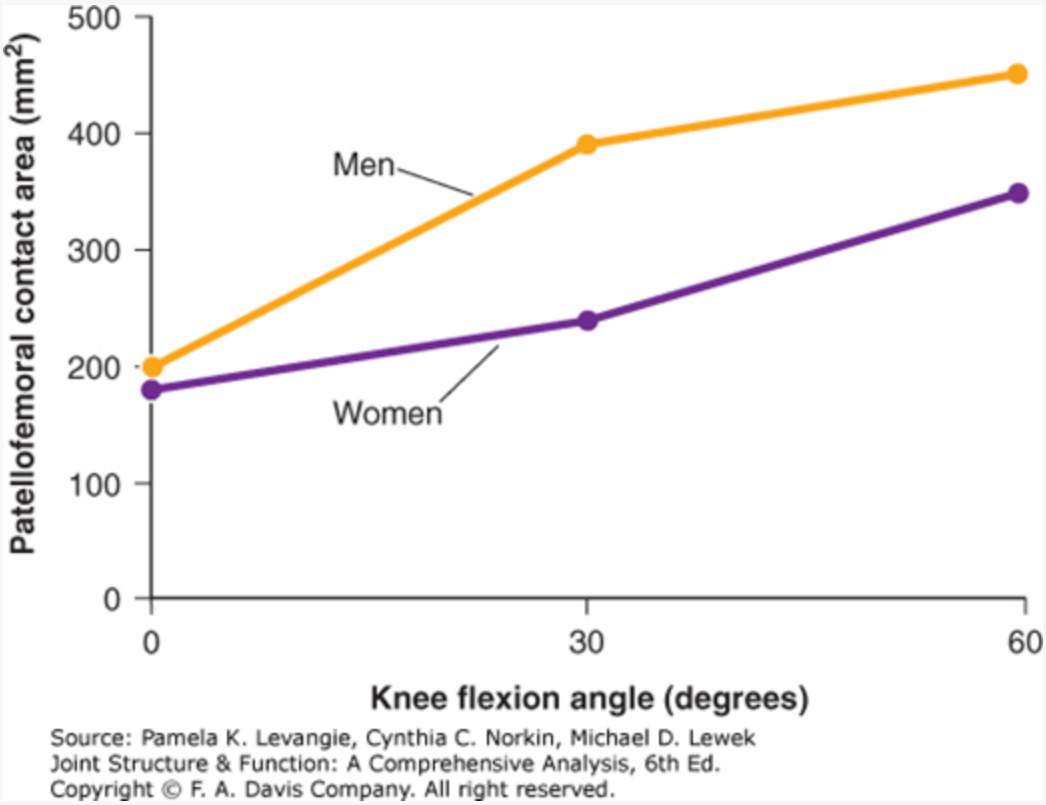
Near full extension, only the inferior pole of the patella makes contact with the femur. As flexion continues, the contact area moves superiorly and then laterally along the patella. By full flexion, only the lateral and odd facets are making contact with the femur.



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FIGURE 11-39

Contact between the articulating surfaces of the patella and femur is at a minimum in full knee extension. Patellofemoral contact area increases as the knee progresses from knee extension into knee flexion. (*Data from Besier TF, Draper CE, Gold GE, et al: Patellefemoral joint contact area increases with knee flexion and weightbearing. J Orthop Res 23:345, 2005.)*



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Motions of the Patella

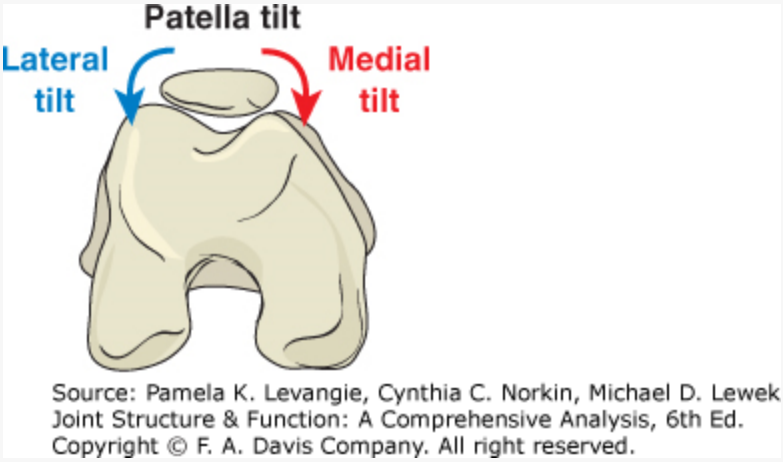
The relative movement between femoral, tibial, and patellar surfaces during knee motion is more intricate than what a simple hinge joint represents. As the contact between the patella and the femur changes with knee joint motion, the patella simultaneously translates and rotates on the femoral condyles. These movements are influenced by and reflect the patella's relationship to both the femur and the tibia. When the femur is fixed and the knee is flexing, the patella (fixed to the tibial tuberosity via the patellar tendon) is pulled down by the tibia, gliding inferiorly and rotating on the femoral condyles with the distal apex of the patella moving posteriorly. This sagittal plane rotation of the patella as the patella travels (or "tracks") down the intercondylar groove of the femur is termed **patellar flexion.** Knee extension brings the patella back to

its original position in the femoral sulcus, with the apex of the patella pointing inferiorly at the end of the normal ROM. This patellar motion of gliding superiorly while rotating up and around the femoral condyles is referred to as **patellar extension.**

In addition to patellar flexion and extension, the patella tilts around a longitudinal axis (proximal to distal through the patella), shifts medially and laterally in the frontal plane, and spins or rotates around an anteroposterior axis (perpendicular to the patella). Tilting about the longitudinal axis is termed **lateral/medial patellar tilt** and is named for the direction in which the anterior surface of the patella is moving relative to the femoral condyles (Fig. 11–40). Lateral patella tilt occurs when the lateral edge of the patella approximates the surface of the lateral femoral condyle, and medial patella tilt occurs when the medial edge of the patella moves toward the medial femoral condyle.[20] The direction and amount of patellar tilt is influenced by surrounding knee structures, such as the asymmetrical femoral condyles, the degree of medial or lateral tibial rotation, and tightness in soft tissues such as the lateral retinaculum.[208–210]

FIGURE 11-40

A superior view of the patellofemoral articulation. Medial and lateral tilt is named for the direction toward which the anterior surface of the patella is tipping, with the medial surface approximating the medial femoral condyle during medial tilt and vice versa during lateral tilt.

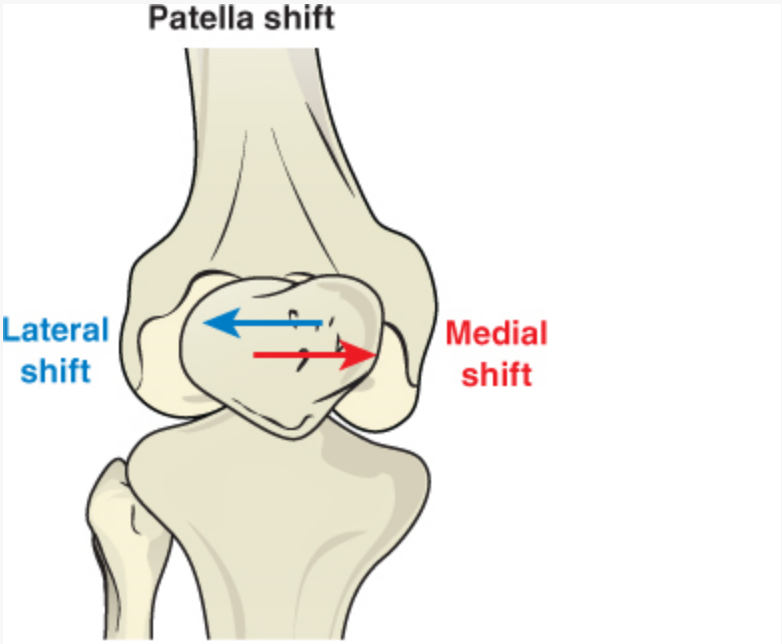


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Lateral and **medial patellar shifts** are translations (gliding) that occur in a plane of motion, rather than rotation around an axis (Fig. 11–41). A lateral patellar shift is defined as the patella moving toward the lateral femoral condyle in the frontal plane.[207] A lateral patellar shift is commonly used during the patella apprehension test to evaluate the stability of the patella within the femoral groove.[211]

FIGURE 11-41

Medial and lateral shift is named on the basis of the direction in which the patella is moving toward the medial and lateral femoral condyles.



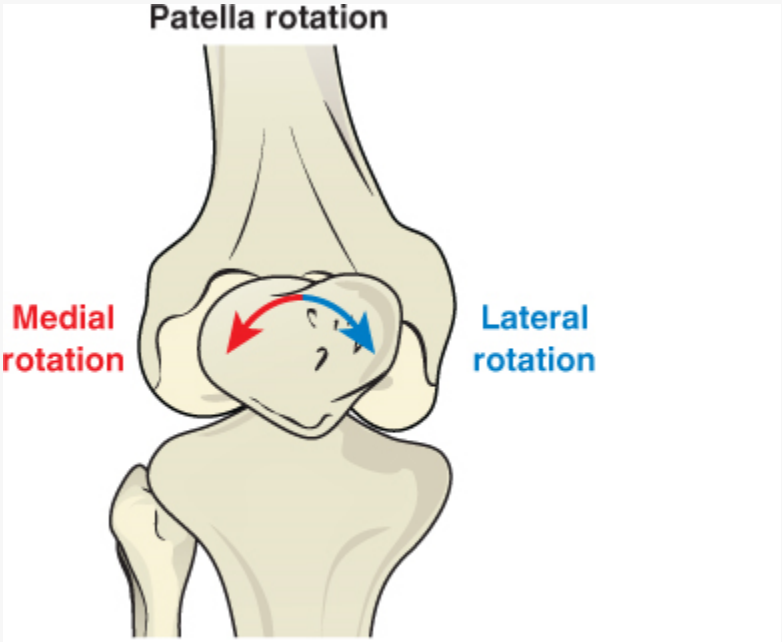
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Rotation of the patella about an anteroposterior axis (termed **medial/lateral rotation** of the patella) is referenced by the movement of the distal apex of the patella (Fig. 11–42). Medial rotation occurs when the patella spins around the anteroposterior axis with the apex (bottom) of the patella pointing toward the medial femoral condyle, and the base (top) of the patella moving closer to the lateral femoral condyle.[207] Patellar rotation, like patellar tilt, is necessary for the patella to remain seated between the femoral condyles as the femur undergoes axial rotation on the tibia. Because the inferior aspect of the patella is “tied” to the tibia via the patellar tendon, the inferior patella continually points toward the tibial tuberosity while moving with the femur.[212] Therefore, when the knee is in some flexion and there is medial rotation of the tibia on the fixed femur, the inferior pole of the patella will point medially as the patella medially rotates. Similarly, as the tibia rotates laterally, the patella will rotate laterally to keep the apex aligned

with the tibial tuberosity. During knee flexion, the patella medially rotates as the tibia medially rotates to “unlock” the knee. The patella then laterally rotates approximately 5° as the knee flexes from 20° to 90° because of the asymmetrical configuration of the femoral condyles.[209]

FIGURE 11-42

Medial rotation of the patella is shown in red. The inferior pole of the patella follows the tibial tuberosity during medial rotation of the tibia. Lateral rotation of the patella is shown in blue. The inferior pole of the patella follows the tibial tuberosity during lateral rotation of the tibia



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The patella, although firmly attached to soft tissue stabilizers (e.g., the extensor retinaculum), undergoes translational motions that are dependent on the degree of tibiofemoral ROM. The patella translates superiorly and inferiorly with knee extension

and flexion, respectively. Understanding these motions is important because they can influence knee joint function. During active knee extension, for example, if superior gliding of the patella is restricted, quadriceps function can be compromised, and knee extension may be limited. During tibiofemoral flexion, the patella glides inferiorly. A restricted inferior glide could therefore limit knee flexion. Simultaneous medial-lateral shifting of the patella accompanies superior-inferior patellar gliding.[209] The patella is typically situated slightly laterally in the femoral sulcus with the knee in full extension. As knee flexion is initiated, the patella is pushed medially by the large lateral femoral condyle. As knee flexion proceeds past 30°, the patella may shift slightly laterally or remain fairly stable as the patella is firmly engaged within the femoral condyles. Consequently, the patella shifts first medially and then laterally as the knee moves from full extension into flexion. Failure of the patella to glide, tilt, rotate, or shift appropriately can lead to restrictions in knee joint ROM, maltracking of the patellofemoral joint, or pain from compression of the patellofemoral articular surfaces. Therefore, passive and active mobility of the patella should be assessed clinically to determine the presence of hypermobility or hypomobility with respect to the femur.



Patient Application 11–3

Steven is a 68-year-old male patient with a 10-year history of progressing right knee pain. Radiographic imaging completed 3 years prior revealed osteoarthritis in the patellofemoral and medial tibiofemoral compartments. Steven's chief complaint is an impairment of flexing his knee during activities of daily living such as tying his shoe or squatting to retrieve objects from the floor. A portion of the physical examination during his initial evaluation included assessment of tibiofemoral ROM and patellofemoral joint mobility. What restrictions might be present that are contributing toward Steven's chief complaint?

Steven is likely to demonstrate an impairment in right knee flexion ROM. A deficit in

passive ROM similar in magnitude to the deficit in active ROM would indicate the likely deficits in muscle activation and strength are not contributing to the lack of ROM. Instead, restrictions in normal joint mobility may be present at either or both the tibiofemoral and patellofemoral joint mobility. An impairment in inferior patellar glide with accompanying impairments in either medial or lateral patellar gliding could be contributing to Steven's impairment in knee flexion ROM and activity limitations, with activities of daily living requiring the knee to flex. Targeted patellar mobilizations aimed at restoring joint mobility may result in improved knee flexion ROM and result in improved patient function.

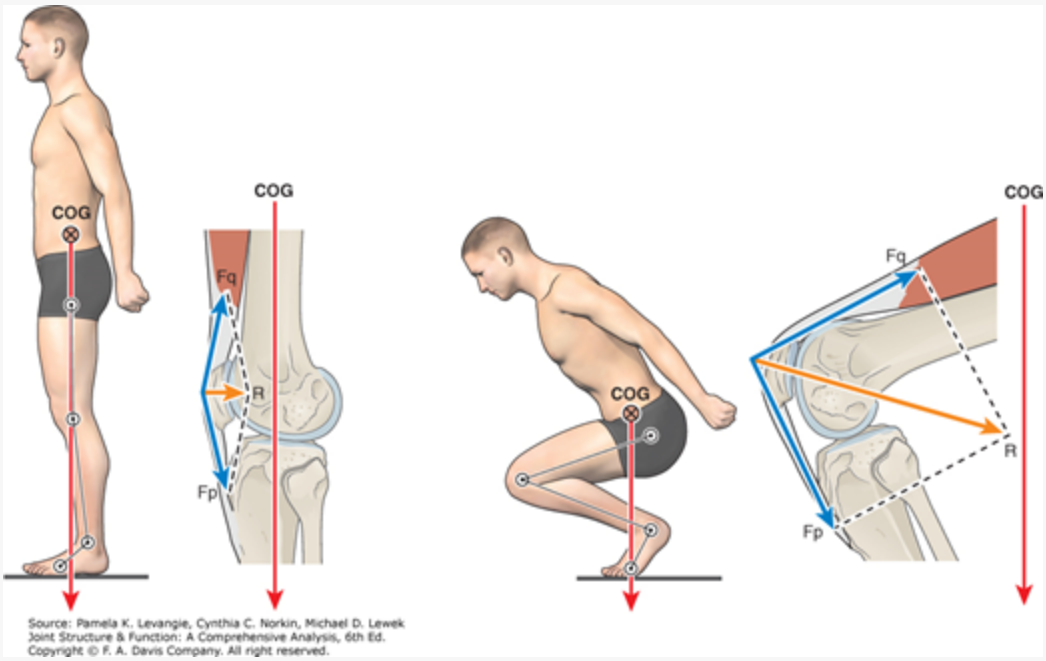
Patellofemoral Joint Stress

The patellofemoral joint experiences very high stresses during typical activities of daily living, making it a common location for degenerative changes and osteoarthritis to occur.[213–215] Joint stress (force per unit area) can be influenced by altering either joint forces or joint surface contact areas. The patellofemoral joint reaction (contact) force is influenced by both the magnitude of the quadriceps force and the knee angle. As the quadriceps contracts, the patella is pulled superiorly by the quadriceps tendon, with the pull resisted inferiorly by the patella tendon. The combination of these pulls produces a posterior compression force of the patella on the femur that varies with the amount of knee flexion.[203] At full extension, the quadriceps posterior compressive force on the patella is minimized and due exclusively to the origin of the vastus medialis and vastus lateralis muscles on the posterior femur.[190] Despite the small contact area between the patella and the femur in full extension, this minimal posterior compressive force allows for low patellofemoral joint stress with the knee at full extension. These low joint stress levels provide rationale for the use of straight-leg-raising exercises as a way of improving quadriceps muscle strength without creating or exacerbating patellofemoral pain.

As knee flexion progresses from full extension, the angle of pull between the quadriceps tendon and the patellar tendon decreases, which increases the joint reaction force and in turn produces greater patellofemoral joint compressive forces (Fig. 11–43).[203] This increased compression occurs whether the quadriceps muscle is active or passive during knee flexion. If the quadriceps muscle is inactive, then elastic tension alone increases within the quadriceps tendon and patellar tendon as knee joint flexion increases. If the quadriceps muscle is active, then both the active tension and passive elastic tension will contribute to increasing the joint compressive force. The total joint reaction force is therefore influenced by the magnitude of active and passive pull of the quadriceps and also by the angle of knee flexion.

FIGURE 11-43

Patellofemoral joint reaction forces are partially explained by the knee flexion angle. As the knee is flexed further, the *patellofemoral* compressive load is increased. COG = center of gravity.



Patellofemoral joint reaction forces can become very high during routine daily activities. During the stance phase of walking, when peak knee flexion is only approximately 20°, the patellofemoral compressive force is approximately 25% to 50% of body weight.[144] With greater knee flexion and greater quadriceps activity, as during running, patellofemoral compressive forces have been estimated to reach over 10 times body weight.[216] Deep knee flexion exercises that require large magnitudes of quadriceps activity can also substantially increase these compressive forces.[216] The large compressive forces created by the quadriceps are distributed over relatively small patellofemoral contact areas and can therefore lead to large joint stresses that the patellofemoral joint must endure. At the normal patellofemoral joint, the medial facet bears the larger stress levels due to smaller joint contact areas compared with the lateral facet.[203] Several mechanisms help minimize or dissipate the patellofemoral joint stress on the patella in general and on the medial facet in particular. In full extension,

minimal compressive forces are present on the patella; therefore, no compensatory mechanisms are necessary. As knee joint flexion proceeds, the area of patellar contact gradually increases, dispersing the increased compressive force.[190,203] This simultaneous increase in contact area and compressive force helps to minimize patellofemoral joint stress until approximately 90° of flexion. Specifically, from 30° to 70° of flexion, the magnitude of contact force is higher at the thick cartilage of the medial facet near the central ridge. This articular cartilage is among the thickest hyaline cartilage in the human body, enabling it to withstand the substantial compressive forces transmitted across the medial facet of the patella. Within this same ROM, the patella has its greatest effect as a pulley, as the moment arm of the quadriceps is maximized. The larger moment arm requires less quadriceps muscle force to produce the same torque, thereby minimizing patellofemoral joint compression. As flexion proceeds, the moment arm diminishes, which necessitates an increase in force production by the quadriceps. As knee flexion continues beyond 90°, patellofemoral stress will increase as the patellar contact area once again diminishes. However, some of this increasing compressive force on the patella is dissipated by the quadriceps tendon now contacting the femoral condyles.[217]

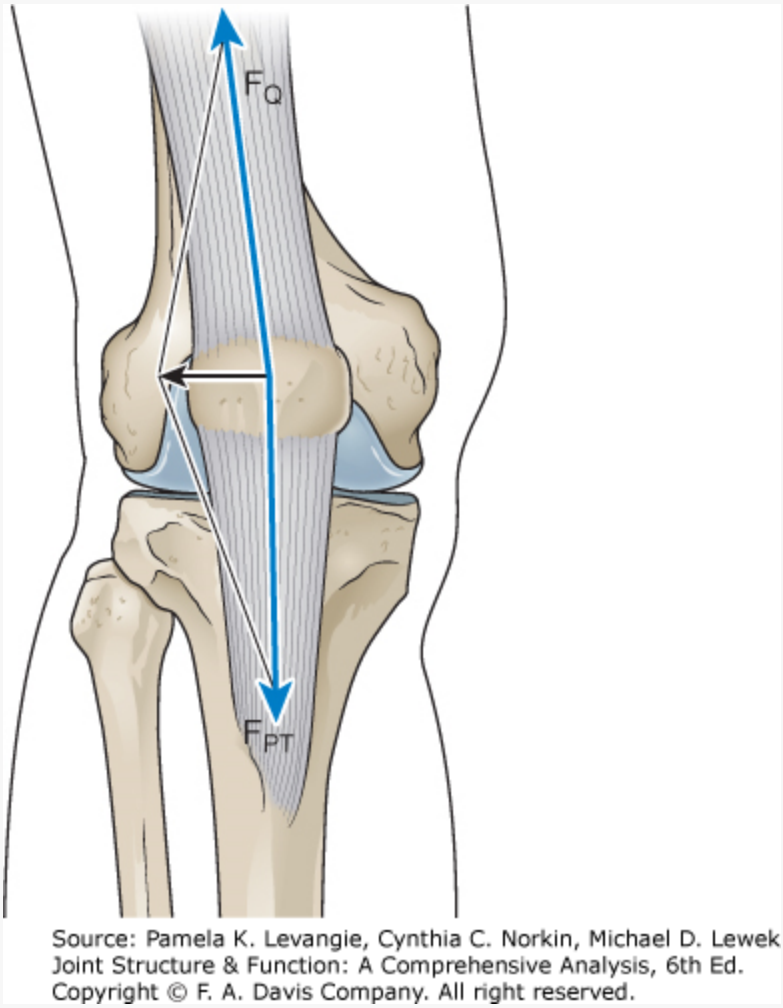
The vertical position of the patella can also significantly influence patellofemoral joint stress. Singerman and colleagues demonstrated that in the presence of patella alta, the onset of contact between the quadriceps tendon and femoral condyles is delayed.[217] As flexion increases, patellofemoral compressive forces will therefore continue to rise. In contrast, with patella baja, the contact between the quadriceps tendon and the femoral condyles occurs earlier in the range, resulting in a concomitant reduction in the magnitude of the patellofemoral contact force.[217–219]

Frontal Plane Patellofemoral Joint Stability

The patellofemoral joint may experience frontal plane instability near full knee extension. In the extended knee, instability can arise because the patella sits on the shallow aspect of the superior femoral sulcus where bony stability and patellofemoral compression from the quadriceps is limited. In addition, the action lines of the quadriceps and the patellar tendon do not coincide because of the physiological valgus that normally exists between the tibia and femur, resulting in a slight lateral pull of the patella (Fig. 11–44). Soft tissue stabilizers must assume more responsibility for medial-lateral stability in extension due to the resultant lateral pull of the patella where bony stability is reduced. Once knee flexion is initiated and the patella begins to slide down into the femoral sulcus (at about 20° of flexion), medial-lateral stability is substantially increased by the presence of bony stability. However, the concomitant increase in compression of the patella against the femoral condyles can lead to joint pain and degeneration. Whether the patella is at risk for instability or for increased compression, the position, mobility, and control of the patella in the frontal plane are of utmost concern, and are influenced by the relative tension in both the longitudinal and transverse stabilizers of the patella.

FIGURE 11-44

Because of the obliquity (physiological valgus angle) between the long axis of the femur and the tibia, the pull of the quadriceps (F_Q) and the pull of the patellar tendon (F_{PT}) lie at a slight angle to each other, producing a slight lateral force on the patella.



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The longitudinal stabilizers of the patella consist of the quadriceps tendon superiorly and the patellar tendon inferiorly. The patellotibial ligaments, which are part of the extensor retinaculum and reinforce the joint capsule, are also considered longitudinal stabilizers (see Fig. 11–12).[74] The longitudinal stabilizers provide passive increases in patellofemoral compression (see Fig. 11–43), which in turn helps to stabilize the patella in the medial-lateral direction. In the extended knee, where patellofemoral compression is minimal, the patella is in a relatively unstable position. When extension is exaggerated, as in genu recurvatum, the pull of the quadriceps muscle and patellar

tendon may actually distract the patella away from the femoral sulcus, further increasing the potential for frontal plane patellar instability.

The transverse stabilizers of the patellofemoral joint are composed of the superficial portion of the extensor retinaculum. The retinaculum provides active stabilization through direct connections of the vastus medialis and vastus lateralis muscles to the patella. The thickest portion of the medial retinaculum is the medial patellofemoral ligament (described earlier), which provides approximately 60% of the passive restraining force against lateral translation (lateral shift) of the patella.[70] Excessive medial translation is limited by the lateral patellofemoral ligament, which attaches the patella to the iliotibial band laterally.[74] Another important passive stabilizer is the anteriorly protruding lateral lip of the femoral sulcus, which acts as a buttress to excessive lateral patellar shift (see Fig. 11–37). Even large lateral forces will not sublux or dislocate the patella, provided that the lateral lip of the femoral sulcus is of sufficient height. A flattened shape of this femoral notch, called trochlear dysplasia, can lead to patellar subluxation or complete dislocation from relatively small lateral forces imposed on the patella.[203] Both the longitudinal and transverse structures can therefore influence the medial-lateral positioning of the patella to remain within the femoral sulcus, as well as influence patellar tracking as the patella slides down the femoral condyles and into the intercondylar groove.

Patellar hypermobility may result in patellar subluxations or dislocations, whereas hypomobility may yield pain due to greater patellofemoral stresses. Passive mobility of the patella is maximized when the knee is fully extended and the quadriceps are relaxed. A frontal plane imbalance in the passive tension (e.g., lax medial patellofemoral

ligament) or a change in the line of pull of the dynamic structures can influence and change the orientation of the patella, often most notable when the knee joint is in extension and the patella sits on the relatively shallow superior femoral sulcus. Abnormal forces, however, may influence the excursion of the patella even in its more secure location within the intercondylar groove with the knee in flexion.

As previously noted, tension in the active or stretched quadriceps muscle creates compression between the patella and the femur, which increases patellofemoral stability. The net force on the patella is determined by the resultant pull of the four muscles that constitute the quadriceps femoris and by the pull of the patellar tendon. Each of the segments of the quadriceps contributes to frontal plane mobility and stability because of their varying orientations along the femur. As noted earlier, the pull of the vastus lateralis muscle is normally 35° lateral to the long axis of the femur, whereas the pull of the proximal portion of the vastus medialis muscle (vastus medialis longus) is approximately 15° to 18° medial to the femoral shaft, with the distal fibers (vastus medialis oblique) oriented 50° to 55° medially (Fig. 11–31A).[190,191]

Because the vastus medialis and vastus lateralis muscles not only pull on the quadriceps tendon but also exert a pull on the patella through their retinacular connections, complementary function is critical. Weakness of the quadriceps muscle may substantially increase the resultant lateral forces on the patella.[194] Anatomical variations in orientation of the quadriceps may also contribute to asymmetrical pulls on the patella. In general, the vastus medialis oblique inserts into the superomedial aspect of the patella about one-third to one-half of the distance on the medial border. In instances of patellar malalignment, the vastus medialis oblique insertion site may be

located less than a fourth of the distance on the patella's medial aspect, and as a result, the vastus medialis muscle cannot effectively counteract the lateral motion of the patella.[220]

Patient Application 11–4

Lauren is a 17-year-old adolescent with a history of right patellar instability who underwent distal realignment surgery. The goal of distal realignment surgery is to improve the stability of the patella within the femoral sulcus. This is accomplished by moving the tibial tubercle to shift the alignment of the patellar tendon. The disruption of her extensor tendon during surgery could influence her ability to use her quadriceps postoperatively and may contribute to further extensor weakness. With each quadriceps contraction, she is pulling on the patellar tendon, which, if sore, could cause her to reduce quadriceps activation to avoid pain. Poor quadriceps activation after surgery could lead to an even weaker, atrophied quadriceps muscle than what is already present due to the surgical procedure. If proper quadriceps activation with resulting superior glide of the patella is not achieved, the surgical outcome could be poor, with continued pain and dysfunction around her patella. Without proper quadriceps muscle function, dynamic control of the patella may still be inadequate, and episodes of patellar instability may still exist. Poor control of the patella can also lead to a hypomobile patella with limited medial-lateral or superior-inferior glides. Achieving an active quadriceps contraction with superior patellar glide will be an important goal for Lauren to achieve early after surgery to attain the desired outcome of the distal realignment procedure.

Expanded Concepts 11–12

Selective Strengthening of the Vastus Medialis Oblique

It was previously assumed that a weak vastus medialis oblique contributed to diminished medial glide of the patella, which resulted in attempts to devise exercise regimens to selectively strengthen the vastus medialis oblique. However, both portions of the vastus medialis muscle, like the other components of the quadriceps, are innervated by the femoral nerve, and preferential activation of the vastus medialis over other quadriceps muscles, such as the vastus lateralis, is not possible.[221] Further, atrophy of the entire quadriceps muscle, not just the vastus

medialis, is present in patients with patellofemoral pain.[194] In the absence of evidence supporting differential recruitment of the vastus medialis oblique, strengthening of the vastus medialis oblique portion of the vastus medialis muscle can be accomplished through whole quadriceps strengthening, using techniques such as biofeedback or neuromuscular electrical stimulation if activation deficits exist. In this way, it can be ensured that the quadriceps, and specifically the vastus medialis oblique, is being sufficiently overloaded to promote muscle strength and hypertrophy.

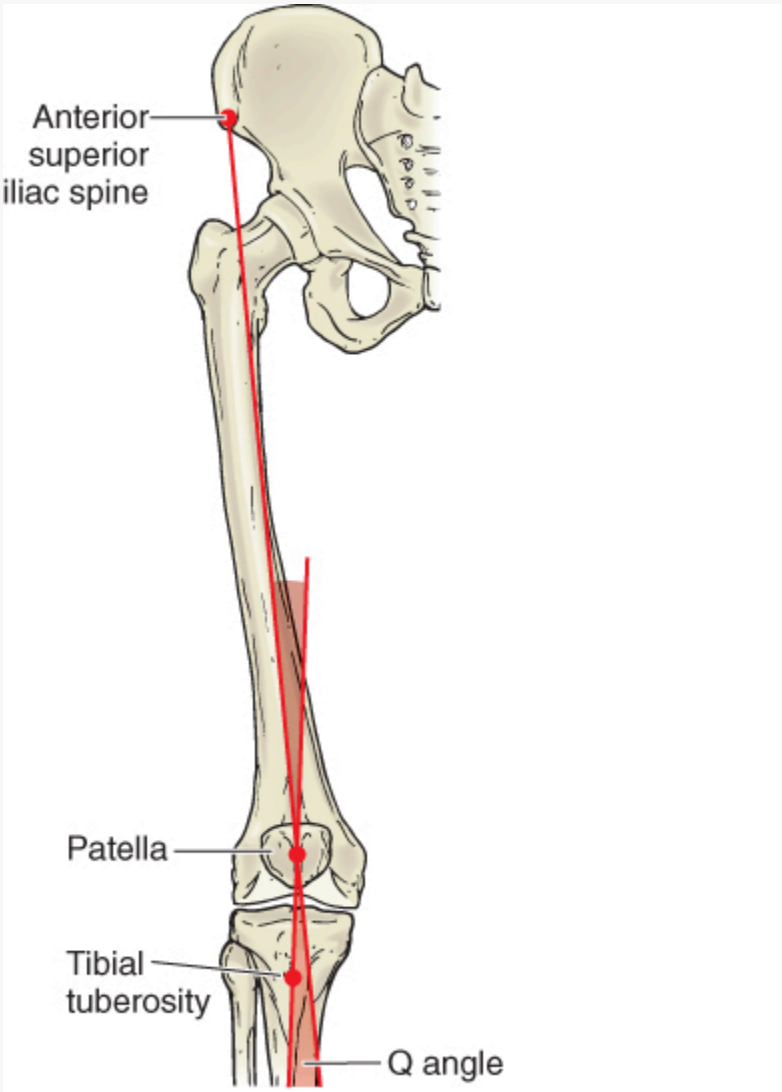
Asymmetry of Patellofemoral Stabilization

The orientation of the quadriceps' resultant pull with respect to the pull of the patellar tendon provides information about the net force on the patella in the frontal plane. The net effect of the pull of the quadriceps and the patellar tendon can be assessed clinically using a measurement called the **Q angle** (quadriceps angle). The Q angle is the angle formed between a line connecting the anterior superior iliac spine to the midpoint of the patella (representing the direction of pull of the quadriceps) and a line connecting the tibial tuberosity and the midpoint of the patella (Fig. 11–45). A Q angle of 10° to 15° measured with the knee either in full extension or slight flexion is considered normal.[222] Alterations in alignment that increase the Q angle may increase the lateral force on the patella. An increase in this lateral force may increase the compression between the lateral patellar facet and the lateral facet of the femoral sulcus. In the presence of a large enough lateral force, the patella may actually sublux or dislocate over the femoral sulcus when the quadriceps muscle is activated with an extended knee. The Q angle is usually measured with the knee at or near full extension because lateral forces on the patella are typically more problematic in this position. With the knee flexed, the posterior compressive force is greater and the patella is set more firmly within the deeper part of the femoral sulcus, such that even a very large

lateral force on the patella is unlikely to result in dislocation. Furthermore, the Q angle will diminish with knee flexion as the tibia rotates medially in relation to the femur.

FIGURE 11-45

The Q angle is the angle between a line connecting the anterior superior iliac spine to the midpoint of the patella and the extension of a line connecting the tibial tubercle and the midpoint of the patella.



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It has been postulated that women have a slightly greater Q angle than do men because of the presence of a wider pelvis, increased femoral anteversion, and a relative knee valgus angle. However, other authors have disputed this, and the presence of a gender difference in Q angle is still a matter of debate.[222,223] Although an excessively large Q angle of 20° or more is usually an indicator of some structural malalignment, an apparently normal Q angle will not necessarily ensure the absence of problems. Large Q angles are thought to create excessive lateral forces on the patella that may predispose the patella to pathological changes. One problem with using the Q angle as a measure of the lateral pull on the patella is that the line between the anterior superior iliac spine and the mid patella is only an estimate of the line of pull of the quadriceps and does not necessarily reflect the actual line of pull. If a substantial imbalance exists between the vastus medialis and vastus lateralis muscles in a patient, the Q angle may lead to an incorrect estimate of the lateral force on the patella because the actual pull of the quadriceps muscle is no longer along the estimated line. Unfortunately, we cannot isolate the muscle forces from the vastus medialis and vastus lateralis. Furthermore, a patella that sits in an abnormal lateral position in the femoral sulcus, because of imbalanced forces, will yield a smaller Q angle because the patella lies more in line with the anterior superior iliac spine and tibial tuberosity.

There are several abnormalities that can yield increased lateral forces on the patella. An imbalance between the vastus lateralis and vastus medialis muscles could alter frontal plane stability of the patella. Some in vivo neurophysiological investigations of patients with patellofemoral pain have demonstrated delayed timing and magnitude of muscle activation in the vastus medialis compared with the vastus lateralis.[224] However, muscle activation does not provide direct information regarding muscular forces acting on the

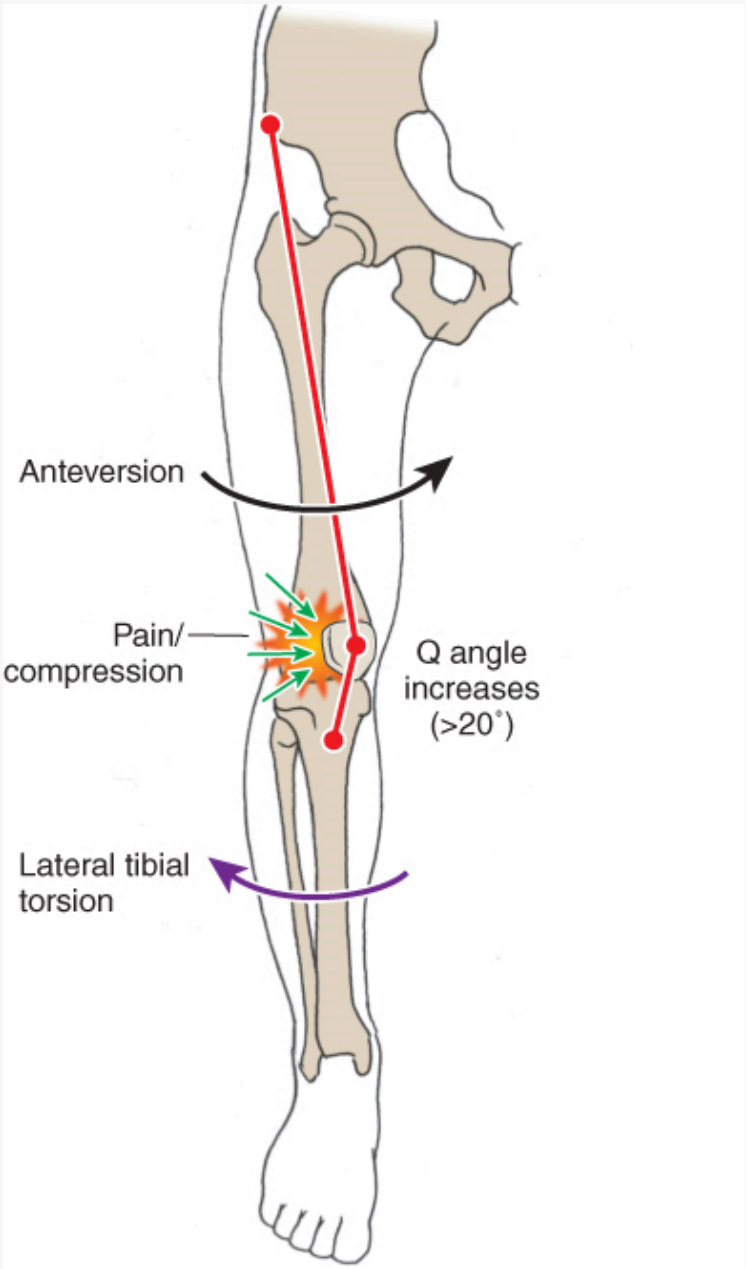
patella. Muscles with equal activation levels can produce different levels of muscle force because of different cross-sectional areas of the respective muscle. Further, delayed muscle activation can be influenced by the positioning of electrodes used to measure muscle activation on each muscle. Therefore, an imbalance in muscle activation is not evidence for an imbalance in muscle forces. Despite these limitations, muscle activation is often studied because noninvasive experimental techniques to directly measure individual muscle forces do not exist. It is not known whether a difference in force exists between the vastus medialis and lateralis in patients with patellofemoral pain.[224] Further, as noted earlier, patients with patellofemoral pain do not demonstrate selective atrophy of the vastus medialis muscle in comparison to the other quadriceps muscles.[194] At this time, the interactions between decreased vastus medialis activity, knee pain, and patellar malalignment and its effect on patellofemoral dysfunction remain controversial.

The presence of a tight iliotibial band could limit the patella's ability to perform its normal medial shift during flexion, which would contribute to increased stress under the lateral facet of the patella.[72] With knee flexion, the iliotibial band exerts a lateral pull on the patella. A loaded iliotibial band can cause lateral shifting and tilting of the patella in mid ranges of knee flexion.[72] The increased lateral tilt could focus the patellofemoral load on the lateral facet, increasing joint stress. Genu valgum can further increase the obliquity of the femur (see Fig. 11–5B) and, concomitantly, the lateral pull of the quadriceps. In contrast, individuals with genu varum exhibit less obliquity of the femur (Fig. 11–5C), and therefore should have a diminished lateral pull of the quadriceps. The transverse plane deviation of medial femoral torsion (or femoral anteversion) generally results in the femoral condyles being turned in (medially rotated). The medially

oriented femoral sulcus carries the patella medially and increases the Q angle. Likewise, lateral tibial torsion increases the Q angle due to the increased obliquity of the patellar tendon. When medial femoral torsion and lateral tibial torsion coexist, the Q angle will increase substantially, resulting in a considerable lateral force on the patella (**Fig. 11–46**). As we will see in Chapter 12, the presence of excessive or prolonged pronation in the foot can contribute to excessive or prolonged medial rotation of the lower extremity that moves the patella medially, increasing the Q angle and promoting a greater lateral force on the patella in a way similar to that of medial femoral torsion. Each of these conditions can predispose the patella to excessive stress laterally or to lateral subluxation or dislocation.

FIGURE 11-46

Increased medial femoral torsion (femoral anteversion) and tibial lateral torsion will result in a larger Q-angle and an increased lateral force on the patella.



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Factors other than the alignment and balance of the quadriceps muscle components can also influence patellar positioning. Laxity of the medial retinaculum or adaptive shortening of the lateral retinaculum may contribute to a laterally tilted patella in the femoral sulcus. It is currently unknown whether such changes in the passive structures

are primary or secondary to abnormalities in the dynamic stabilizers.

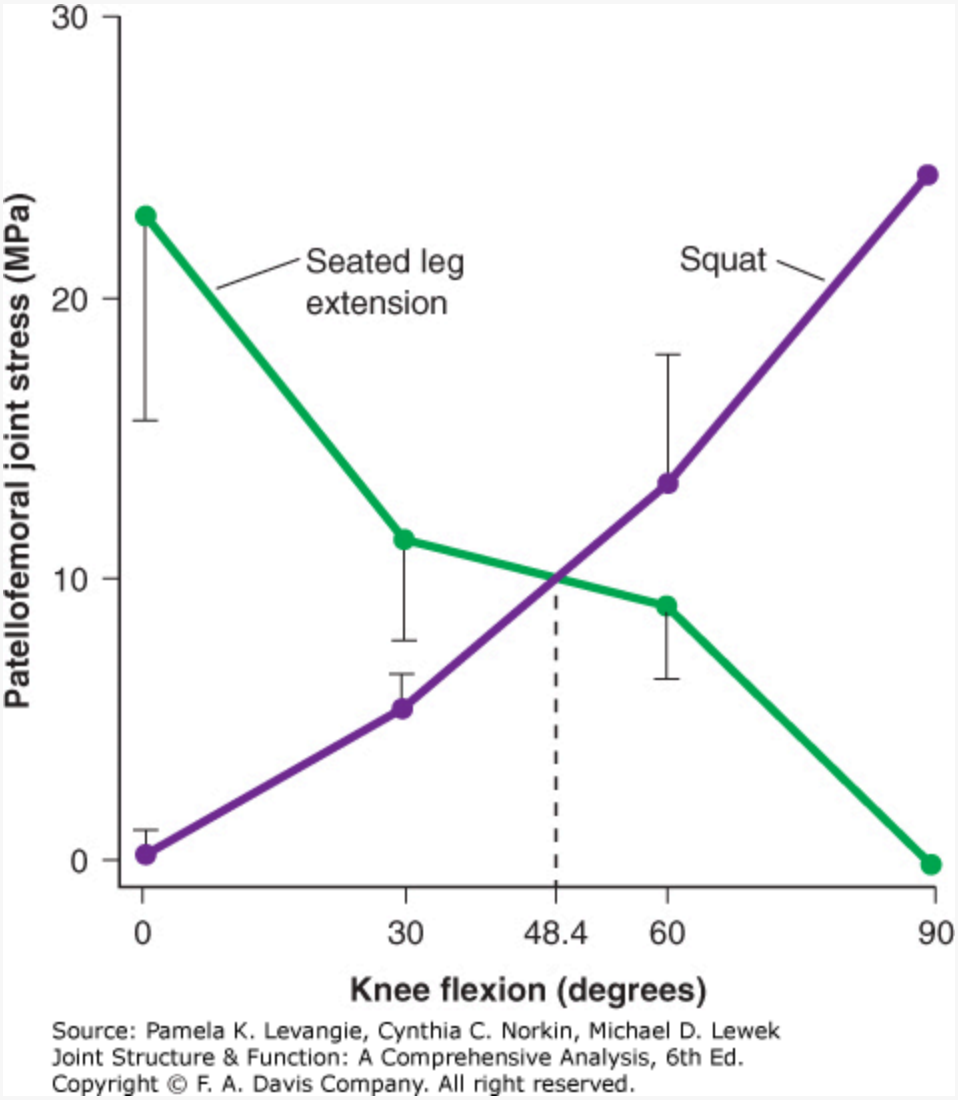
Weightbearing Versus Non-Weightbearing Exercises With Patellofemoral Pain

Both weightbearing and non-weightbearing exercises are prescribed and effective in reducing pain for patients with patellofemoral pain.[225] Each mode of exercise influences the patellofemoral joint differently depending on the knee's position within the ROM. Quadriceps strengthening needs to be completed within pain-free knee ROMs.[225] We noted earlier, non-weightbearing extension exercises, such as the seated knee extension, require increased quadriceps torque as extension progresses (quadriceps force increases with decreasing knee flexion angle; see Fig. 11–35). Increased torque by the quadriceps near full knee extension is necessary to compensate for the increased moment arm of the resistance. However, the greater compressive force generated by the increased quadriceps contraction can be detrimental for an individual with patellofemoral pain, especially if the irritated tissue is located on the inferior aspect of the patella that is in contact with the femur near extension. In contrast, a weightbearing exercise typically requires greater quadriceps activity with greater knee flexion (e.g., at the bottom of a squat) as the resistance moment arm increases (see Fig. 11–36). During weightbearing exercises, greater knee flexion will therefore increase the compressive force across the patellofemoral joint both because of increased force demands on the quadriceps muscle and because of the increased patellofemoral compression that occurs in positions of greater knee flexion. Substantial patellofemoral compression may aggravate patellofemoral pain. Exercise recommendations for patients with patellofemoral pain can be based on changing patellofemoral joint stress with weightbearing and non-weightbearing exercises and knee flexion angle (**Fig. 11–47**). It has been recommended that those with patellofemoral pain avoid deep flexion while

doing weightbearing extension exercises and avoid the final 30° of extension during non-weightbearing knee extension exercises.[214,226]

FIGURE 11-47

Patellofemoral joint stress is greater during loaded non-weightbearing exercises than weightbearing exercises when the knee was closer to knee extension. Patellofemoral joint stress was higher, however, during weightbearing exercises when knee flexion exceeds approximately 50°. *(Data from Powers CM, Ho KY, Chen YJ, et al: Patellofemoral joint stress during weightbearing and non-weightbearing quadriceps exercises. J Orthop Sports Phys Ther 44: 320, 2014.)*



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Expanded Concepts 11–13

Patellofemoral Pain: Weightbearing Versus Non-Weightbearing Exercises

The use of weightbearing exercises has occasionally been promoted as safer and "more functional" than non-weightbearing exercises.[226] There are, however, numerous activities performed throughout the day in a non-weightbearing position. Although other muscles are forced to work concomitantly during weightbearing situations to control other joints, this is not always the best strategy for strengthening. The use of non-weightbearing exercise isolates the target muscle, which may make strengthening more effective.[227,228] With regard to safety (e.g.,

patellofemoral joint stress), an understanding of how joint stress and tissue strain change throughout the knee ROM must be considered. For example, non-weightbearing quadriceps exercises produce larger patellofemoral joint stress closer to full extension.[214] In contrast, during weightbearing extension exercises, patellofemoral joint stress is minimal near full extension but increases with increasing flexion (Fig. 11–47). Rehabilitation programs using both weightbearing and non-weightbearing quadriceps strengthening exercises have resulted in substantial decreases in pain levels in patients with patellofemoral pain, and the use of both strategies is recommended to provide both quadriceps-specific strengthening and functionally-specific training.[225,229]

Life Span and Clinical Considerations

The joints of the knee complex, like other joints in the body, are subject to developmental defects, injury, and disease processes. A number of factors, however, make the knee joint unique in its development of various pathologies. The knee, unlike the shoulder, elbow, and wrist, must support the body weight and at the same time provide considerable mobility. The knee's anatomical complexity is necessary to dissipate the enormous forces applied through the joint as two of the longest levers in the body meet at the knee complex.

Tibiofemoral Joint Injury

The tremendous forces applied through the knee have the potential to contribute to numerous injuries and degenerative damage. In addition, participation in physical fitness and sports activities that involve jumping, pivoting, cutting, or repetitive cyclic loading among all age groups and both sexes can increase the risk of injury to the menisci, ligaments, bones, bursae, and musculotendinous structures.

Meniscal injuries are common and usually occur as a result of sudden rotation of the

femur on the fixed tibia when the knee is in flexion. Medial meniscus injuries are more prevalent than lateral meniscus injuries, in part because the pivot point during axial rotation in the flexed knee occurs through the medial meniscus, and also because the more rigidly attached medial meniscus makes it more susceptible during sudden loads. Ligamentous injuries may occur as a result of a force that causes the joint to exceed its normal ROM. Although excessive forces may cause ligamentous tears, lower-energy forces may similarly cause disruption in ligaments weakened by aging, disease, immobilization, steroids, or vascular insufficiency. Cyclic loading (whether short-term and intense or over a prolonged period) may also affect a ligament's viscoelasticity and stiffness. A weakened ligament may take 10 months or more to return to normal stiffness once the underlying problem has been resolved. After a ligament injury or reconstruction, the new or damaged tissue must be protected to minimize excessive stress through the healing tissue. Absence of tissue stress, however, is also detrimental, because the new tissue will not adapt and become stronger under unloaded conditions. Rehabilitation of a repaired or reconstructed ligament, therefore, is a balance in avoiding either too much or too little applied stress.

The bony and cartilaginous structures of the tibiofemoral joint may be injured either by the application of a large direct force, such as during a twist or fall, or by repetitive, lower loads exerted by abnormal ligamentous and muscular forces. Knee osteoarthritis is often seen in older adults and is particularly common in women. This progressive erosion of articular cartilage may be initiated by things such as a previous traumatic joint injury, obesity, malalignment, instability, or quadriceps muscle weakness. Tibial plateau fractures can occur when large axial loads are applied through the joint. Knee joint instability, as frequently seen in the knee after anterior cruciate ligament injury,

can lead to progressive changes in the articular cartilage, menisci, and other ligaments attempting to restrain the increased joint mobility. The presence of ligamentous instability induces abnormal forces through the joint, and excessive shearing at the joint surface can occur. The excessive laxity present after ligamentous injuries must be controlled to avoid episodes of giving way. Because the knee has poor bony congruency, the muscles must provide greater dynamic control of all fine and gross movements of the tibiofemoral joint in the absence of one or more ligaments. Increased muscular co-contraction, however, may generate greater compressive forces through the joint, contributing to articular cartilage degeneration. An improved method of providing dynamic stability to a lax joint, therefore, is to generate isolated muscle contractions as needed, rather than a massive co-contraction to stiffen the joint.

The numerous bursae and tendons at the knee are also subject to injury. The cause of injuries to these structures may be either a direct blow or prolonged compressive or tensile stresses. Inflammation of bursa, or bursitis, is common after either blunt trauma or repetitive low-level compressions, which can irritate the tissue. The prepatellar bursa, the superficial infrapatellar bursa (known as **housemaid's knee** when it is inflamed), and the bursa beneath the pes anserinus are common locations for injury. Acute inflammation of a tendon, or tendinitis, results from repetitive low-level stresses to the tendon tissues. It is frequently caused by overuse of the affected muscle and can occur in response to a previous ligamentous injury. Tendinosis is a chronic degeneration of a tendon without the presence of inflammation, and may result from unhealed tendonitis. Another potential source of pain and dysfunction in the knee joint is the irritation of a patellar plica. Classic symptoms include pain with prolonged sitting, stair climbing, and during resisted knee extension exercises. In flexion, the medial

patellar plica is drawn over the medial femoral condyle and can become compressed beneath the patella, causing inflammation. If the inflamed plica becomes fibrotic, it may create a secondary synovitis around the femoral condyle, and deterioration of the condylar cartilage may occur. A thickened or inflamed superior plica may erode the superior aspect of the medial facet of the patella.

Patellofemoral Joint Injury

Abnormal pathomechanics of the patellofemoral joint can predispose the knee to patellofemoral dysfunctions such as excessive pressure on the lateral facets of the patella, lateral subluxation, or lateral dislocation. Both patellar instability and increased patellofemoral compression are commonly associated with knee pain, poor tolerance of sustained passive knee flexion (as in sitting for long periods), “giving way” of the knee, and exacerbation of symptoms by repeated use of the quadriceps on a flexed knee. Consequently, diminished use of the quadriceps often results, leading to muscle atrophy and further deterioration of patellar control. As muscle function declines, patellofemoral dysfunction may progress, necessitating a series of controlled situations to generate hypertrophy of the quadriceps through knee ranges with minimized pain.

Increased lateral patellar compression can be caused by a tight iliotibial band, large Q angle (e.g., as in genu valgum or femoral anteversion with lateral tibial torsion), relative vastus medialis muscle weakness, or patellar hypomobility. Patellar hypermobility may be due to lax medial structures or a short lateral femoral condyle, both of which increase the risk of lateral patellar subluxation or dislocation. After a lateral patellar subluxation or dislocation, the medial retinaculum is stretched or torn as the patella deviates toward or slips over the lateral lip of the femoral sulcus or condyle. The return

of the laterally dislocated patella into the femoral sulcus may affect the medial patella (occasionally causing an osteochondral fracture). There are a host of other pathologies that can occur around the patellofemoral joint, including pain from the lateral patellofemoral ligament, inflammation of the medial patellar plica (discussed previously), and pain from the quadriceps tendon or the patellar tendon.

Cartilaginous changes seen on the lateral patellar facet were once considered to be diagnostic of patellofemoral dysfunction, and the term **chondromalacia patella** (softening of the cartilage) was assigned. With the knowledge that similar cartilaginous changes can be found in asymptomatic knees and that the medial patellar facet can show greater change without symptoms or progressive cartilage deterioration, more general diagnoses have been used, including **patellofemoral arthralgia** or **patellofemoral pain syndrome.** More general terminology suggests that the damage extends beyond the articular cartilage. Cartilage is aneural and therefore cannot be the cause of pain.[230] Instead, patients with patellofemoral pain can experience discomfort from damage to subchondral bone, the synovial membrane, and ligamentous or musculotendinous structures. Advances in diagnostic imaging techniques allow for patellar tracking during dynamic tasks. The effects that femoral and tibial rotation have on patella position have warranted interventions that control the hip, pelvis, and ankle motion when treating patients with patellofemoral joint injury and pain. A comprehensive multi-joint evaluation is therefore necessary to identify the pathomechanics underlying the myriad knee problems that might develop.

Summary

- A thorough knowledge of normal structure and function of the knee joint can be used to predict or understand the immediate impact of a specific injury and the secondary effects on intact structures.
- The variety of forces transmitted through the knee complex arises from gravity (weightbearing forces), muscles, ligaments, and other passive soft tissue structures. Any alteration of the knee's anatomy can substantially influence these forces and have a dramatic impact on the function of the knee joint.
- Damage to the tibiofemoral joint or the patellofemoral joint can result from either a large rapid load or the accumulation of smaller repetitive loads.
- An understanding of both the primary and secondary effects of injury is important to gain a full appreciation for the pathogenesis of knee disorders.

Study Questions

1. Describe the congruency of the tibiofemoral joint. What bony and soft tissue factors enhance or reduce its stability?
2. How does genu valgum alter the mechanical axis of the lower extremity? What effect can this have on the compressive loading of the articular cartilage in each tibiofemoral compartment?
3. Describe the menisci of the knee, including their function, shape, and attachments.

4. Which ligaments of the knee are considered capsular ligaments? Which ligaments of the knee are extracapsular?

5. What are the patella plicae, and what implications do they have in knee joint dysfunction?

6. Which knee joint ligaments contribute to anteroposterior stability of the knee joint?

7. Which ligaments contribute to medial-lateral stability of the knee joint?

8. Identify and describe which muscles are capable of altering strain on the anterior cruciate ligament.

9. Identify the major bursae of the knee joint. Which of these are continuous with the synovial capsule of the knee?

10. Describe the intra-articular movement of the femur and the tibia as the knee moves from full extension into flexion in both the weightbearing and non-weightbearing position.

11. Describe the movement and change in the shape of the menisci during knee motion. How do their attachments contribute to the movement?

12. Describe the automatic axial rotation mechanism of the knee, including the structures responsible for its occurrence.

13. At which point in the knee's ROM is axial rotation greatest? Which muscles

produce active medial rotation? Lateral rotation?

14. Which muscles that cross the knee joint are biarticulate? How does the position of the hip joint and ankle joint alter each of these muscle's function at the knee?

15. What function or functions does the patella serve at the knee joint?

16. How does the patella move in relation to the femur in normal motions? How is knee function affected if the patella is hypomobile?

17. Describe the contact of the patella with the femur at rest in full extension. How does the contact location and resultant joint contact stress change as flexion progresses?

18. Is the patella equally effective as an anatomical pulley at all points in the knee range of motion? At which point or points is it most effective? Least effective?

19. What is the Q angle of the knee joint? How does it and other muscular and ligamentous features of the knee affect the frontal plane stability of the patellofemoral joint?

20. Why is ascending stairs commonly cited as producing knee pain? Relate this to patellofemoral joint compression.

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Chapter 12: The Ankle and Foot Complex

RobRoy L. Martin

Anatomy Overview

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MUSCLE ACTIONS OF THE ANKLE AND FOOT			
TABLE KEY: Primary muscle Secondary muscles			
Sagittal		Dorsiflexion	Plantarflexion
	Talocrural joint	Tibialis anterior Extensor digitorum longus Extensor hallucis longus Fibularis tertius	Gastrocnemius Soleus Plantaris Tibialis posterior Flexor hallucis longus Flexor digitorum longus Fibularis longus Fibularis brevis
		Flexion	Extension
	Metatarsophalangeal (MTP) joint	Lumbricals Flexor hallucis	Extensor digitorum longus (digits 2–5)

		brevis Interossei Flexor digiti minimi brevis Flexor digitorum longus (digits 2–5) Flexor digitorum brevis (digits 2–5) Flexor hallucis longus (digit 1)	Extensor digitorum brevis Extensor hallucis longus (digit 1)
	Proximal interphalangeal (PIP) joint	Flexor digitorum longus (digits 2–5) Flexor digitorum brevis (digits 2–5) Flexor hallucis longus (digit 1)	Extensor digitorum longus (digits 2–5) Extensor digitorum brevis Extensor hallucis longus (digit 1)
	Distal Interphalangeal (DIP) Joint	Flexor digitorum longus (digits 2–5)	Extensor digitorum longus (digits 2–5) Extensor digitorum brevis
Frontal		Inversion	Eversion

		Tibialis anterior Tibialis posterior Flexor digitorum longus Flexor hallucis longus	Fibularis longus Fibularis brevis Fibularis tertius Extensor digitorum longus

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FOOT AND ANKLE MUSCLE ATTACHMENTS		
Muscles	**Proximal Attachment**	**Distal Attachment**
Tibalis anterior	Lateral condyle and superior 2/3 of anterolateral surface of tibia and interosseous membrane	Medial and inferior surfaces of medial cuneiform and base of 1st metatarsal
Extensor digitorum longus	Lateral condyle of tibia and superior 3/4 of medial surface of fibula and interosseous membrane	Middle and distal phalanges of digits 2–5
Extensor hallucis longus	Middle part of anterior surface of fibula and interosseous membrane	Dorsal aspect of base of distal phalanx of digit 1 (hallux)
Fibularis longus	Head and superior 2/3 of lateral surface of tibia	Base of 1st metatarsal and medial cuneiform

Fibularis brevis	Inferior 2/3 of lateral surface of fibula	Dorsal surface of tuberosity on lateral side of base of 5th metatarsal
Fibularis tertius	Inferior 1/3 of anterior surface of fibula and interosseous membrane	Dorsum of base of 5th metatarsal
Gastrocnemius	Lateral head: lateral aspect of lateral condyle of femur Medial head: Popliteal surface of femur; superior to medial femoral condyle	Posterior surface of calcaneus via calcaneal tendon
Soleus	Posterior aspect of head and superior 1/4 of posterior surface of fibula; middle third of medial border of tibia	Posterior surface of calcaneus via calcaneal tendon
Plantaris	Inferior end of lateral supracondylar line of femur; oblique popliteal ligament	Posterior surface of calcaneus via calcaneal tendon
Flexor hallucis longus	Inferior 2/3 of posterior surface of fibula and interosseous membrane	Base of distal phalanx of digit 1 (hallux)
Flexor digitorum longus	Medial part of posterior surface of tibia inferior to soleal line	Base of distal phalanges of digits 2–5

Tibialis posterior	Interosseous membrane; posterior surface of tibia inferior to soleal line; posterior surface of fibula	Tuberosity of navicular, cuneiform, cuboid, and sustentaculum tali of calcaneus; bases of 2nd, 3rd, and 4th metatarsals
Lumbricals	Tendons of flexor digitorum longus	Medial aspect of expansion (digits 2–5)
Flexor hallucis brevis	Plantar component of cuboid and lateral cuneiforms	Both sides of base of proximal phalanx (digit 1)
Plantar interossei	Plantar aspect of medial sides of shafts of metatarsals 3–5	Medial sides of bases of phalanges of digits 3–5
Dorsal interossei	Adjacent sides of shafts of metatarsals 1–5	Medial side of proximal phalanx of digit 2 Lateral sides of proximal phalanx of digits 2–4
Flexor digiti minimi brevis	Base of 5th metatarsal	Base of proximal phalanx of digit 5
Flexor digitorum brevis	Medial tubercle of tuberosity of calcaneus; plantar aponeurosis	Both sides of middle phalanges (digits 2–5)
Extensor digitorum brevis	Distal and lateral surface of calcaneus in front of groove for fibularis brevis	Long extensor tendons of digits 2–4

Introduction

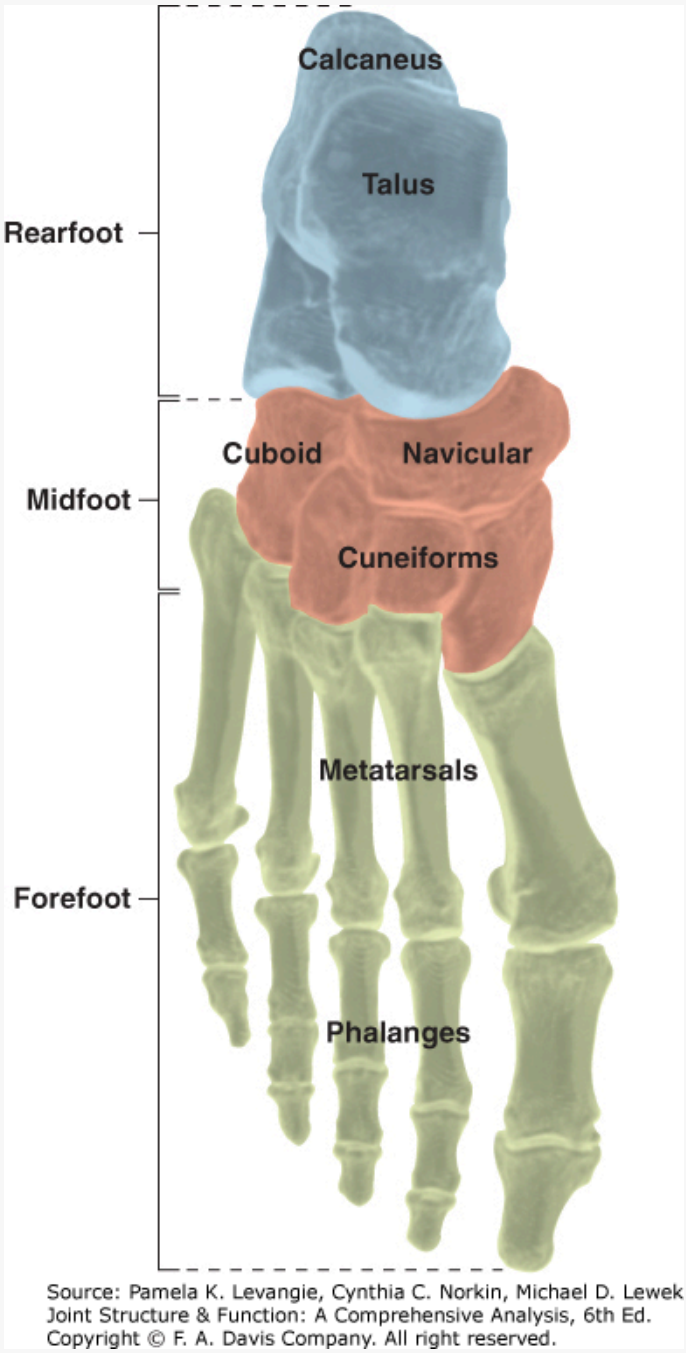
The ankle/foot complex is structurally analogous to the wrist/hand complex of the upper extremity, with adaptations that optimize its primary role to bear weight. The complementing structures of the ankle/foot complex permit both stability and mobility,

depending on current needs. The foot is able to sustain large weightbearing stresses while accommodating to a variety of surfaces and activities. The foot must be stable to provide an adequate base of support and to function as a rigid lever for pushing off when walking, running, or jumping. In contrast, the foot must also be mobile to adapt to uneven terrain, absorb shock as the foot hits the ground, and control forces imposed by the more proximal joints of the lower extremity. The ankle/foot complex meets these diverse requirements through the integrated movements of its 28 bones that form 25 component joints. These joints include the proximal and distal tibiofibular joints, talocrural (ankle) joint, talocalcaneal (subtalar) joint, talonavicular and calcaneocuboid joints (transverse tarsal joints), five tarsometatarsal joints, metatarsophalangeal joints, and nine interphalangeal joints.

To facilitate description and understanding of the ankle/foot complex, the bones of the foot are traditionally divided into three functional segments. These are the **rearfoot** (posterior segment), composed of the talus and calcaneus; the **midfoot** (middle segment), composed of the navicular, cuboid, and three cuneiform bones; and the **forefoot** (anterior segment), composed of the metatarsals and the phalanges (Fig. 12–1). These terms are commonly used in descriptions of ankle/foot dysfunction or deformity and are similarly useful in understanding normal ankle and foot function.

FIGURE 12-1

Functional segments and bones of the foot.



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Ankle or foot pathology can be related to the integrated multifunctional structures of the ankle/foot complex combined with the demands of stability and mobility placed upon these structures. Additionally, the ankle and foot complex must not only respond to forces from the ground but also from imposed forces of the spine, pelvis, hip, and

knee. Structural abnormalities can lead to altered movements between joints and contribute to excessive stresses that result in injury to the ankle/foot complex.[1]

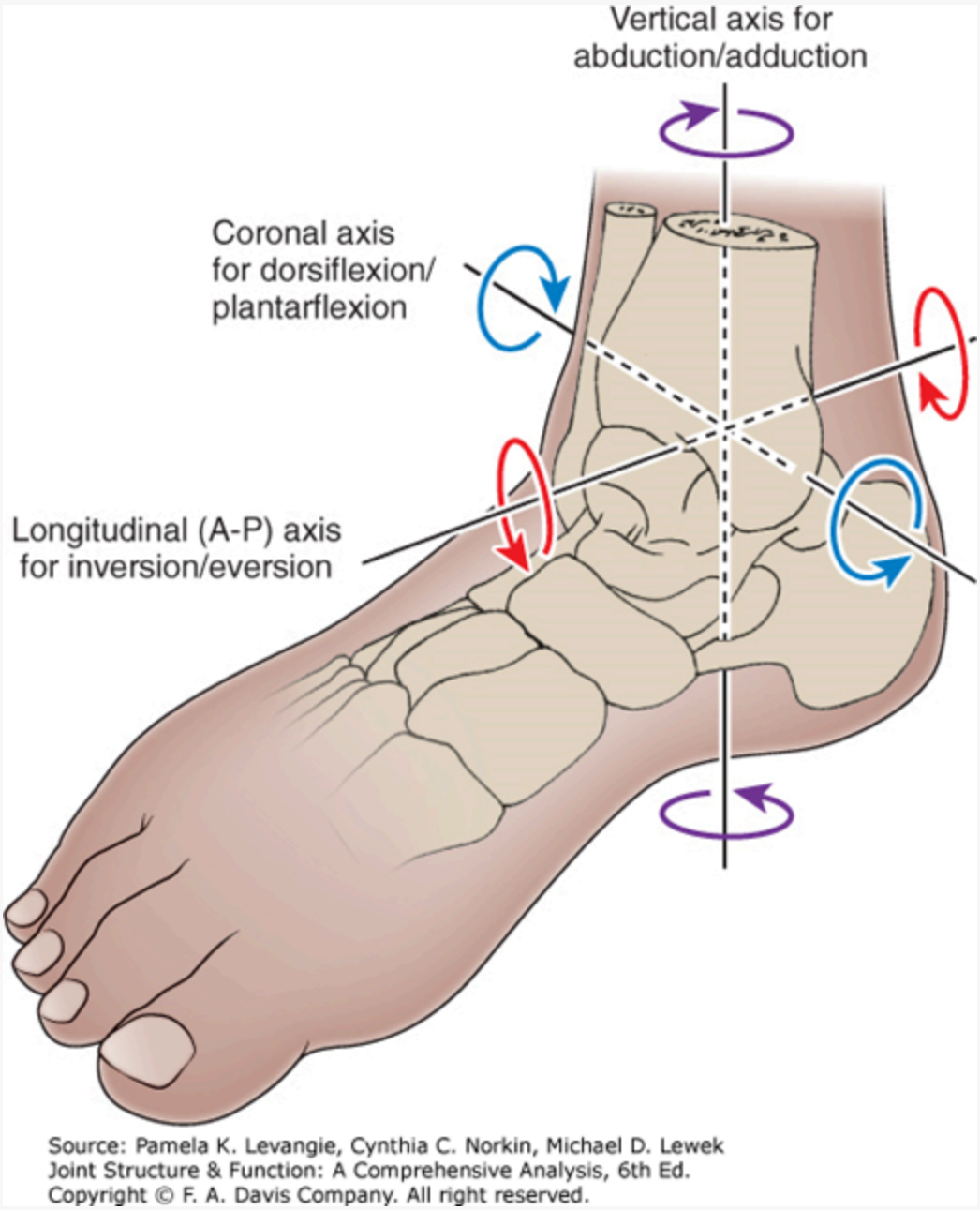
Definitions of Motion

A unique set of terms is used to refer to motion of the foot and ankle. The same terms are used at most of the joints of the ankle and foot, and, consequently, it is useful to describe them at the outset. As we have seen at other joint complexes, few if any of the joint axes lie in the cardinal planes; more commonly, the joint axes are oblique and cut across all three planes of motion. The obliquity of the axes and implications for motion and function will be described in detail as we present individual joints.

The three motions of the ankle/foot complex that *approximate* cardinal planes and axes are dorsiflexion/plantarflexion, inversion/eversion, and abduction/adduction (Fig. 12–2).

FIGURE 12-2

"Cardinal" axes for the motions of the ankle/foot complex.



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Dorsiflexion and plantarflexion are motions that occur approximately in the sagittal plane around a coronal axis. **Dorsiflexion** decreases the angle between the leg and the dorsum of the foot, whereas **plantarflexion** increases this angle. At the toes, motion around a similar axis is termed **extension** (bringing the toes up), whereas the opposite motion is **flexion** (bringing the toes down or curling them). Inversion and eversion occur

approximately in the frontal plane around a longitudinal (anteroposterior [A-P]) axis that runs through the length of the foot. **Inversion** occurs when the plantar surface of the segment is brought toward the midline; **eversion** is the opposite. Abduction and adduction occur approximately in the transverse plane around a vertical axis. **Abduction** is when the distal aspect of a segment moves away from the midline of the body (or away from the midline of the foot in the case of the toes); **adduction** is the opposite.

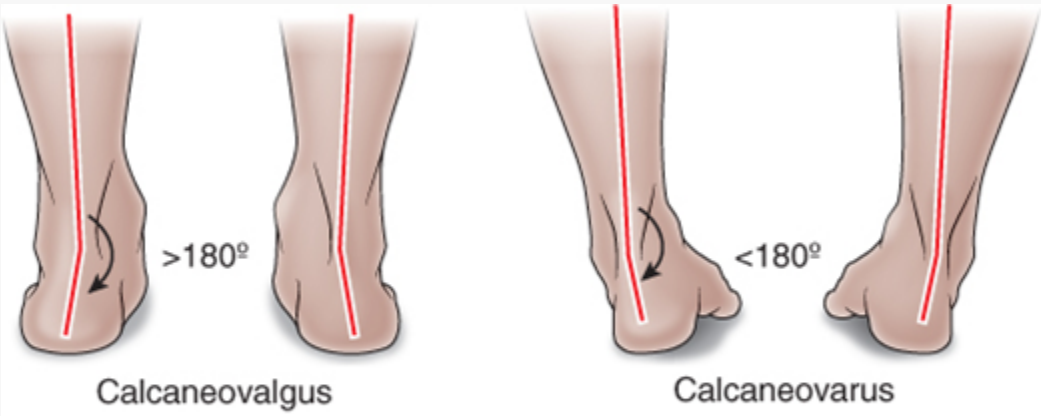
Pronation/supination in the foot are motions that occur around an axis that lies at an angle to each of the axes for "cardinal" motions of dorsiflexion/plantarflexion, inversion/eversion, and abduction/adduction. Consequently, pronation and supination are terms used to describe "composite" motions that have components of, or are coupled to, each of the cardinal motions. In non-weightbearing, **pronation** is motion about an axis that results in coupled motions of dorsiflexion, eversion, and abduction. Likewise, **supination** is a motion about an axis that results in coupled motions of plantarflexion, inversion, and adduction. The proportional contribution that each of the coupled motions makes to pronation/supination is dependent on and varies with the angle of the pronation/supination joint axis.

Valgus and varus are terms that may be used for the ankle/foot complex in several ways, depending on the context. The definitions that we used throughout discussion of other joints in other chapters will not change. That is, **valgus** refers to an increase in the medial angle between two bones (or movement of the distal segment away from the midline); **varus** refers to the opposite. However, valgus and varus are sometimes used to refer to *fixed deformities* in the ankle/foot complex, whereas at other times the terms

are used to describe or as synonyms for other normal motions. An example of common usage is to describe the fixed or weightbearing position of the posterior calcaneus in relation to the posterior midline of the leg, with an increase in the medial angle between the two reference lines being valgus of the calcaneus (or calcaneovalgus) and a decrease being varus of the calcaneus (or calcaneovarus; Fig. 12–3). We will define the use and context as we encounter these terms in descriptions of ankle/foot structure and function.

FIGURE 12-3

The term valgus (or calcaneovalgus) refers to an increase in the medial angle between the calcaneus and posterior leg. The term varus (or calcaneovarus) refers to a decrease in the medial angle between the calcaneus and posterior leg.



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Foundational Concepts 12–1

Ankle/Foot Terminology

As we have seen at other joints, terminology used to describe motions around a joint

or of a segment is often not consistent among investigators. This is very much the case for the ankle/foot complex. Because the anterior surface of the leg and the top of the foot are embryologically dorsal surfaces, dorsiflexion may also be referred to as extension, and plantarflexion may be referred to as flexion.[2] Although the flexion/extension terminology is commonly used for the toes, it may also be applied to the ankle. Some sources use the term “inversion/eversion” to refer to the composite motion of “supination/pronation”. As we proceed to describe the joints and their motions, we will see that some of these terminology differences are not really as problematic as they might initially seem.

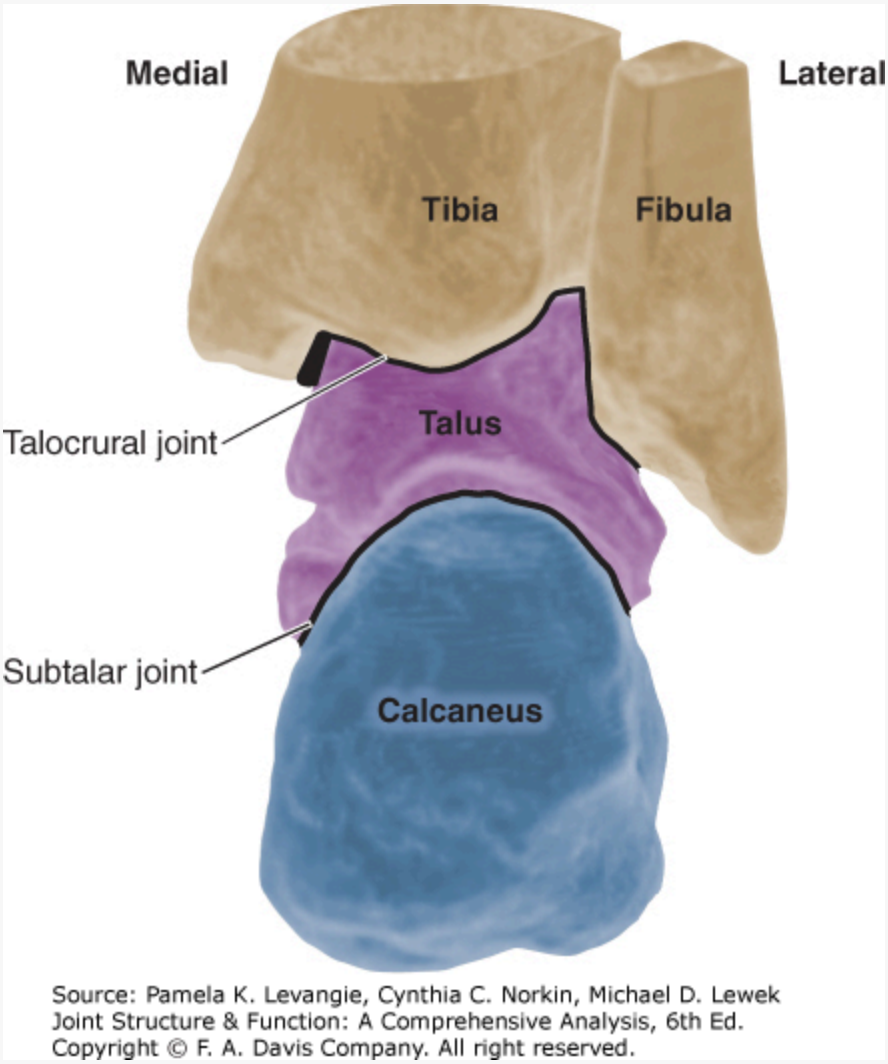
Joint Structure

The Ankle Joint

The term **ankle** refers specifically to the talocrural joint defined as the articulation between the distal tibia and fibula proximally and the body of the talus distally (Fig. 12–4). The ankle is a synovial hinge joint with a joint capsule and associated ligaments.

FIGURE 12-4

The ankle joint is formed by the tibia and fibula (mortise) proximally and by the talus distally. This posterior view shows the talocrural joint proximal to the talus and the subtalar joint distal to the talus.



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Proximal Articular Surfaces

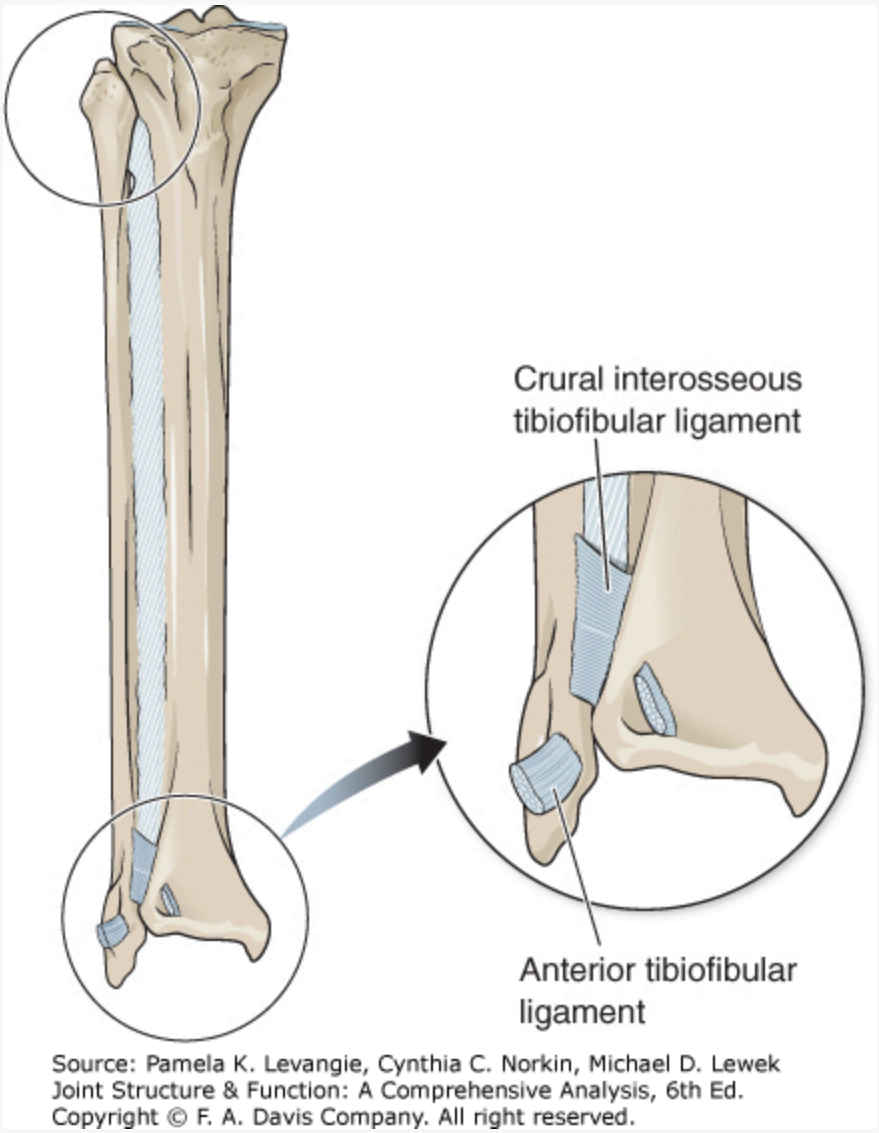
The proximal segment of the ankle is composed of the concave surface of the distal tibia (known as the tibial plafond) and of the tibial and fibular malleoli. These three facets form an almost continuous concave joint surface that extends more distally on the fibular (lateral) side than on the tibial (medial) side (see Fig. 12–4), and more distally on the posterior margin of the tibia than on the anterior margin. The structure of the distal tibia and two malleoli resembles and is referred to as a mortise. A common example of a mortise is the gripping part of a wrench. Either the wrench can be fixed

(fitting a bolt of only one size) or it can be adjustable (permitting use of the wrench on a variety of bolt sizes). The adjustable mortise is more complex than a fixed mortise because it combines mobility and stability functions. The mortise of the ankle is adjustable, relying on the proximal and distal tibiofibular joints to both permit and control the changes in the mortise width.

The proximal and distal tibiofibular joints (Fig. 12–5) are anatomically distinct but functionally linked to the ankle. Unlike their upper extremity counterparts (i.e., the proximal and distal radioulnar joints), the tibiofibular joints do not add any degrees of freedom to the more distal ankle and foot. However, the tibiofibular joints do contribute to total ankle motion. Fusion of the radioulnar joints would have little effect on wrist range of motion (ROM), whereas fusion of the tibiofibular joints may impair normal ankle function.

FIGURE 12-5

Proximal and distal tibiofibular joints.



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Proximal Tibiofibular Joint

The proximal tibiofibular joint is a plane synovial joint formed by the articulation of the head of the fibula with the posterolateral aspect of the tibia. The facets of the proximal tibiofibular joint are fairly flat and can vary in configuration between individuals. Generally, a convex tibial facet and concave fibular facet is the most common articulation pattern.[3] The proximal tibiofibular joint is surrounded by a joint capsule that is reinforced by anterior and posterior tibiofibular ligaments. Although the

proximal tibiofibular joint is generally thought to be anatomically separate from the knee joint, communication between these joints can occur.[4] Studies looking at proximal tibiofibular joint movement have shown variation with respect to fibular translation and rotation that occurs with ankle dorsiflexion.[5] The motion at this joint is consistently small; however, symptoms of instability can occur following trauma to this area.[6] The relevance of motion at the proximal and distal tibiofibular joints will be seen when ankle joint motion is discussed.

Distal Tibiofibular Joint

The distal tibiofibular joint is a syndesmosis, or fibrous union, between the concave facet of the tibia and the convex facet of the fibula. The distal tibia and fibula do not actually come into contact with each other but are separated by fibroadipose tissue. Although there is no joint capsule, there are several associated ligaments. Because the proximal and distal joints are linked (the tibia, fibula, and tibiofibular joints are part of a closed chain), all the ligaments that lie between the tibia and fibula contribute to stability at both joints.

The ligaments of the distal tibiofibular joint are primarily responsible for maintaining a stable mortise. The **anterior** and **posterior tibiofibular ligaments** and **interosseous membrane** provide support to the distal tibiofibular joint.[7] The interosseous membrane directly supports both proximal and distal tibiofibular articulations. Although the distal tibiofibular joint is an extremely strong articulation, injuries can occur when the talus is forcibly dorsiflexed and abducted within the ankle mortise.[8] These injuries can compromise the integrity of the joint causing the fibula and tibia to separate and are diagnosed as a high or syndesmotic ankle sprain. If the force were to continue, fracture

of the fibula proximal to the distal tibiofibular ligaments could result.[9]

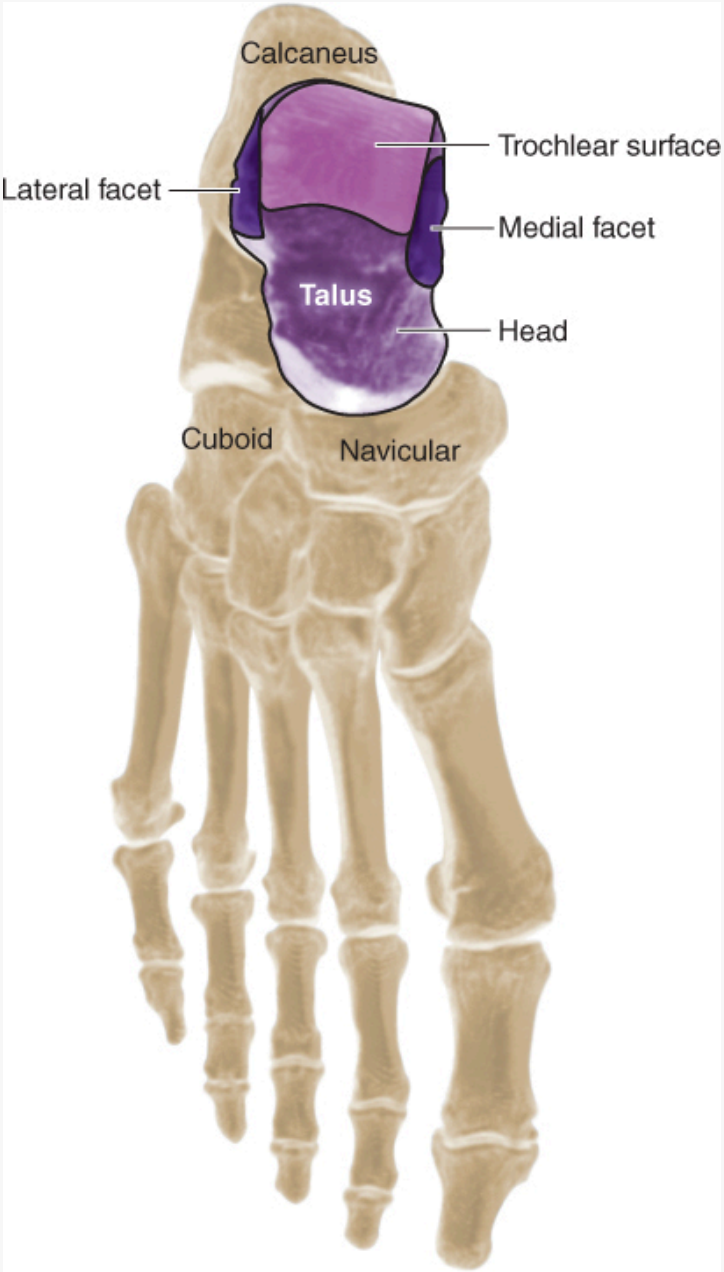
The function of the ankle (talocrural) joint is dependent on stability of the tibiofibular mortise. The ankle mortise would be unable to grasp and hold on to the talus if the tibia and fibula were permitted to separate or if one side of the mortise were missing. This would be similar to a wrench that could not perform its function of grasping a bolt if the two pincer segments moved apart every time a force was applied to the wrench. Conversely, the ankle mortise must have some mobility for normal function. The mobility role of the mortise belongs primarily to the fibula. The fibula is generally thought to have little weightbearing function. One study found the fibula transmitted no more than 10% of weightbearing forces.[10] However, the amount of axial load through the fibula may be related to positioning as greater weightbearing forces were transmitted through the fibula when the ankle was in a position of eversion.[11] Because the fibula is mobile, movement at the proximal and distal tibiofibular joints must be linked.

Distal Articular Surface

The body of the talus (Fig. 12–6) forms the distal articulation of the ankle joint. The body of the talus has three articular surfaces: a large lateral (fibular) facet, a smaller medial (tibial) facet, and a trochlear (superior) facet. The large, convex trochlear surface has a central groove that runs at a slight angle to the head and neck of the talus. The body of the talus also appears wider anteriorly than posteriorly, which gives it a wedge shape. The articular cartilage covering the trochlea is continuous with the cartilage covering the more extensive lateral facet and the smaller medial facet.

FIGURE 12-6

The body of the talus with its trochlear (superior) surface, medial (tibial) facet, and lateral (fibular) facet form the distal aspect of the ankle joint.



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In addition to the bony articulations, the structural integrity of the ankle joint is maintained throughout the ROM of the joint by a number of supportive soft tissue structures.

Capsule and Ligaments

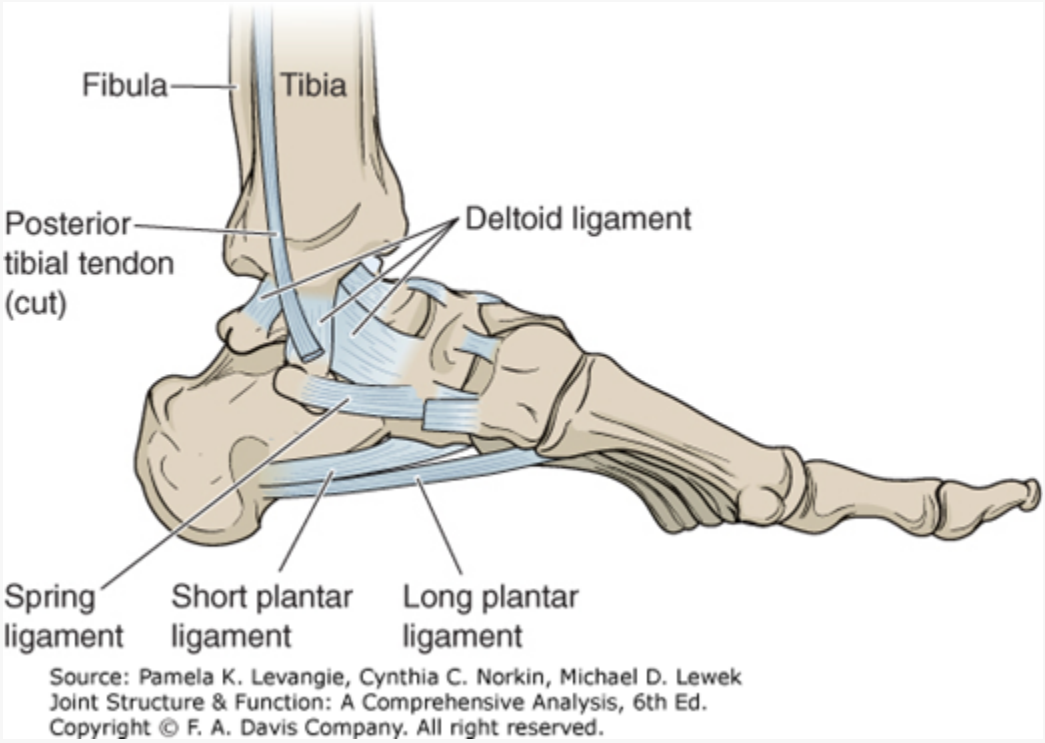
The capsule of the ankle joint is fairly thin and especially weak anteriorly and posteriorly. Therefore, the stability of the ankle depends on an intact ligamentous structure. The ligaments that support the proximal and distal tibiofibular joints (the crural tibiofibular interosseous ligament, the anterior and posterior tibiofibular ligaments, and the tibiofibular interosseous membrane) are important for stability of the mortise and, therefore, for stability of the ankle. Two other major ligament complexes maintain contact and congruence of the mortise and talus and control medial-lateral ankle joint stability. These are the medial collateral ligament (MCL) and the lateral collateral ligament (LCL). Portions of these ligaments also provide support for the subtalar (or talocalcaneal) joint that they also cross.[12] Therefore, function of the collateral ligaments at the ankle joint can be difficult to separate from their function at the subtalar joint.

The MCL is commonly called the **deltoid ligament.** As its name implies, the deltoid ligament is fan-shaped. It has superficial and deep fibers that arise from the borders of the tibial malleolus and insert in a continuous line anterior to posterior on the navicular, talus, and calcaneus (**Fig. 12–7**).[13] The deltoid ligament as a whole is extremely strong, which partially accounts for the relative low prevalence of injury to these ligaments. Eversion or pronation of the ankle and talus (i.e. outward rotation of the foot combined with inward rotation of the tibia) can injure the deltoid ligament.[14,15]

However, these forces may actually fracture and displace (avulse) the tibial malleolus before the deltoid ligament tears.

FIGURE 12-7

Medial ligaments of the posterior ankle/foot complex.

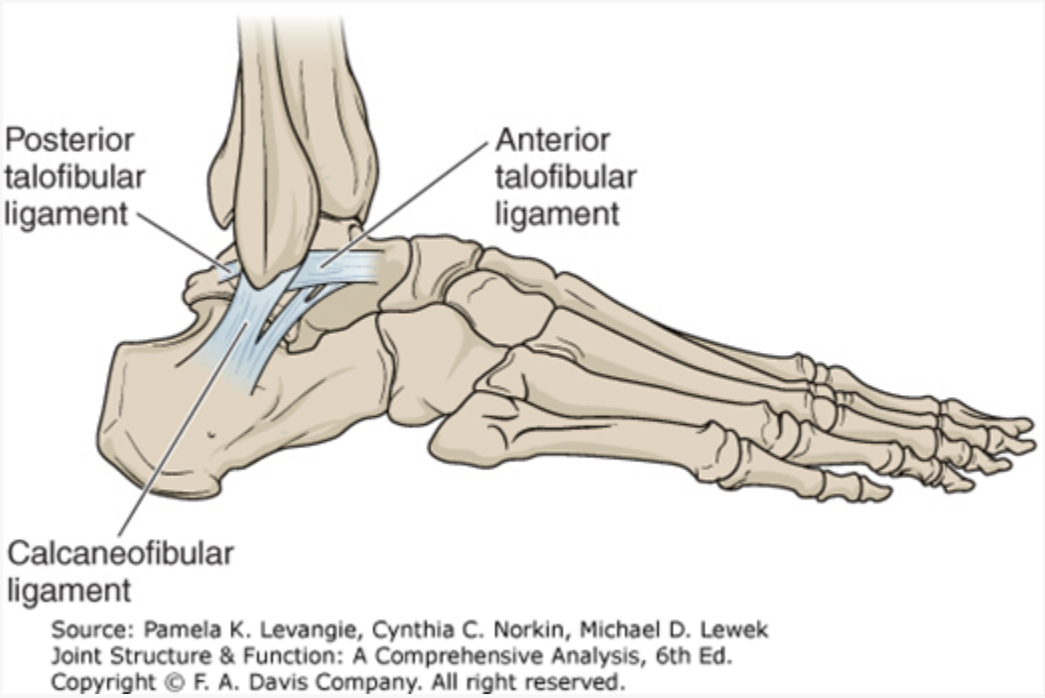


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The LCL is composed of three distinct bands that are commonly referred to as separate ligaments. These are the **anterior** and **posterior talofibular** ligaments and the **calcaneofibular** ligament (Fig. 12–8). The anterior and posterior ligaments run in a fairly horizontal position, whereas the longer calcaneofibular ligament is nearly vertical.[16] In contrast with the MCL, the LCL helps control inversion or supination of the ankle and talus (i.e., inward rotation of the foot combined with outward rotation of the tibia).[14,17] The components of the LCL are weaker and more susceptible to injury than the MCL.

FIGURE 12-8

Lateral ligaments of the posterior ankle/foot complex.



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Foundational Concepts 12–2

Summary of Studies Investigating Stresses Applied to the LCL

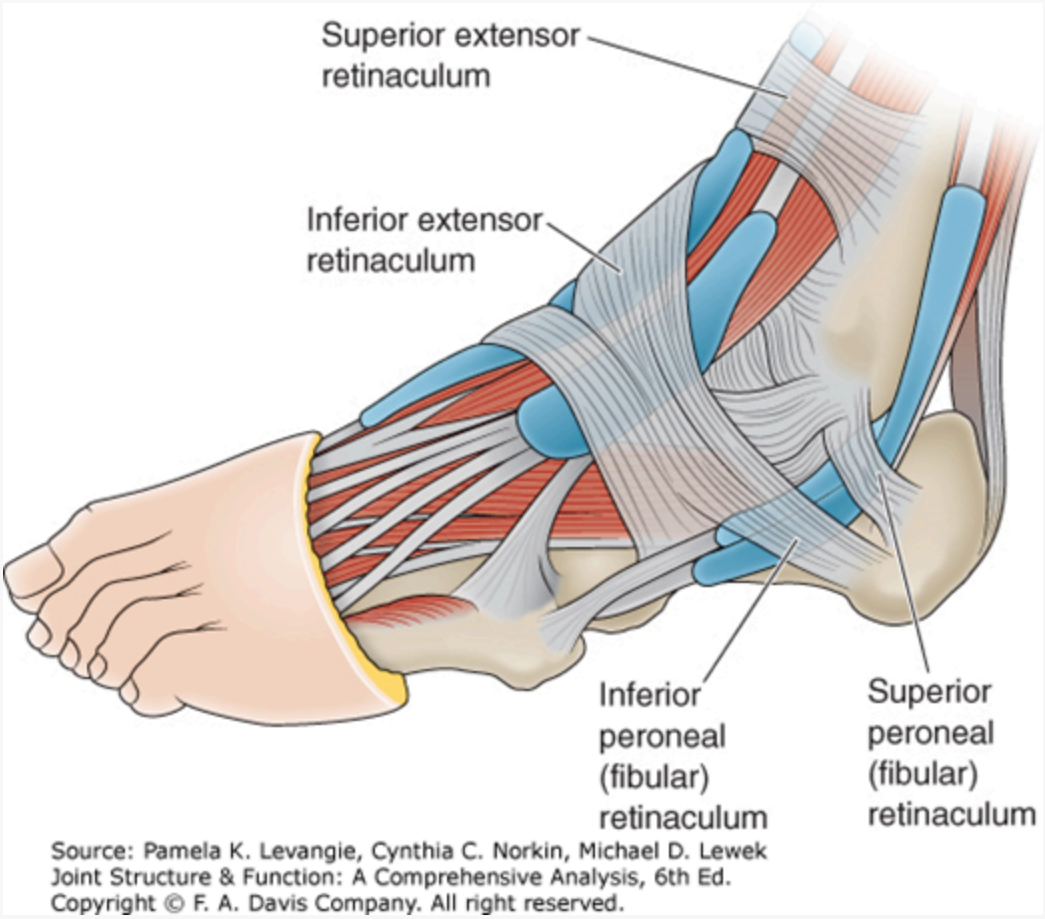
1. In comparison with the other two ligaments that make up the LCL, the anterior talofibular ligament is the weakest and most commonly injured.[18] This ligament is stressed when the ankle is moved into greater degrees of plantarflexion, adduction, and inversion.[19] Rupture of the anterior talofibular ligament often results in anterolateral rotatory instability of the ankle.[20]
2. The calcaneofibular is stressed when the ankle is dorsiflexed and inverted while the posterior talofibular ligament is stressed when the ankle is dorsiflexed and abducted.[19] The posterior talofibular ligament is rarely torn in isolation, but can be injured in more severe dislocations.
3. The contribution of each of the LCL components in restraining motion of the talus in the mortise depends on the position of the ankle joint.[14,17] Therefore,

to assess these ligaments, testing needs to be done with the ankle in multiple positions and planes. The anterior drawer and talar tilt are clinical ligament stress tests used to assess the anterior talofibular and calcaneofibular ligaments, respectively. Although these tests are commonly used, there appears to be poor diagnostic accuracy in either identifying the degree of ligamentous disruption or the particular ligaments involved.[21]

In addition to the MCL and LCL, portions of the extensor and peroneal retinaculae of the ankle are also credited with contributing to stability at the ankle joint.[22,23] The inferior band of the **superior peroneal retinaculum** (see Fig. 12–9), which lies close and parallel to the calcaneofibular ligament, appears to reinforce that ligament.[23] Because the ankle collateral ligaments and retinaculae also contribute to stability of the subtalar joint they will be discussed again in that context.

FIGURE 12-9

The superior and inferior extensor retinacula; the superior and inferior peroneal retinacula.



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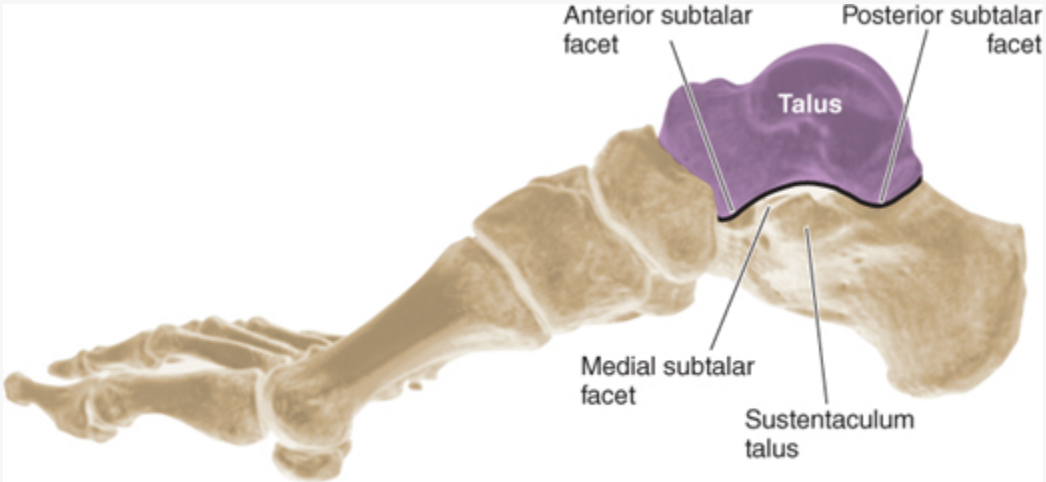
The Subtalar Joint

The talocalcaneal, or subtalar, joint is a composite joint formed by three separate plane articulations between the talus superiorly and the calcaneus inferiorly. The subtalar joint articulating surfaces are highly variable, but the posterior articulation is consistently the largest of the three articulations found between the talus and calcaneus. The posterior articulation is formed by a concave facet on the undersurface of the body of the talus and a convex facet on the body of the calcaneus. The smaller anterior and middle talocalcaneal articulations are formed by two convex facets on the inferior body and neck of the talus and two concave facets on the calcaneus (Fig. 12–10). The anterior and middle articulations, therefore, have an intra-articular

configuration that is the reverse of that found at the posterior facet. Between the posterior articulation and the anterior and middle articulations, there is a bony tunnel formed by a **sulcus** (concave groove) in the inferior talus and superior calcaneus. This funnel-shaped tunnel, known as the tarsal canal, runs obliquely across the foot. Its large end (the **sinus tarsi)** lies just anterior to the fibular malleolus (Fig. 12–11); its small end lies posteriorly below the tibial malleolus and above a bony outcropping on the calcaneus called the **sustentaculum tali** (see Fig. 12–10). The tarsal canal and ligaments running the length of the tarsal canal divide the posterior articulation and the anterior and middle articulations into two separate non-communicating joint cavities. The posterior articulation has its own capsule whereas the anterior and middle articulations share a capsule with the **talonavicular joint.**[24]

FIGURE 12-10

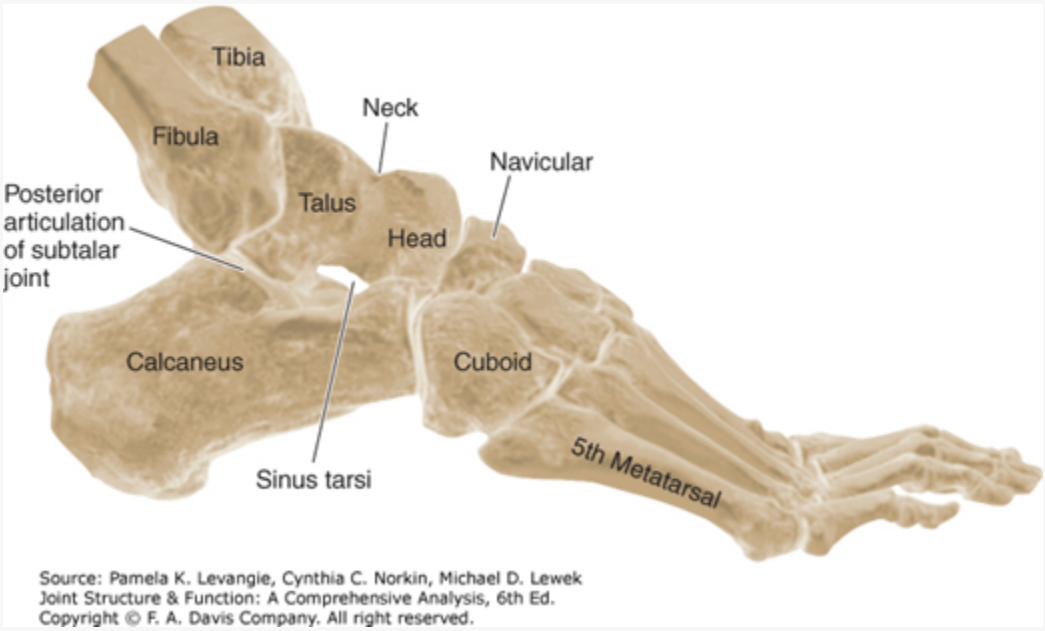
Medial view of the foot, showing the talus sitting on the calcaneus (the subtalar joint).



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FIGURE 12-11

Lateral view of the foot, showing the talus sitting on the calcaneus (the subtalar joint). The sinus tarsi is the lateral opening of the tarsal canal.



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Wang and colleagues found that the subtalar articular surfaces, although smaller than those of the ankle joint surfaces, showed a similar proportion of contact across surfaces under similar conditions.[25] These investigators found that the posterior facet received 75% of the force transmitted through the subtalar joint. Given the larger contact area of the posterior facet, it was determined that the pressure in the posterior facet was similar to that at the middle and anterior facets. Like the ankle joint, the subtalar joint rarely undergoes degenerative change unless damaged by high stresses (e.g., fracture).

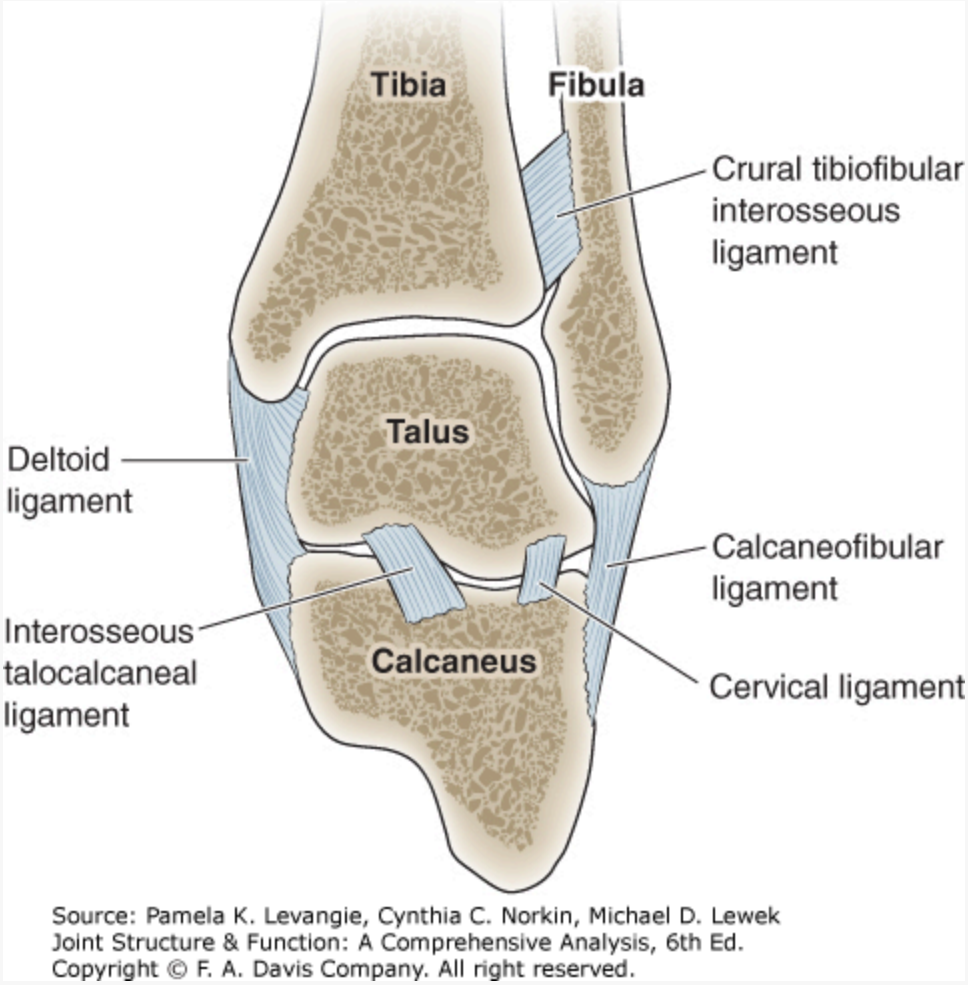
Ligaments

The subtalar joint is a stable joint that is rarely dislocated. It has a congruent osseous

anatomy as well as strong ligamentous support. The subtalar joint receives support from the ligamentous structures that support the ankle, as well as from ligamentous structures that only cross the subtalar joint.[24] Harper, through anatomical studies, has described a number of structures contributing to the lateral support of the subtalar joint.[26] These included, from superficial to deep, the calcaneofibular, lateral talocalcaneal, cervical, and interosseous talocalcaneal ligaments. The cervical ligament (**Fig. 12–12**) is the strongest of the talocalcaneal structures.[26,27] It lies in the anterior sinus tarsi and joins the neck of the talus to the neck of the calcaneus (hence its name). The interosseous talocalcaneal ligament lies more medially within the tarsal canal and follows an oblique path from the talus to the calcaneus (see Fig. 12–12).[28] Divisions of the inferior extensor retinaculum also provide stability to the subtalar joint superficially as well as through connections within the tarsal canal.[28] Although the subtalar joint is considered to be very stable, controversy exists as to the hierachy of support from bony configuration and the relevant ligaments (i.e., calcaneofibular ligament, cervical ligament, and interosseous ligament).[29]

FIGURE 12-12

The ligaments of the subtalar joint (in a posterior cross-sectional view).



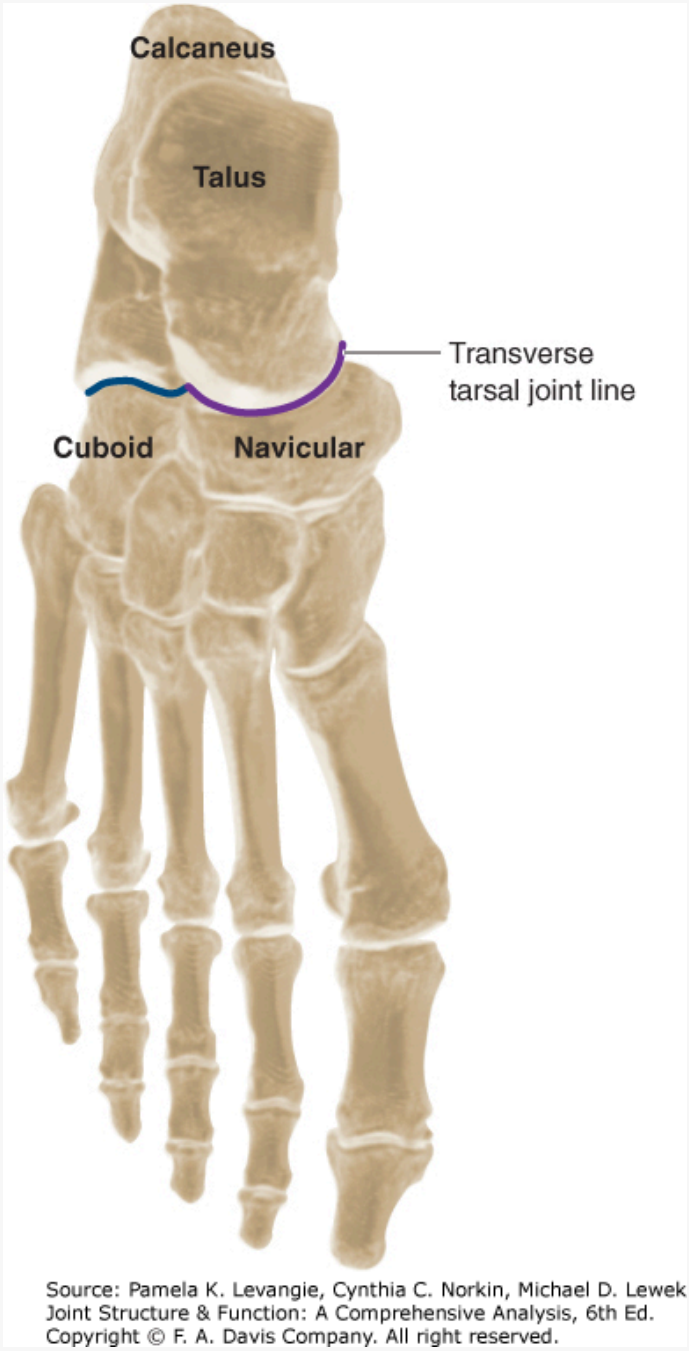
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Transverse Tarsal Joint

The transverse tarsal joint, also called the midtarsal or Chopart joint, is a compound joint formed by the talonavicular and calcaneocuboid joints (Fig. 12–13).

FIGURE 12-13

The talonavicular joint and calcaneocuboid joint form a compound joint known as the transverse tarsal joint line that transects the foot.



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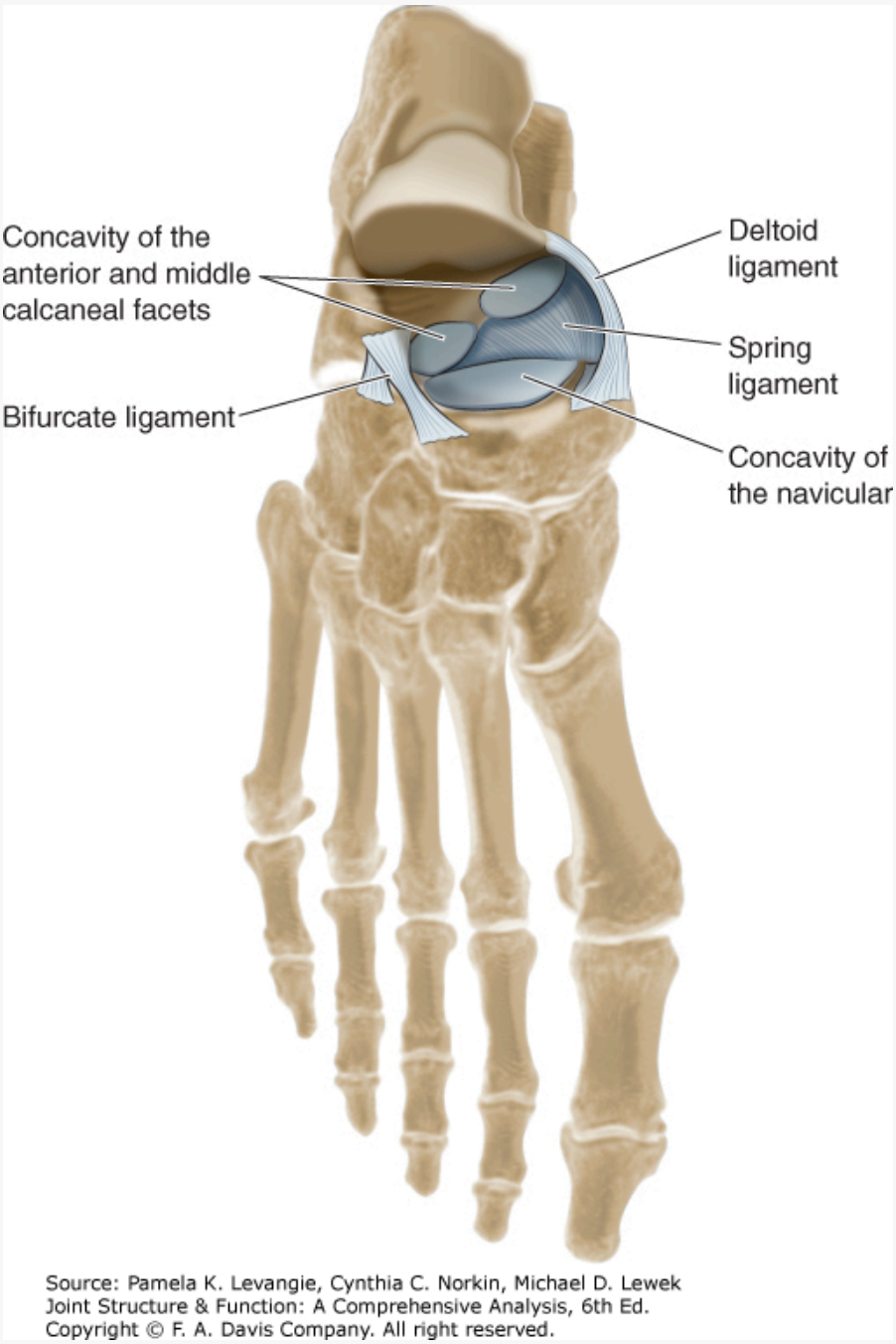
Talonavicular Joint

The proximal portion of the talonavicular articulation is formed by the anterior portion of the head of the talus, with the distal articulation formed by the concave posterior aspect of the navicular bone. We also noted earlier that the talar head articulates

inferiorly with the anterior and middle facets of the calcaneus as the anterior part of the subtalar joint. A single joint capsule encompasses the talonavicular joint facets and the anterior and middle facets of the subtalar joint. The inferior aspect of this joint capsule is formed by the plantar calcaneonavicular ligament (spring ligament) that spans the gap between the calcaneus and navicular immediately below the talar head. The capsule is reinforced medially by the deltoid and laterally by the bifurcate ligament. Given these structural relationships, the large convexity of the head of the talus can be considered the “ball” that is received by a large “socket.” This “socket” is formed anteriorly by the concavity of the navicular bone, inferiorly by the concavities of the anterior and middle calcaneal facets and by the plantar calcaneonavicular ligament, medially by the deltoid ligament, and laterally by the bifurcate ligament (Fig. 12–14).

FIGURE 12-14

With the talus removed, this superior view shows the concavity (“socket”) formed by the navicular bone anteriorly, the deltoid ligament medially, the medial band of the bifurcate ligament laterally, and the spring (plantar calcaneonavicular) ligament inferiorly.



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The spring (plantar calcaneonavicular) ligament (see Figs. 12–7 and 12–14) is a triangular sheet of ligamentous connective tissue arising from the sustentaculum tali of the calcaneus and inserting on the inferior navicular bone. The spring ligament is continuous medially with a portion of the deltoid ligament of the ankle and joins

laterally with the medial band of the bifurcate ligament.[30] Recent evidence suggests it is composed of three bands: superomedial, medioplantar oblique, and inferoplantar longitudinal.[31–33] The spring ligament plays an important role in supporting the head of the talus and talonavicular joint, as well as functioning as one of the main static or passive stabilizers of the medial longitudinal arch.[30,34] We already noted that the talonavicular facets and the anteriorly located talocalcaneal facets share a joint capsule. The large posterior facet of the subtalar joint is contained within its own capsule and is physically separated from the capsule containing the talonavicular joint by the tarsal canal and the ligaments within the canal. However, the talonavicular joint and the subtalar joint are linked in the weightbearing foot. Weightbearing eversion/inversion and abduction/adduction of the talus on the calcaneus during subtalar supination/pronation involve simultaneous movement of the head of the talus on the relatively fixed navicular bone. In weightbearing, therefore, the talonavicular and subtalar joints are both anatomically and functionally related.[35]



Foundational Concepts 12–3

Talar Linkages

Because of this anatomic and functional linkage of the talus to the structures below and anterior to it, the subtalar and talonavicular joints have been referred to by the compound term **talocalcaneonavicular joint.**[2] However, it might be argued that even this compound term is incomplete. The talus in weightbearing also can be considered to act as a ball bearing between three joints: (1) the tibiofibular mortise (the ankle joint) superiorly, (2) the calcaneus (the subtalar joint) inferiorly, and (3) the navicular bone (the talonavicular joint) anteriorly. In weightbearing supination/pronation, the tibia/fibula (ankle mortise) dorsiflexes and plantarflexes on the talus. Abduction/adduction and inversion/eversion of the talus during supination/pronation not only occurs on the calcaneus and the navicular bone but also affects the position of the mortise as the body of the talus laterally and medially rotates.

The ligaments of the talonavicular joint include the spring and bifurcate ligaments. In addition, the talonavicular articulation is also supported by the **dorsal talonavicular ligament** and receives support from the ligaments of the subtalar joint—including the MCL and LCL, the inferior extensor retinacular structures, and the cervical and interosseous talocalcaneal ligaments. Support is also received from the ligaments that reinforce the adjacent calcaneocuboid joint, which forms the remainder of the transverse tarsal joint and to which the talonavicular joint is linked functionally.

Calcaneocuboid Joint

The calcaneocuboid joint is formed proximally by the anterior calcaneus and distally by the posterior cuboid bone (see Fig. 12–13). The articular surfaces of both the calcaneus and the cuboid bone are complex, being reciprocally concave/convex both side to side and top to bottom. The reciprocal shape makes available motion at the calcaneocuboid joint more restricted than that of the ball-and-socket–shaped talonavicular joint. The calcaneocuboid joint, like the talonavicular joint, is linked in weightbearing to the subtalar joint.[35] In weight-bearing subtalar supination/pronation, the inversion/eversion of the calcaneus on the talus causes the calcaneus to move simultaneously on the relatively fixed cuboid bone. As the calcaneus moves at the subtalar joint during weightbearing activities, it must meet the conflicting intra-articular demands of the opposing saddle-shaped surfaces, which results in a twisting motion.

The calcaneocuboid articulation has its own capsule that is reinforced by several important ligaments. The capsule is reinforced laterally by the lateral band of the bifurcate ligament (also known as the **calcaneocuboid ligament),** dorsally by the **dorsal calcaneocuboid ligament,** and inferiorly by the **plantar calcaneocuboid (short plantar)**

and the **long plantar ligaments.** The long plantar ligament is the most important of these ligaments, because the inferiorly located long plantar ligament spans the calcaneus and the cuboid bone and then continues on distally to the bases of the second, third, and fourth metatarsals. Therefore the long plantar ligament provides support to the transverse tarsal joint as well as the lateral longitudinal arch of the foot.[36] The extrinsic muscles of the foot also provide important support for the transverse tarsal joint as they pass medial, lateral, and inferior to the joint.

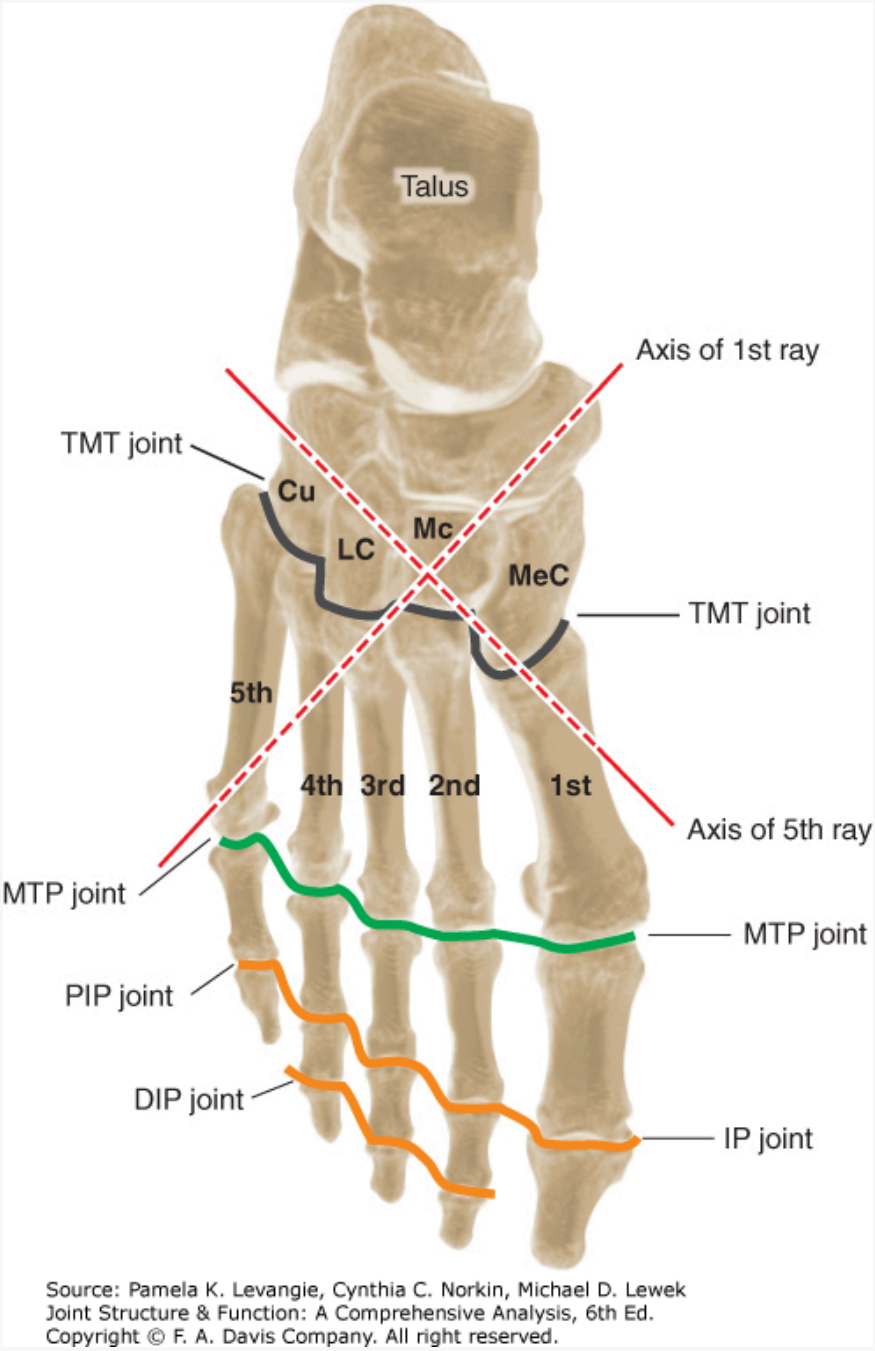
Tarsometatarsal Joints

The tarsometatarsal (TMT) joints are plane synovial joints formed by the distal row of tarsal bones (posteriorly) and the bases of the metatarsals (Fig. 12–15). The first (medial) TMT joint is composed of the articulation between the base of the first metatarsal and the medial cuneiform bone, and has its own articular capsule. The second TMT joint is composed of the articulation of the base of the second metatarsal with a mortise formed by the middle cuneiform bone and the sides of the medial and lateral cuneiform bones. This joint is set more posteriorly than the other TMT joints with restricted motion. The third TMT joint, formed by the third metatarsal and the lateral cuneiform, shares a capsule with the second TMT joint. The bases of the fourth and fifth metatarsals, with the distal surface of the cuboid bone, form the fourth and fifth TMT joints. These two joints also share a common joint capsule. Small plane articulations exist between the bases of the metatarsals to permit motion of one metatarsal on the next. Numerous dorsal, plantar, and interosseous ligaments reinforce each TMT joint. In addition, there is a deep transverse metatarsal ligament that spans the heads of the metatarsals on the plantar surface and is similar to that found in the hand.[37,38] Just as the deep transverse metacarpal ligament contributed to stability of the more proximally

located carpometacarpal (CMC) joints, the deep transverse metatarsal ligament contributes to stability of the proximally located TMT joints by preventing excessive motion and splaying of the metatarsal heads.[39]

FIGURE 12-15

Tarsometatarsal (TMT), metatarsophalangeal (MTP), and interphalangeal (IP: PIP and DIP) joints of the foot, showing the axes of the first and fifth TMT joints. CU, cuboid; LC, lateral cuneiform; MC, middle cuneiform; MeC, medial cuneiform.



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Metatarsophalangeal Joints

The five metatarsophalangeal (MTP) joints are condyloid synovial joints with two degrees of freedom: extension/flexion (or dorsiflexion/plantarflexion) and abduction/adduction. The MTP joints are formed proximally by the convex heads of the

metatarsals and distally by the concave bases of the proximal phalanges (see Fig. 12–15).

Expanded Concepts 12–1

Metatarsal Length

The lengths of the five metatarsals may vary between individuals and are classified into three types. Index plus is characterized by the first metatarsal being longer than the second, with the other remaining three progressively decreasing in length. Index plus minus is characterized by the first and second metatarsal being the same length, with the other remaining three metatarsals progressively decreasing in length. Index minus, or Morton's foot, is classified by the second metatarsal being longest followed progressively by the first, third, fourth and fifth.[40] A Morton's type foot has been hypothesized to cause an increase force through the second metatarsal, predisposing it to injury, such as stress fractures. The relationship between metatarsal length and injury pattern, however, is unclear as the literature has not fully supported this hypothesis in athletes.[40,41]

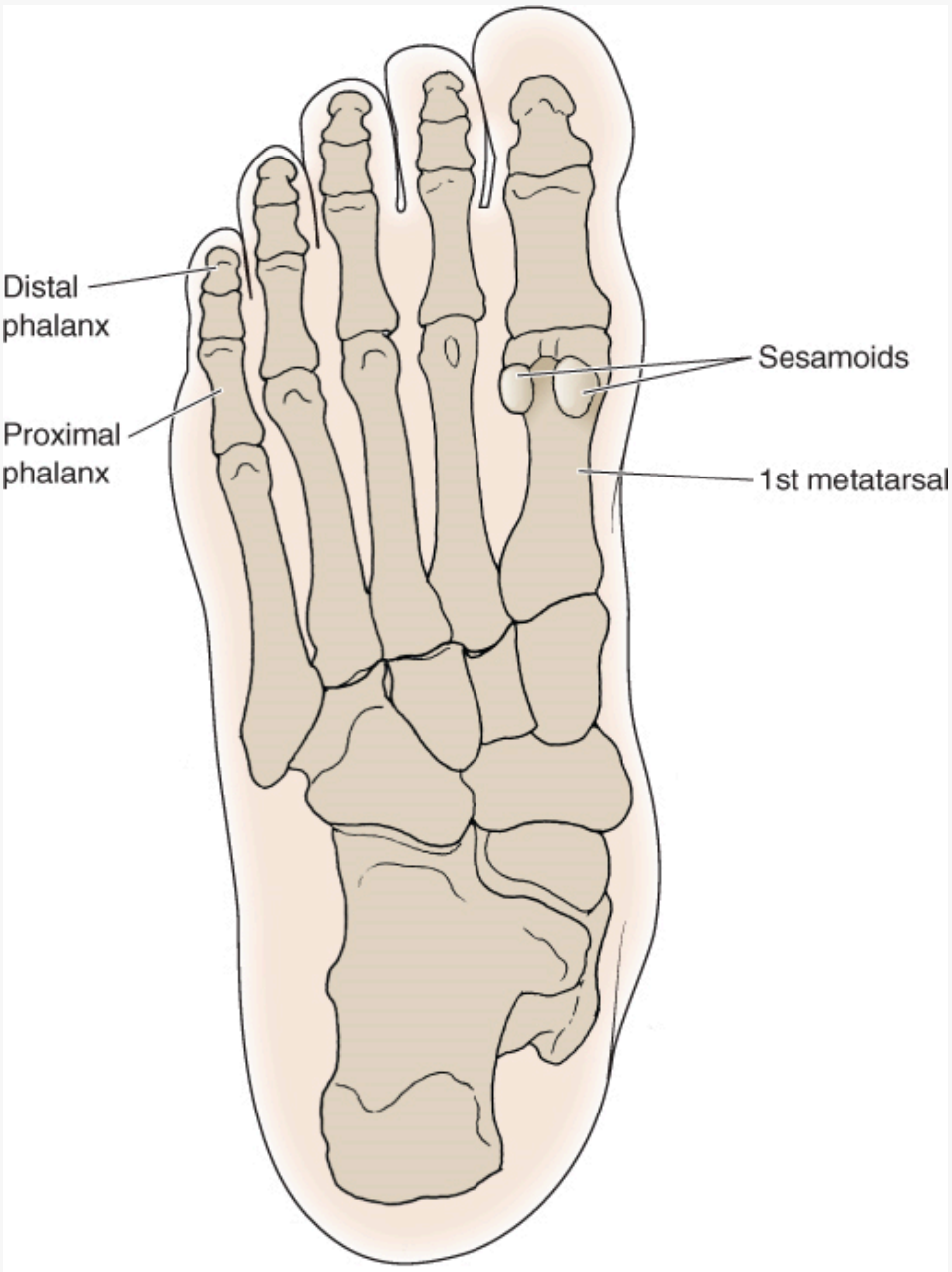
The structure of the MTP joints is analogous to the structure of the metacarpophalangeal (MCP) joints of the hands, with a few exceptions. Unlike the MCP joints, the range of MTP extension exceeds the range of MTP flexion. All metatarsal heads bear weight in stance. Consequently, the articular cartilage must remain clear of the weightbearing surface on the plantar aspect of the metatarsal head. This structural requirement restricts the available range of MTP flexion. Also in contrast to the hand, there is no opposition available at the first MTP joint; the first toe **(hallux)** moves exclusively in the same planes as the other four digits.

The first MTP joint has two sesamoid bones associated with it that are located on the plantar aspect of the first metatarsal head (Fig. 12–16). These are analogous to the sesamoid bones on the volar surface of the MCP joint. In the neutral position of the first

MTP joint, the sesamoid bones lie in two grooves, separated by the intersesamoid ridge, on the metatarsal head.[42,43] The ligaments associated with the sesamoid bones form a triangular mass that stabilizes the sesamoid bones within their grooves.[42,44] The sesamoid bones serve as anatomic pulleys for the **flexor hallucis brevis** muscle and protect the tendon of the **flexor hallucis longus** muscle from weightbearing trauma. The flexor hallucis longus is protected as it passes through a tunnel formed by the sesamoid bones and the intersesamoid ligament that connects the sesamoid bones across their plantar surfaces.[42,45] Unlike the sesamoid bones of the thumb, the sesamoid bones of the first metatarsal absorb weightbearing stress, along with the relatively large quadrilaterally shaped first metatarsal head.[44–46] In toe extension greater than 10°, the sesamoid bones no longer lie in their grooves and may become unstable. Chronic lateral instability of the sesamoid bones may lead to MTP deformity.[46]

FIGURE 12-16

The two sesamoid bones can easily be seen sitting on the head of the first metatarsal.



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Expanded Concepts 12–2

Sesamoiditis

The sesamoid bones and their supporting structures can become traumatized with excessive loading, leading to acute fracture, stress fracture, osteonecrosis, or

chondromalacia. Inflammatory conditions of the sesamoids, which result in pain under the first metatarsal head, are generally known as sesamoiditis.[47] Active individuals who participate in prolonged running, jumping, or gymnastics may be more susceptible, particularly if deformities such as pes cavus (high arch foot) exist.[47–49] Conservative treatment focuses on protecting the region from continued stresses by modifying activity, shoe wear accommodation, and orthotic devices. If a fracture is identified or if the symptoms do not improve, surgical excision of part or all of the sesamoid bones may be necessary.[45,48,50]

Stability of the MTP joints is provided by a joint capsule, **plantar plates, collateral ligaments,** and the deep transverse metatarsal ligament. The plantar plates are structurally similar to the volar plates in the hand. These fibrocartilaginous structures in the four lesser toes are each connected to the base of the proximal phalange distally and blend with the joint capsule proximally. The plates of the four lesser toes are interconnected by the deep transverse metatarsal ligament and **plantar aponeurosis.**[51,52] The collateral ligaments of the MTP joints, like those at the MCP joint, have two components: a phalangeal portion that parallels the metatarsal and phalange, and an accessory component that runs obliquely from the metatarsal head to the plantar plate.[51-53] The plantar plates protect the weightbearing surface of the metatarsal heads and, with the collateral ligaments, contribute to stability of the MTP joints. The plantar plate therefore serves as a central stabilizing structure with a fibrocartilaginous composition that allows it to withstand compressive loads.[51,52] At the first MTP joint, the sesamoid bones and thick plantar capsule are in place of the plantar plates found at the other toes.[44]

Interphalangeal Joints

The interphalangeal (IP) joints of the toes are synovial hinge joints with one degree of

freedom: flexion/extension. The great toe has only one IP joint connecting two phalanges, whereas the four lesser toes have two IP joints (proximal and distal IP joints) connecting three phalanges (see Fig. 12–16). Each phalanx is virtually identical in structure to its counterpart in the hand, although substantially shorter in length. Consequently, the reader is referred to Chapter 9 for details on IP joint structure of the thumb and fingers to understand the structure of the IP joints of the toes. The toes function to smooth the weight shift to the oppesite foot in gait and help maintain stability by pressing against the ground in standing. Injuries to the IP joints are relatively uncommon but may include dislocations and fractures.

Joint Function

The Ankle Joint

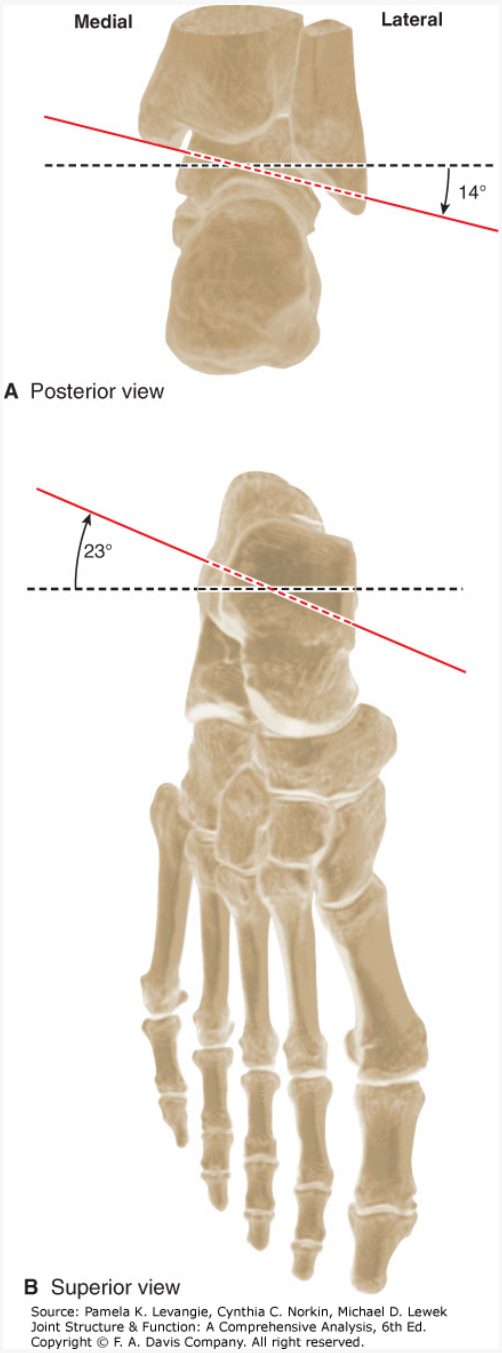
Talocrural Axis

In a neutral ankle position, the joint axis passes approximately through the fibular malleolus and the body of the talus and through or just below the tibial malleolus (Fig. 12–17).[54] The fibular malleolus and its associated fibular facet on the talus are located more distally (Fig. 12–17A) and posteriorly (Fig. 12–17B) than the tibial malleolus and its associated tibial facet. The more posterior position of the fibular malleolus is due to the normal torsion or twist that exists in the distal tibia in relation to the tibia's proximal plateau. This tibial torsion (or tibiofibular torsion, because both the tibia and fibula are involved with the rotation in the transverse plane) accounts for the toe-out position of the foot in normal standing.[55] The torsion in the tibia is similar to the torsion found in the shaft of the femur, although normally reversed in direction. The amount of lateral tibial torsion increases from birth until 10 years of age.[56] As tibial torsion increases, the

axis of the ankle joint is positioned more laterally in the transverse plane. Although variations exist, clinical measures have reported values that approximate 19° of lateral torsion for both adult males and females.[57,58] It should be noted that larger values for tibial torsion have been reported in studies that used computerized tomography.[56]

FIGURE 12-17

The axis of the ankle joint. **A.** Posterior view showing the mortise around the body of the talus and the average 14° inclination of the ankle axis from the transverse plane. **B.** Superior view showing the ankle axis rotated, on average, 23° from the frontal plane.



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The ankle joint classically is considered to have one degree of freedom, with dorsiflexion/plantarflexion occurring between the talus and the mortise. At the ankle, dorsiflexion refers to a motion of the head of the talus (see Fig. 12–6) dorsally (or upward) whereas the body of the talus moves posteriorly in the mortise. Plantarflexion

is the opposite motion of the head and body of the talus. Because of the lower position of the fibular malleolus, the axis of the ankle is declined on the lateral side. A classic anatomical study found this downward angulation to average 14°.[59] However, the amount of angulation may be highly variable between individuals. Stiehl used a simple hinged model with a level indicator to demonstrate how an axis that is oriented more distally and more posteriorly on the lateral side will create a motion across three planes (triplanar motion) while moving around a single fixed axis.[60] He showed that dorsiflexion of the foot around a typically oriented ankle axis will not only bring the foot up (dorsiflexion) but will also simultaneously bring it slightly lateral to the leg (abduction) and appear to turn the foot longitudinally away from the midline (eversion). Conversely, plantarflexion around the same single oblique ankle axis will result in the foot going down, moving medial to the leg (adduction), and appearing to turn the foot longitudinally toward the midline (inversion). When the foot is weightbearing, the same relative pattern of motion exists when the tibia and fibula move on the fixed foot. In weightbearing ankle dorsiflexion, the leg (tibia and fibula) will move forward and lateral to the foot, as well as appear to rotate medially in the transverse plane. The opposite occurs during weightbearing ankle plantarflexion.

It should be noted, however, that although a pure hinge model (e.g., goniometry) helps to explain ankle motion, anatomical studies have demonstrated that the instantaneous axis of rotation for the ankle joint changes throughout the ROM.[54,61–63] Talar rotation within the mortise, in both the transverse plane around a vertical axis (talar rotation or talar abduction/adduction) and in the frontal plane around an A-P axis (talar tilt or talar inversion/eversion), must occur to maintain tibiotalar congruency.[64] These rotations, however, are relatively small in comparison with sagittal plane dorsiflexion and

plantarflexion motion. For example, the literature has reported that only 7° of medial and 10° of lateral talar rotation occur in the transverse plane. Talar tilt in the frontal plane was found to average 5° or less.[65–68] Clearly, the majority, but not all, of ankle motion occurs in the sagittal plane.



Expanded Concepts 12–3

Talocrural Joint Arthroplasty

Ankle replacement (total ankle arthroplasty) is being used more commonly to treat ankle arthritis. First introduced in the 1970s the long-term results of total ankle arthroplasty demonstrated unacceptable failure rates. The use of a hinge type prosthesis with a constrained design and fixed ankle axis was thought to contribute to loosening of some of these first generation prostheses. In the 1990's second generation total ankle replacements were developed. These newer prostheses were designed to better represent normal ankle anatomy and joint kinematics. This included allowing rotation of the talus to occur in multiple planes.[69,70]

A commonly referenced source notes normal ankle joint ROM is 20° for dorsiflexion and 50° for plantarflexion.[71] Large variations in ankle motion can exist between individuals, depending on measurement technique and subject characteristics.[72] Differences in technique include the amount of force used to determine when end ROM occurs and whether or not talocrural joint motion is isolated.[73] Ten degrees of ankle dorsiflexion is considered the minimal amount needed to ambulate without deviation.[74]

During ankle joint dorsiflexion/plantarflexion, the shape of the body of the talus facilitates joint stability. The trochlear (superior) surface of the talus is wider anterior than posterior (see Fig. 12–17B). When the foot is weightbearing, dorsiflexion occurs by the tibia rotating over the fixed talus. As the tibia rotates over the talus, the concave tibiofibular segment slides anteriorly on the trochlear surface of the talus. The wider

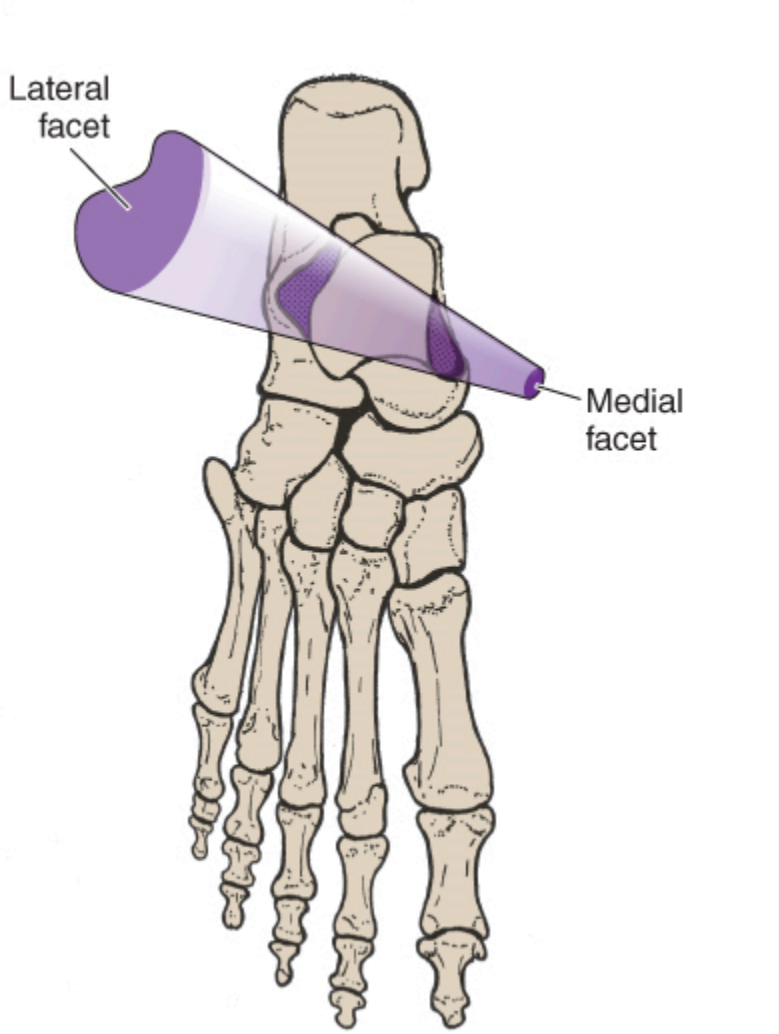
anterior portion of the talus will “wedge” into the mortise, separating the tibia and fibula to enhance stability of the ankle joint. In contrast, the loose packed position of the ankle joint is in plantarflexion, when the narrower posterior body of the talus is in contact with the mortise. That the ankle is less stable in plantarflexion is consistent with the typical mechanism of injury to the lateral ankle ligaments (i.e., plantarflexion and inversion).[14] The ability of the ankle to distribute weightbearing forces combined with the mechanical properties of the tibiotalar articular cartilage reduces the risk of osteoarthritis at the talocrural joint.[69] However, trauma may lead to ankle arthritis not only because of direct cartilage damage but also structural changes that lead to biomechanical abnormalities. Therefore posttraumatic arthritis is the most common type of arthritis occurring at the ankle.[75]

The asymmetry in size and orientation of the lateral and medial facets of the ankle joint contributes to changes in the ankle mortise that occur during ankle dorsiflexion. The lateral (fibular) facet is substantially larger than the medial (tibial) facet, and its surface is oriented slightly obliquely to that of the medial facet (see Fig. 12–17). Inman and Mann proposed that the body of the talus can be thought of as a segment of a cone lying on its side with its base directed laterally.[76] The cone should be visualized as “truncated” or cut off on either end at slightly different angles (**Fig. 12–18**).[55] The asymmetry in size and orientation of the facets means that the distal fibula moving on the larger lateral facet of the talus must undergo a greater displacement (in a slightly different plane) than the tibial malleolus as the tibia and fibular move together during dorsiflexion. The greater arc of motion for the fibular malleolus compared with the tibial malleolus results in superior/inferior motion and medial/lateral rotation of the fibula that requires mobility of the fibula at both the proximal and the distal tibiofibular

joints.

FIGURE 12-18

The three articular surfaces of the talus (the trochlea, smaller medial facet, and larger lateral facet) can be pictured as part of a cone-shaped surface, with ends of the cone cut off (the larger ends of the cone facing laterally).



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Although fibular motion can occur in transverse, sagittal, and frontal planes with ankle plantarflexion and dorsiflexion, the amount of motion seems to be highly variable between individuals.[77] Differences in fibular motion may be related to orientation of the proximal tibiofibular facet, with more mobility available in the facets that are more vertical. Effectively, however, mobility of the fibula at the tibiofibular joints should be considered a component of normal ankle motion.



Expanded Concepts 12–4

Tibiofibular and Ankle Joint Linkage

Some mobility of the fibula appears to be required at the proximal and distal tibiofibular joints to allow the talus to posteriorly rotate fully into the ankle mortise during ankle dorsiflexion. An injury that disrupts the ligaments supporting the distal tibiofibular joint can cause increased motion of the fibula.[78] However, the role of fibular movement in allowing full ankle dorsiflexion remains unclear. Clinically, a limitation in dorsiflexion can be seen after long periods of immobilization or when the distal tibiofibular joint is fixated surgically with open reduction internal fixation (ORIF). Many times this lack of motion is attributed to reduced fibular movement. However, this is not consistent with cadaveric studies that found surgical fixation of the distal tibiofibular joint did not affect dorsiflexion ROM.[79] The functional implications for restricted movement of the fibula (or mortise) are therefore unclear.

Ankle dorsiflexion and plantarflexion movements are limited primarily by soft tissue restrictions. Active or passive tension in the triceps surae (**gastrocnemius** and **soleus muscles**) is the primary limitation to dorsiflexion. Typically, less ankle dorsiflexion ROM is seen with the knee extended compared with a flexed knee position. This can be attributed to the gastrocnemius muscle lengthening across two joints when the knee is extended.[80] Ankle dorsiflexion is limited by the soleus muscle and the posterior talocrural joint capsule when the knee is flexed. Tension in the **tibialis anterior, extensor**

hallucis longus, and **extensor digitorum longus** muscles is the primary limit to plantarflexion. The medial and lateral collateral ankle ligaments are under constant tension during dorsiflexion and plantarflexion, and therefore help to guide the sagittal motion.[81] The more important function of these ligaments appears to be in minimizing side-to-side movement or rotation of the talus in the mortise. The ligaments are assisted in that function by the muscles that pass on either side of the ankle. The **tibialis posterior, flexor hallucis longus,** and **flexor digitorum longus** muscles help protect the medial aspect of the ankle, whereas the **fibularis (peroneus) longus** and **fibularis (peroneus) brevis** muscles protect the lateral aspect. Bony checks of any of the potential ankle motions are rarely encountered unless there is extreme hypermobility (as may be found among gymnasts or dancers) or a failure of one or more of the other restraint systems. A more complete analysis of the function of the muscles crossing the ankle will be presented later as all muscles of the ankle cross at least two and generally three or more joints of the ankle and foot.

The Subtalar Joint

Although the subtalar joint is composed of three articulations, the alternating convex-concave facets limit the potential mobility of the joint. Together, the three surfaces provide a triplanar movement around a single joint axis. When the talus moves on the posterior facet of the calcaneus, the articular surface of the talus should, theoretically, slide in the *same* direction as the bone moves—a concave surface moving on a stable convex surface. However, at the middle and anterior joints, the talar surfaces (again, theoretically) should glide in a direction opposite to movement of the bone—a convex surface moving on a stable concave surface. Motion of the talus on the calcaneus, therefore, is a complex twisting or screw-like motion that can proceed only as long as

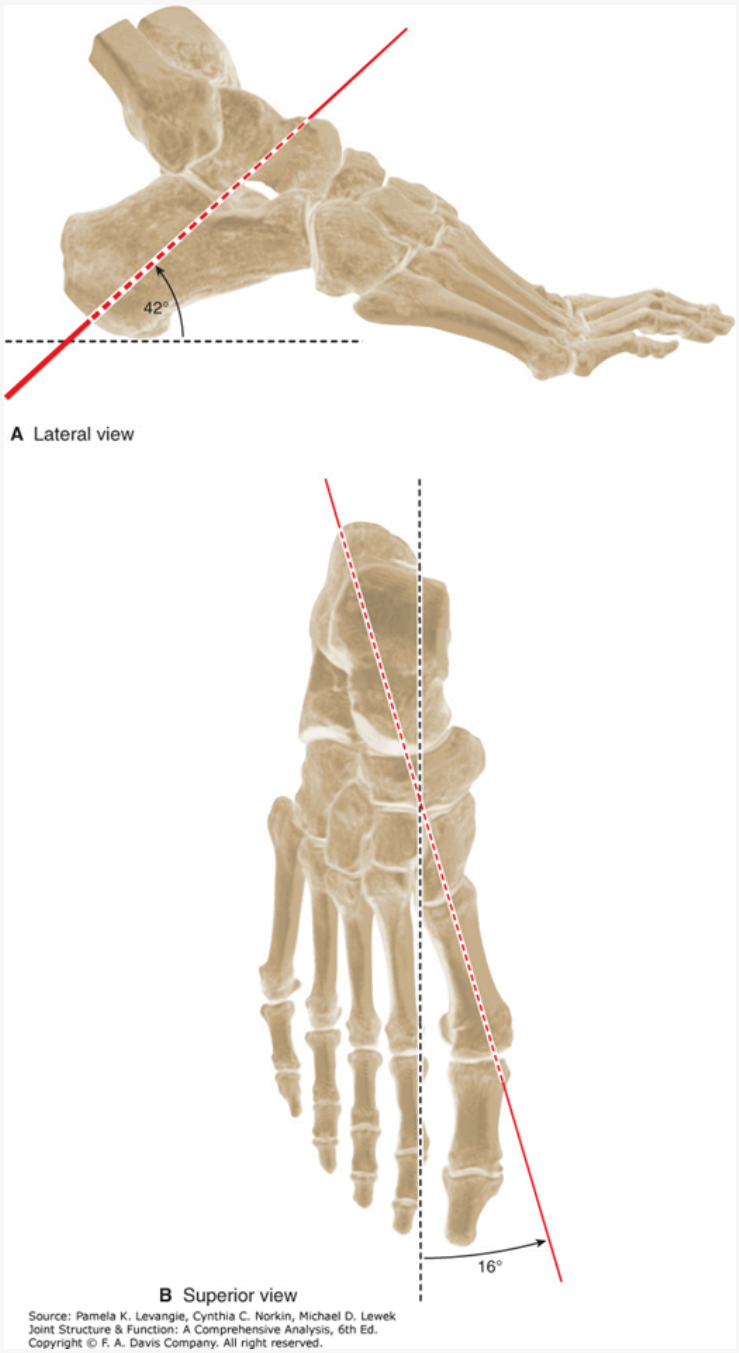
the facets can accommodate simultaneous and opposite motions across the surfaces. The result is a triplanar motion of the talus around a single oblique joint axis, producing the motion of supination/pronation.

The Subtalar Axis

The axis for subtalar supination/pronation has been the subject of many investigations that indicate substantial variability, even among healthy individuals without impairments (Fig. 12–19). Manter reported that the average subtalar axis was (1) inclined 42° upward and anteriorly from the transverse plane (with a broad inter-individual range of 29° to 47°; Fig. 12–19A), and (2) inclined medially 16° from the sagittal plane (with a broad inter-individual range of 8° to 24°; see Fig. 12–19B).[82] Clearly, motion about this oblique axis will cross all three planes. Supination/pronation, like the triplanar ankle joint motion, can be modeled by a single oblique hinge joint.[83] Although the triplanar motions of pronation/supination can be described by its three component (cardinal) motions, these subtalar component motions are coupled and cannot occur independently. The coupled motions must occur simultaneously as the calcaneus (or talus) twists across the subtalar joint's three articular surfaces.

FIGURE 12-19

Axis of the subtalar joint is: **A.** Inclined up from the transverse plane approximately 42°, and **B.** Inclined medially from an A-P axis approximately 16°.



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To understand the components of subtalar pronation/supination, we can consider how the subtalar axis varies from the cardinal axes shown in Figure 12–2. If the subtalar joint axis were vertical, the motion around that axis would produce abduction/adduction; if the subtalar axis were longitudinal, the motion would be inversion/eversion; and if the

subtalar axis were coronal, the motion would be plantarflexion/dorsiflexion. In reality, the subtalar axis lies about halfway between being longitudinal and being vertical. Consequently, pronation/supination includes about equal magnitudes of eversion/inversion and abduction/adduction. The subtalar axis is inclined only very slightly toward being a coronal axis (~16°) and therefore has only a small component of dorsiflexion/plantarflexion. The contribution of each of the coupled movements to supination or pronation will depend greatly on individual differences in inclination of the subtalar axis. As one example, if the subtalar axis is inclined upwardly only 30° (rather than the average of 42°), the relative amount of inversion/eversion will be much greater than the relative amount of adduction/abduction because the axis is closer to being longitudinal. We will now examine how subtalar joint component motions are coupled to create the complex motions of pronation/supination in both non-weightbearing and weightbearing positions.

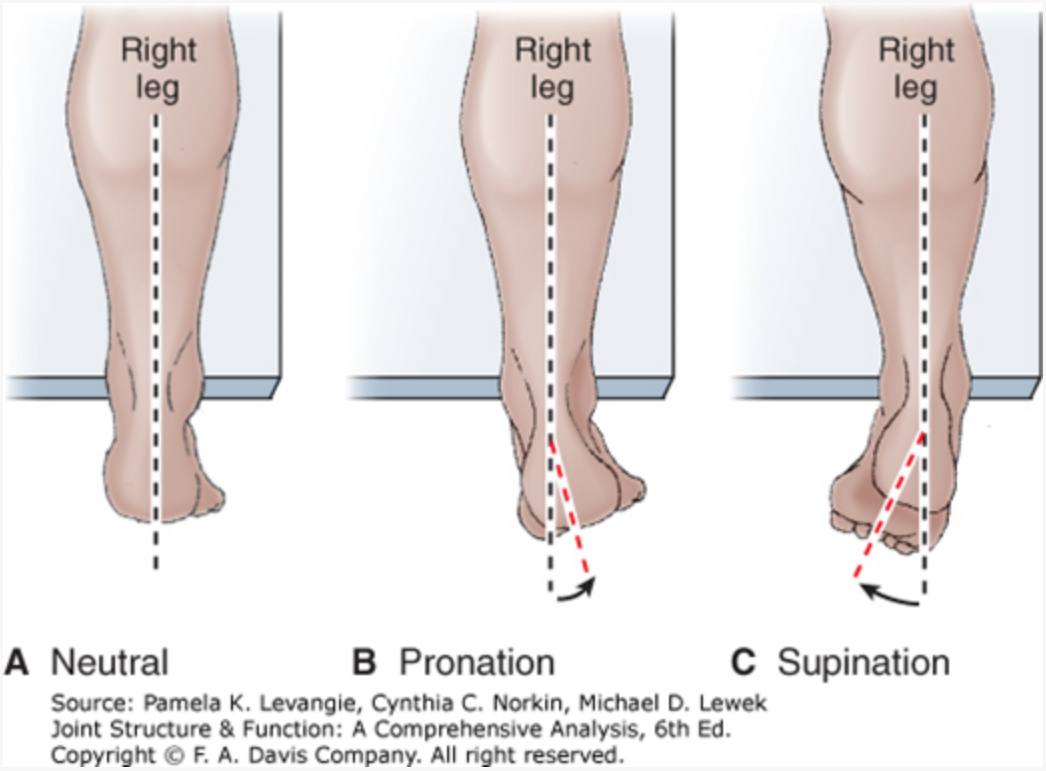
Non-Weightbearing Subtalar Joint Motion

In non-weightbearing supination and pronation, subtalar motion is described by motion of its distal segment (the calcaneus) on the relatively stationary talus and lower leg, where the reference point on the calcaneus is its anteriorly located head (see Fig. 12–6). Non-weightbearing supination is composed of the coupled calcaneal motions of adduction, inversion, and plantarflexion; pronation of the non-weightbearing calcaneus on the fixed talus and lower leg is composed of the coupled motions of abduction, eversion, and dorsiflexion (Table 12–1). The most readily observable of the coupled motions of the calcaneus during pronation and supination are eversion and inversion, respectively (Fig. 12–20). These motions of the calcaneus are often observed at the posterior calcaneus, with the subject prone and the foot and lower leg over the end of

the plinth. Eversion (Fig. 12–20A) may also be referred to as a valgus movement of the calcaneus, and inversion (Fig. 12–20B) as a varus movement of the calcaneus. The eversion and inversion components of pronation/supination appear as if they are occurring in isolation. However, the coupled components of calcaneal abduction and dorsiflexion must simultaneously accompany eversion, and the coupled components of calcaneal adduction and plantarflexion must simultaneously accompany inversion.

FIGURE 12-20

Non-weightbearing motion at the right subtalar joint. **A.** The right rearfoot is shown in neutral. **B.** Pronation of the subtalar joint is observable as eversion (valgus movement) of the calcaneus, although the coupled motions of dorsiflexion and abduction of the calcaneus must also be occurring. **C.** Supination of the subtalar joint is observable as inversion (varus movement) of the calcaneus, although the coupled motions of plantarflexion and adduction of the calcaneus must also be occurring.



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Table 12-1

Summary of Coupled Subtalar Motions: Coupled Movements of Subtalar Pronation/Supination

Foundational Concepts 12–4

Terminology Revisited

Although the apparent interchangeable use of the terms pronation/supination and eversion/inversion to describe the composite uniaxial motions of the subtalar joint appears contradictory, the "disagreement" in terminology is not as discrepant as it first appears. When the composite term "pronation" (a triplanar motion) is used to

describe subtalar motion, the coupled calcaneal component of “eversion” will always be part of the motion. When the component term “eversion” is used to describe subtalar motion, then the other coupled calcaneal components of “pronation” (i.e., abduction, dorsiflexion) will always be part of the motion. Consequently, “pronation” and “eversion” are invariably linked, regardless of the terminology frame of reference. The same is true of supination and inversion; whether used as a composite term or a component term, “supination” and “inversion” are invariably linked. It would certainly be less troublesome if a universal definitional framework were accepted. However, the wary reader who understands the association between supination/pronation and inversion/eversion may be able to infer definitions when they are not overtly offered by authors.

Weightbearing Subtalar Joint Motion

When an individual is weightbearing, the *relative* movements between the talus and calcaneus are the same as during non-weightbearing; however, the description of which segment is moving is different. Typically, during a weightbearing movement, we describe the motion of the proximal segment on the distal segment. At the foot, however, when the calcaneus is on the ground, it is generally still free to move around the longitudinal axis (inversion/eversion) but limited in its ability to move around the coronal axis (plantarflexion/dorsiflexion) and vertical axis (adduction/abduction) because of the superimposed body weight. Consequently, the coupled motions that contribute to pronation/supination cannot be accomplished exclusively by the calcaneus, and instead are described also as movement of the talus on the calcaneus. Although the weightbearing calcaneus will continue to contribute to the inversion/eversion component of subtalar motion, the other two coupled components of the subtalar motion (abduction/adduction and dorsiflexion/plantarflexion) will be accomplished by movement of the talus (whereby the head of the talus is used as the reference) on the more fixed calcaneus rather than by movement of the calcaneus on

the relatively fixed talus. As a reminder, the relative motion accomplished at any joint around a given axis remains unchanged whether the distal segment of the joint moves or whether the proximal segment moves. At the foot, however, the terminology is often altered to describe how a segment moves relative to its original position in space. In this scheme, when the proximal segment moves on the distal segment, the motion of the proximal segment is described as the opposite of what was described as occurring to the distal segment. Specifically, in weightbearing subtalar motion, the direction of the component movement contributed by the talus in space is described as the opposite of what the calcaneus would contribute in space, although the same relative motion occurs between the segments. We recognize that this difference in terminology at the foot can be confusing. For the reader who has become accustomed to thinking about movements as one segment relative to another (e.g., knee flexion represents posterior roll of either the tibia on femur, or femur on tibia), it may become challenging to think about joint motion terminology being used to describe movements of one segment in space (e.g., the talus abducts in space on a relatively fixed calcaneus to produce subtalar joint adduction). Please note whether the terminology references the movement of a segment in space (e.g., talus) or the relative movement at a joint (e.g., subtalar). This will provide important information about what is moving.

In weightbearing supination, the calcaneus continues to contribute the component of inversion. However, the calcaneus cannot adduct and plantarflex in weightbearing, and so the remaining coupled components of subtalar supination are accomplished by the head of the *talus* abducting and dorsiflexing in space. Please note that these descriptions of motion of the talus (talar abduction and dorsiflexion in space) are the same relative movements as the calcaneal adduction and plantarflexion that were

previously discussed in the non-weightbearing scenario. Weightbearing subtalar supination (see Table 12–1), therefore, is observable as inversion (or varus movement) of the calcaneus, whereas the dorsiflexion and abduction of the head of the talus in space are reflected in elevation of the medial longitudinal arch and a convexity on the dorsal lateral midfoot. Although subtalar joint supination is a normal foot motion, a foot that appears fixed in this position often is called a "supinated" or **pes cavus foot.**

Weightbearing subtalar pronation is accomplished by the coupled component movements of eversion of the calcaneus and plantarflexion and adduction of the head of the talus in space (see Table 12–1). In standing, the calcaneus can be observed to move into eversion (or valgus movement), whereas talar adduction and plantarflexion in space are reflected in a lowering of the medial longitudinal arch and a bulging or convexity in the plantar medial midfoot. Although subtalar joint pronation is a normal foot motion, a foot that appears fixed in this position often is called "pronated," **pes planus,** or **flat foot.**

The most critical functions of the foot occur in weight-bearing. When the foot is weightbearing and the head remains relatively positioned over one or both feet, the joints of the lower extremity effectively form a closed chain. Consequently, the kinematics and kinetics of the subtalar joint will affect and be affected by more proximal and distal joints. An important consequence of closed-chain subtalar function can be seen in its interdependence with lower extremity or leg rotation.

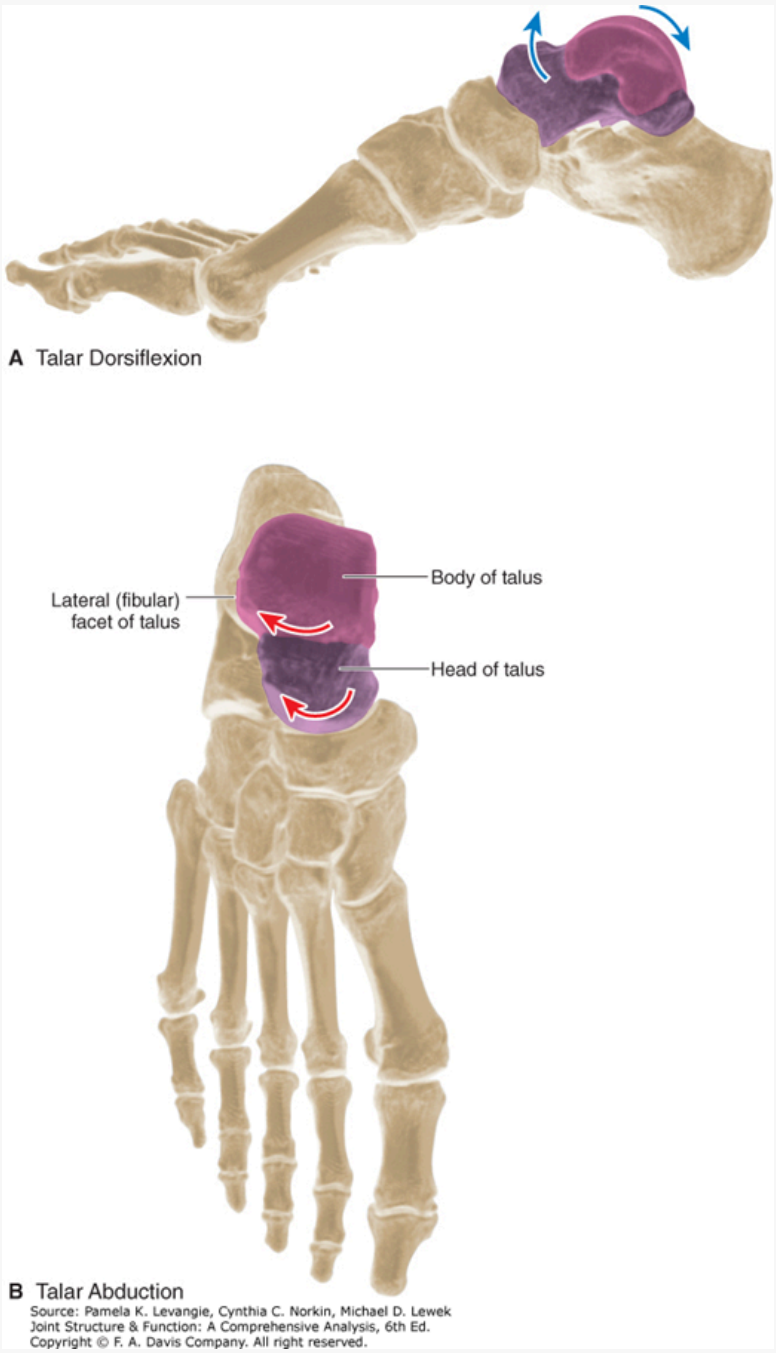
WEIGHTBEARING SUBTALAR JOINT MOTION AND ITS EFFECT ON THE LEG

During weightbearing subtalar supination/pronation, the coupled component motions of dorsiflexion/plantarflexion and abduction/adduction of the talar head in space

require that the body of the talus move as well (Fig. 12–21). The body of the talus is, of course, lodged within the superimposed mortise. Dorsiflexion of the head of the talus requires the body of the talus to slide posteriorly within the mortise (Fig. 12–21A), whereas plantarflexion of the head of the talus requires the body of the talus to move anteriorly within the mortise. The tibia (leg) remains unaffected by the talar dorsiflexion/plantarflexion movement as long as the ankle joint is free to move. However, the ankle joint cannot absorb the coupled component motions of talar abduction/adduction without affecting the leg. For this reason, the tibia can be observed to medially/laterally rotate on the foot during weightbearing adduction/abduction of the talus in space.

FIGURE 12-21

A. Dorsiflexion of the head of the talus during weightbearing subtalar supination slides the body of the talus posteriorly within the tibiofibular mortise. **B.** Abduction of the head of the talus during weightbearing subtalar supination rotates the body of the talus laterally, potentially taking the tibiofibular mortise along with it.



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When the head of the talus abducts in space in weightbearing subtalar supination, the body of the talus must rotate laterally in the transverse plane (see Fig. 12–21B). When the head of the talus adducts in weightbearing subtalar pronation, the body of the talus must rotate medially in the transverse plane. Because the body of the talus can rotate

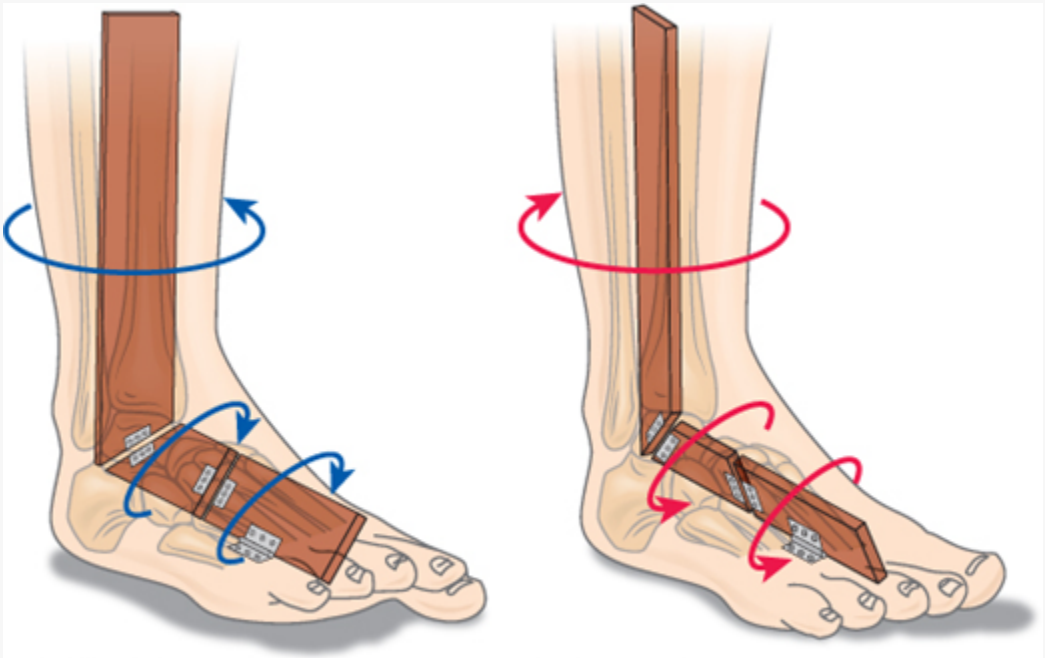
only minimally at most *within* the mortise, rotation of the body of the talus can occur in weightbearing only if the superimposed mortise moves *with* the talus. When the subtalar joint supinates in a weightbearing position, the coupled component of talar abduction rotates the mortise (the tibia and fibula) laterally, producing lateral rotation of the leg. Correspondingly, weightbearing subtalar joint pronation consists of talar adduction, with the body of the talus rotating medially and carrying the superimposed tibia and fibula into medial rotation.

Through the component movements of abduction and adduction of the talus, weightbearing subtalar joint motion directly influences the segments and joints superior to it. A weightbearing subtalar joint maintained in pronation (e.g., a flat foot) can create a medial rotation force on the leg that may influence the knee and hip joints. Just as subtalar pronation and supination may impose rotatory forces on the leg in weight-bearing, so too may rotations of the leg influence the subtalar joint. When a lateral rotatory force is imposed on the weight-bearing leg (as when you rotate to the right around a planted right foot), the lateral motion of the leg carries the mortise and its mated body of the talus laterally. Lateral rotation of the body of the talus (abduction of the head of the talus) cannot occur without its coupled components of talar dorsiflexion and calcaneal inversion, which produce supination of the subtalar joint. A medial rotatory force imposed on the weightbearing leg will necessarily result in subtalar pronation as the talus is medially rotated (adducted) by the rotating tibiofibular mortise and carries with it the coupled components of talar plantarflexion and calcaneal eversion. The interdependence of the leg and talus were mechanically represented by Inman and Mann, who used the concept of the subtalar joint as a mitered hinge.[76] This mitered hinge concept (Fig. 12–22) presents a good visualization

of the concept of the interdependence of the leg and foot through the oblique subtalar axis.

FIGURE 12-22

The subtalar joint can be visualized as a mitered hinge between the leg and the foot. **A.** Medial rotation of the weightbearing leg imposes pronation on the distally located subtalar joint. **B.** Lateral rotation of the leg proximally imposes supination on the distally located subtalar joint.



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This kinematic coupling is thought to play an important role in lower extremity injuries when abnormalities exist.[84] Although this hinge model helps to understand movement between the subtalar joint and leg, in vivo studies have found a great deal of individual

variability in movement coupling.[85–87]

Patient Application 12–1

Arnold Benson is a 63-year-old man seeking intervention for right foot pain. Mr. Benson says he has "flat feet," which seem to have worsened over the past few years. When the foot is maintained in a more pronated position during weightbearing, as is true for Mr. Benson, there can be a sustained medial rotation force on the lower extremity. That force may cause medial rotation at the knee and hip. However, it may be difficult to determine if subtalar pronation is causing the medial rotation of the lower extremity or if the pronation is a result of top down proximal forces from the hip or knee.

Range of Subtalar Motion and Subtalar Neutral

The range of subtalar supination and pronation is difficult to determine objectively because of the triplanar nature of the movement and because the component contributions vary with the inclination of the subtalar axis. The calcaneal inversion/eversion (varus/valgus) component of subtalar motion can be measured in both weightbearing and non-weightbearing positions by using the posterior calcaneus and posterior midline of the leg as reference lines. This technique assumes that the neutral position (0°) is where the two posterior lines align to form a straight line (see Fig. 12–20). For individuals without impairments, 5° to 10° of calcaneal eversion (valgus) and 20° to 30° of calcaneal inversion (varus) have been reported, for a total range of 25° to 40°.[88–90] This measurement technique is problematic, however, because isolating subtalar motion from tibiotalar motion is clinically difficult and as a result, this measurement technique may have problems related to reliability.[91–97] Although the range of calcaneal inversion/eversion is not equivalent in magnitude to those of subtalar supination/pronation, the ranges should be directly proportional.

The variability in the inclination of the subtalar axis described by Manter directly affects the range of the coupled components of subtalar motion.[82] If the axis is inclined upward less than the average of 42° (see Fig. 12–19A), the subtalar axis will more closely approximate a longitudinal axis down the length of the foot; the proportion of inversion/eversion of the calcaneus that is part of subtalar motion will increase, whereas the proportion of coupled abduction/adduction of the calcaneus (or talus) will decrease. Because the change in inclination of the subtalar axis will affect both the foot and leg position in weightbearing, a considerable amount of attention has been given to determining how an individual's subtalar axis might differ from the average (or standard) axis. This has led to an attempt to define an individual's **subtalar neutral** position of the subtalar joint and use this as a reference for a "normal" or "ideal" foot position. An individual's neutral subtalar joint position may deviate from the point at which the midlines of the posterior calcaneus and the posterior leg coincide, however, with a medial increase in that angle referred to as valgus and a decrease referred to as varus (see Fig. 12–3).



Expanded Concepts 12–5

Subtalar Neutral

The subtalar neutral position has been defined differently by various investigators, with some issues raised as to the appropriateness of the concept or the measurement techniques. Root and colleagues defined subtalar neutral position as the point from which the calcaneus will invert twice as many degrees as it will evert.[89] Bailey and colleagues used radiographic evidence to demonstrate that the neutral position of the subtalar joint was not always found two-thirds of the way from maximum supination, although the average neutral subtalar position for their subjects was close to this value.[98] Elveru and colleagues proposed palpating the medial head and neck of the talus while supinating and pronating the subtalar joint, with subtalar neutral as the point where the talus is equally positioned between the

fingers.[93] This technique is fairly subjective, and the inter-rater reliability is generally poor.[93,99] Only one study has noted acceptable reliability when standardized methods and practice were employed as part of the methodology.[100] This study, however, has not been replicated.

The work of Cornwall and McPoil can be used to highlight some of the controversy that exists around the definition and application of the term **subtalar neutral.**[101] Cornwall and McPoil described calcaneal motion during walking in 153 subjects between 18 and 41 years old with no history of foot impairments. They reported that the calcaneus is inverted 3.0° (±2.7°) at heel strike relative to the tibia, and then everts 2.2° (±2.4°) by 55% of stance phase. After a period of eversion, the motion is reversed to 5.5° (±3.2°) of inversion just before the foot leaves the ground.[101] When looking at the overall results of the study it was apparent the subtalar joint does not function about a neutral position during walking but actually functions more in an everted position.[101] This finding was in agreement with their previous work.[102–104] They have concluded that the "neutral" position of the rear foot during the walking cycle was better represented by the resting position of the calcaneus with respect to the lower leg when weightbearing as opposed to the palpated subtalar joint neutral position. The resting position of the calcaneus in relaxed bilateral stance averaged approximately 3.5° of calcaneal valgus angle (eversion) in a group of their subjects.[102] These findings appear to support the conclusion of Åstrom and Arvidson, who noted that the normal weightbearing foot is more pronated than previously thought and that reliance on the palpated subtalar neutral position could lead to overdiagnosis of excessive subtalar pronation.[90] It should also be acknowledged that very few individuals may actually have an "ideal" foot position, and clinical examination may not be able to predict dynamic foot function.[105–107]

The calcaneal inversion/eversion components of subtalar motion have received a large amount of attention because of the relative ease of measurement strategies. The talar dorsiflexion/plantarflexion and abduction/adduction components of weightbearing subtalar motion are a more difficult measure. Estimates of talar abduction/adduction have been made by measuring the tibial rotation that accompanies abduction/adduction of the talus in weightbearing.[101] In vivo measures of subtalar motion during walking using bone anchored markers have been made. A recent study found total talocalcaneal joint motion during walking averaged 6.8°, 9.8°, and 7.5° in the sagittal, frontal, and transverse planes, respectively.[86] It should be noted that this study also found a large variation in motion between subjects.[86]



Foundational Concepts 12–5

Subtalar Joint Neutral Summary

Morton Root, a podiatrist, is credited with describing the theoretical management approach to foot and ankle problems that focused on the subtalar joint neutral position.[89] A basic premise of the approach is that the subtalar joint should be in a neutral position during midstance. The approach to intervention for several foot deformities includes use of an orthotic device to "balance the foot" and achieve a defined subtalar joint neutral position during midstance. However, as outlined, there are a number of problems with this approach. These problems include the subtalar joint not actually being in neutral at midstance, poor reliability of measures, and inability to predict dynamic function from static measures. Additionally, although the subtalar joint position may contribute to our understanding of foot structure and function, the influence of subtalar position must be considered with other interdependent factors, including bony abnormalites (i.e., femoral or tibial rotation); extrinsic factors such as footwear, running surface, and activity level (magnitude and change); and physiological factors such as obesity or disease.[1,108]

When the subtalar joint is non-weightbearing, the motions of the subtalar joint and the

leg are independent and do not influence each other. When the foot is weightbearing, a primary function of the subtalar joint is to absorb the imposed lower extremity transverse plane rotations that occur during walking and other weightbearing activities. Such rotations would otherwise spin the foot on the ground or disrupt the ankle joint by rotating the talus within the mortise. In supination, ligamentous tension draws the subtalar joint surfaces together, which results in locking (close-packing) of the articular surfaces. Conversely, the adduction and plantarflexion of the talus that occur in weight-bearing pronation cause a splaying (spreading) of the adjacent tarsal bones that permits some intertarsal mobility (i.e., loose packed position). The role of the ligaments in contributing to mobility or stability at the subtalar joint, however, is somewhat controversial.[29] Sarrafian believed that the position of the cervical and interosseous talocalcaneal ligaments are along the subtalar axis, which causes these structures to remain tight in pronation and supination.[88] According to this premise, individual shifts in location of the axis or of the ligaments could account for discrepant findings of other investigators.

The subtalar joint is strategically located between the ankle joint proximally and the transverse tarsal joint distally. We have already discussed how motions at the subtalar joint are associated with motions of the leg and ankle joint in weightbearing. We will now focus our attention on motion between the talus and the navicular bone and between the calcaneus and cuboid bones. These articulations are grouped differently according to various authors, but the approach of this chapter will focus on the **transverse tarsal joint** as the primary functional unit accounting for motion in the midfoot. We will also see how motion at the subtalar joint influences motion at the transverse tarsal joint.

Transverse Tarsal Joint

The two joints formed by the talonavicular and calcaneocuboid together present an S-shaped joint line that transects the foot, dividing the rearfoot from the midfoot and forefoot. The navicular and the cuboid bones are considered, in essence, immobile in the weightbearing foot. Transverse tarsal joint motion, therefore, is considered to be motion of the talus and of the calcaneus on the relatively fixed naviculocuboid unit. Motion at the compound transverse tarsal joint, however, is more complex than the relatively simple joint line might suggest, and occurs synchronously with motion at the subtalar joint.[35]

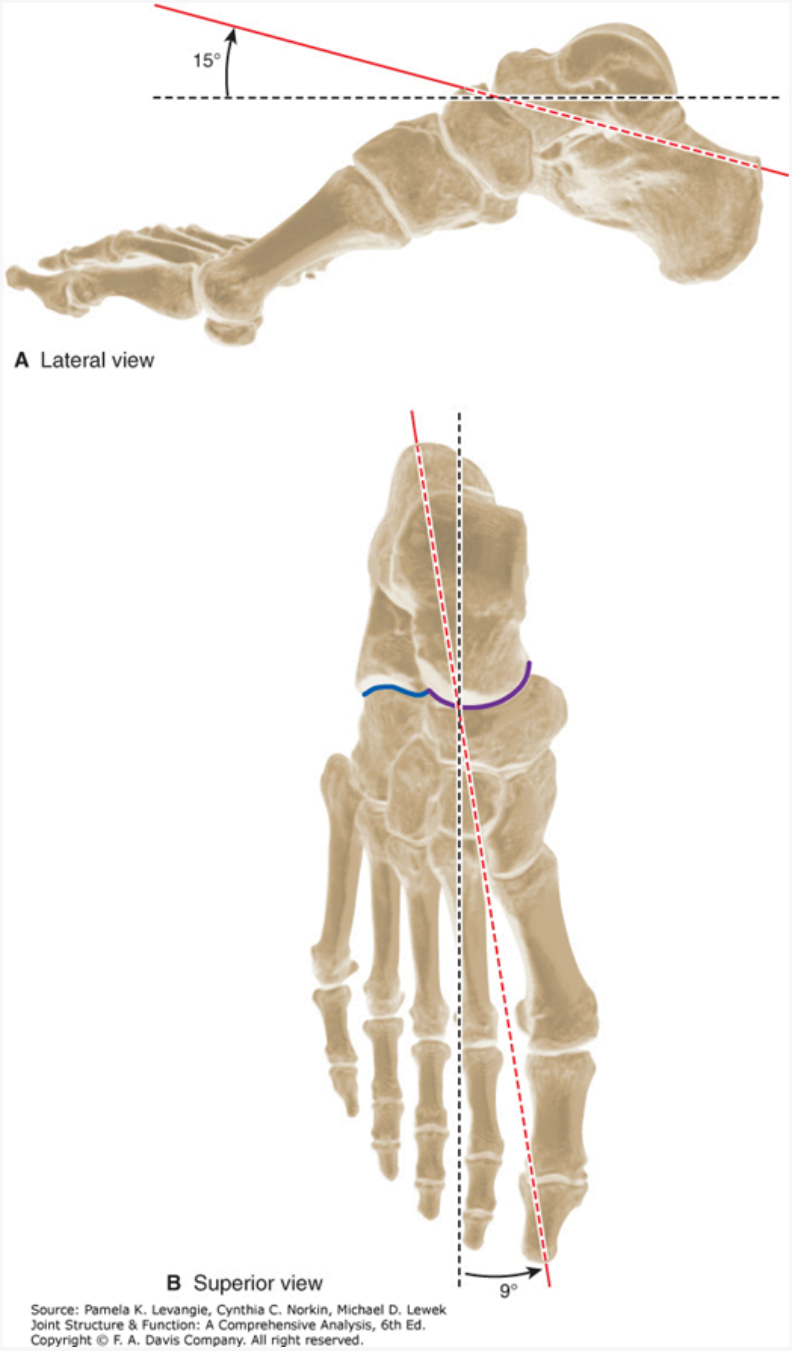
Transverse Tarsal Joint Axes

Movements at the transverse tarsal joint are more difficult to study than movement at the ankle or subtalar joint, because multiple segments and axes are involved. Although the talonavicular and calcaneocuboid joints have some independent movement, motion at one is generally accompanied by at least some motion of the other because of their functional, bony, and ligamentous connections. We continue to rely on the classic works of Elftman, Manter, and Hicks, who proposed longitudinal and oblique axes around which the talus and calcaneus move on the relatively fixed naviculocuboid unit (Fig. 12–23).[59,82,109] The longitudinal axis is nearly horizontal, being inclined 15° upward from the transverse plane (Fig. 12–23A) and angled 9° medially from the sagittal plane[48] (see Fig. 12–23B). This orientation of the longitudinal axis of the transverse tarsal joint produces primarily inversion/eversion movements. Nevertheless, motion around this axis is triplanar, producing supination/pronation with coupled components (small amounts of dorsiflexion/plantarflexion and abduction/adduction) similar to those seen at the subtalar joint but now simultaneously including both the talus and

calcaneus segments moving on the navicular and cuboid segments.

FIGURE 12-23

The longitudinal axis of the transverse tarsal joint is: **A.** Inclined 15° superiorly from the transverse plane, and **B.** Inclined 9° medially from the sagittal plane.

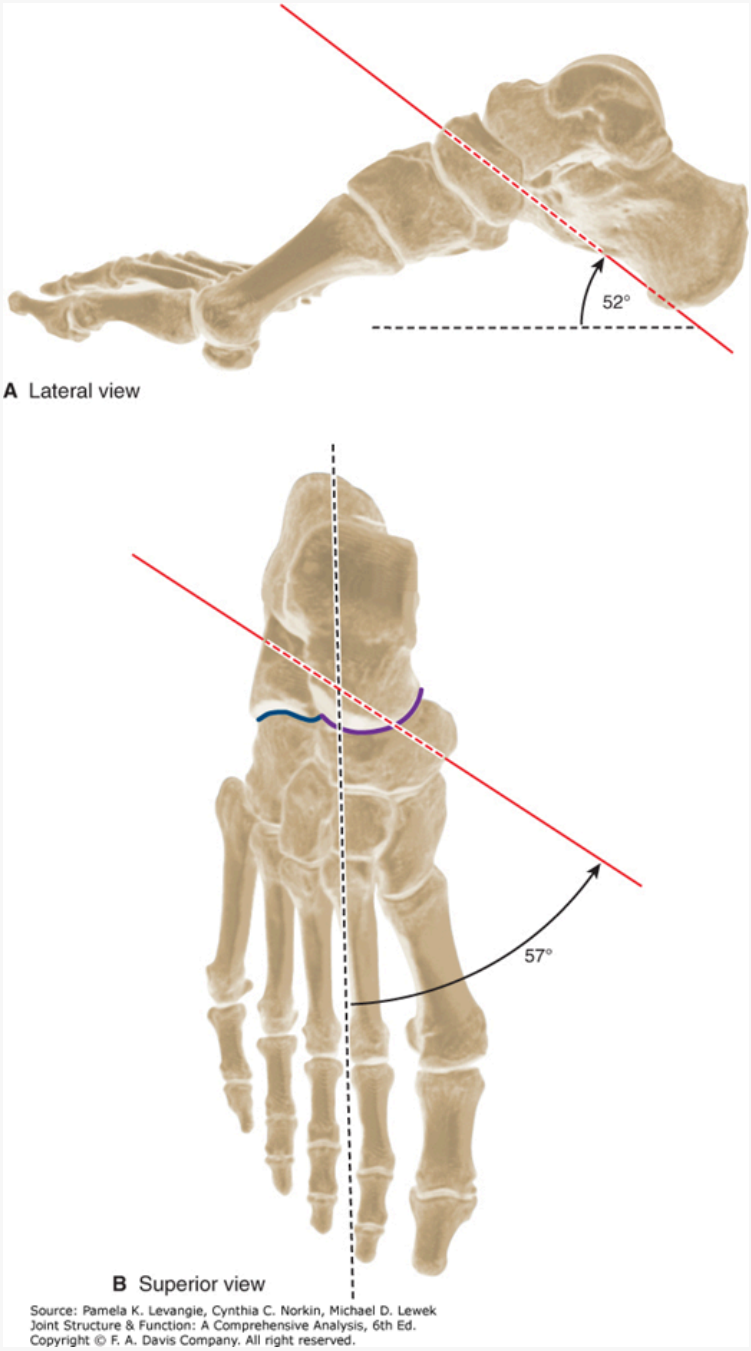


The oblique (transverse) axis of the transverse tarsal joint also contributes to the movements observed at the transverse tarsal joint (Fig. 12–24). This axis is positioned approximately 57° medial to the sagittal plane (Fig. 12–24A) and 52° superior to the transverse plane (see Fig. 12–24B).[82] As a triplanar axis, the oblique axis also provides

supination/pronation with coupled component movements of the talus and calcaneus segments moving together on the navicular and cuboid bones, but dorsiflexion/plantarflexion and abduction/adduction components predominate over inversion/eversion motions. Motions about the longitudinal and oblique axes are difficult to separate and quantify. The longitudinal and oblique axes together provide a total range of supination/pronation of the talus and calcaneus that is about one-third to one-half of the range available at the subtalar joint.[59]

FIGURE 12-24

The oblique axis of the transverse tarsal joint is: **A.** Inclined 52° superiorly from the transverse plane and **B.** Inclined 57° from the sagittal plane.



The proposed longitudinal and oblique axes for the transverse tarsal joint indicate a function similar to that of the subtalar joint. In fact, as already noted, the subtalar and transverse tarsal joints are linked mechanically so that any weightbearing subtalar motion causes the talonavicular and calcaneocuboid joint to move simultaneously as

well. As the subtalar joint supinates, its linkage to the transverse tarsal joint causes the talonavicular and calcaneocuboid joints to begin to supinate. When the subtalar joint is fully supinated and locked (bony surfaces are drawn together), the transverse tarsal joint is also carried into full supination, and its bony surfaces are similarly drawn together into a locked position. When the subtalar joint is pronated and loose-packed, the transverse tarsal joint is also mobile and loose-packed.

The transverse tarsal joint is the transitional link between the rearfoot and the forefoot, serving to (1) add to the supination/pronation range of the subtalar joint and (2) compensate the forefoot for rearfoot position. Compensation in this context refers to the ability of the forefoot to remain flat on the ground (relatively immobile) while the rearfoot (talus and calcaneus) pronates or supinates in response to the terrain or the rotations imposed by the leg. The first of the transverse tarsal joint functions (adding range to supination/pronation) can occur either in the weightbearing foot or in the non-weightbearing foot. The second function requires closer analysis.

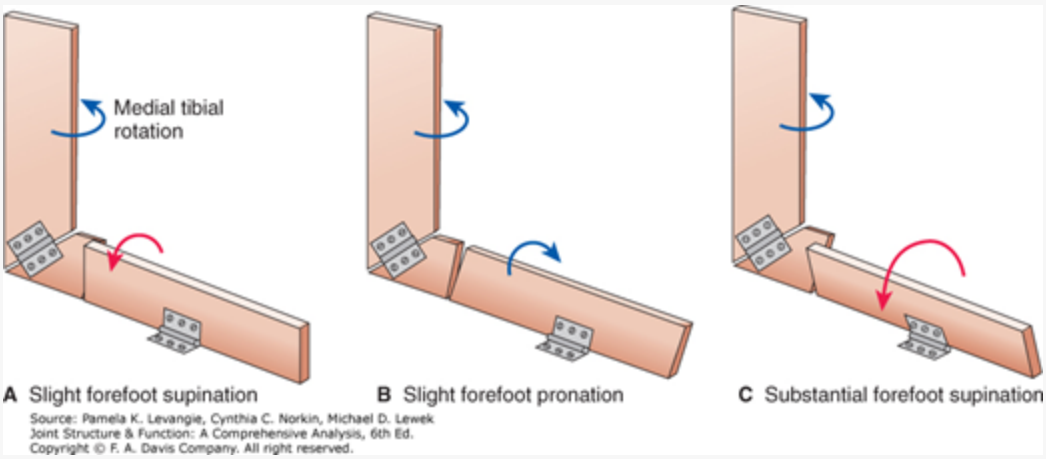
Weightbearing Rearfoot Pronation and Transverse Tarsal Joint Motion

In the weightbearing position, medial rotation of the tibia (as occurs, for example, if someone pivots on a fixed foot) imposes pronation on the subtalar joint. If the pronation force continued distally through the foot, the lateral border of the foot would tend to lift from the ground, diminishing the stability of the base of support, resulting in unequal weightbearing, and imposing stress at multiple joints. This undesirable effect of weightbearing subtalar joint pronation may be avoided if the forefoot remains flat on the ground. This can occur if the transverse tarsal joint is mobile and can effectively “absorb” the rearfoot pronation (allowing the rearfoot to move without passing the

movement on to the forefoot). When the talus and calcaneus move on an essentially fixed naviculocuboid unit, there is a relative supination of the bony segments distal to the transverse tarsal joint (imposed by the rearfoot pronation), with the result that the forefoot remains relatively flat on the ground. The transverse tarsal joint maintains normal weightbearing forces on the forefoot while allowing the rearfoot (subtalar joint) to absorb the rotation of the lower limb. Inman and Mann's mechanical model (Fig. 12–25) nicely represents how a medial rotatory force imposed on the leg acts through the oblique axis of the *subtalar joint* and through the *transverse tarsal joint* to maintain the forefoot in a relatively fixed position.[76] Note that the fixed *forefoot* has effectively moved in a direction opposite to that of the *rearfoot* segment.

FIGURE 12-25

With pronation occurring at the subtalar joint through medial rotation of the leg, the transverse tarsal joint is free to: **A.** Supinate slightly to maintain the relatively fixed position of the forefoot segment; **B.** Pronate slightly as occurs in normal standing; or **C.** Supinate substantially to maintain appropriate weightbearing of the forefoot segment on uneven terrain.



Inman and Mann's model indicates that when the weight-bearing rearfoot (subtalar joint) is pronated, the transverse tarsal joint will supinate (move in a direction opposite to the rearfoot).[76] However, in reality the transverse tarsal joint is relatively free to move either into pronation or supination (depending on the demands of the terrain). In a bilateral standing position on level ground, both the subtalar joint and the transverse tarsal joints pronate slightly (see Fig. 12–25B), presumably to allow the foot to absorb the body's weight. As a result of the pronation, there will be a slight medial rotatory force on the leg. As a person moves into single-limb support and begins to walk, the subtalar joint will continue to pronate, but because the forefoot must remain flat on the floor, the transverse tarsal joint will assume an equal amount of relative supination to maintain proper weightbearing in the forefoot. As a reminder, the relative supination at the transverse tarsal joint in this example arises from the movement of the talus and the calcaneus (rearfoot).

During walking on uneven terrain, as long as the rearfoot is in pronation, the forefoot can move toward either supination or pronation, depending on the demands of the terrain. If, for example, there is a rock under the medial forefoot during walking, the

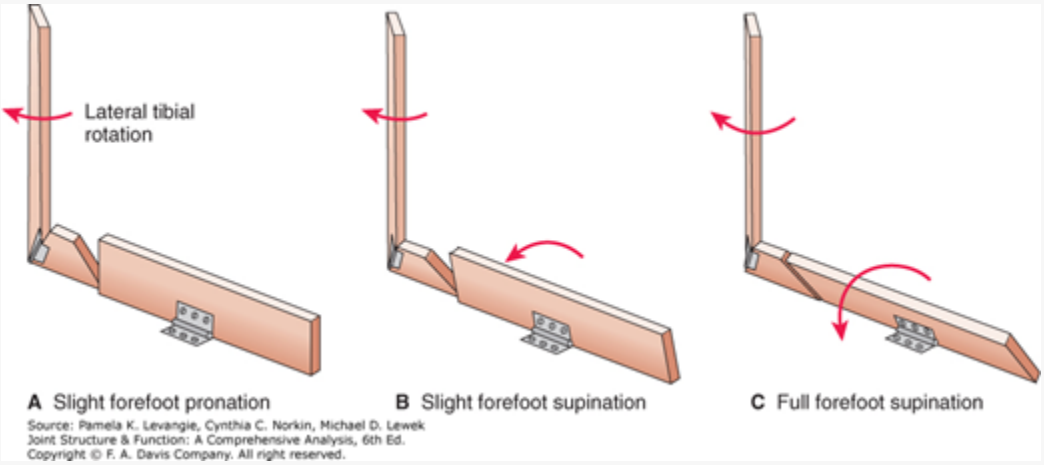
transverse tarsal joint may move into greater supination to maintain appropriate contact of the forefoot with the ground (see Fig. 12–25C). If the supination range is not available at the transverse tarsal joint, the rock may also force the rearfoot into a supinated position (putting the LCLs at risk for injury). With other surface demands, such as standing sideways on a steep hill, the uphill foot must pronate substantially to maintain contact with the ground. Therefore, pronation may be required at the subtalar and the transverse tarsal joints. As long as the subtalar joint is in some degree of pronation, the subtalar and transverse tarsal joints are relatively mobile and free to make compensatory changes (within the limits of the joints' ROM) to maintain contact of the foot with the ground.

Weightbearing Rearfoot Supination and Transverse Tarsal Joint Motion

As shown in the mechanical model of Inman and Mann, a lateral rotatory force on the leg will create subtalar supination in the weightbearing subtalar joint, with a relative pronation of the transverse tarsal joint (opposite motion of the forefoot segment) to maintain appropriate weightbearing on a level surface (Fig. 12–26).[76] Supination of the subtalar joint, however, can proceed to only a certain point before the transverse tarsal joint also begins to supinate. As bony and ligamentous structures of the subtalar joint draw the talus and calcaneus closer together (become increasingly close-packed), the navicular and cuboid bones are also drawn toward the talus and calcaneus; that is, transverse tarsal joint mobility is increasingly limited as the subtalar joint moves toward full supination. With increasing supination of the subtalar joint (caused either by the terrain or by an increased lateral rotatory force on the leg), the transverse tarsal joint cannot absorb the additional rotation but begins to move toward supination as well (see Fig. 12–26B).

FIGURE 12-26

A. With supination occurring at the subtalar joint through lateral rotation of the leg, the transverse tarsal joint has limited ability to pronate to maintain the relatively fixed position of the forefoot segment. **B.** The transverse tarsal joint will begin to supinate with a greater range of subtalar supination and lateral rotation of the leg or will **C.** Fully supinate along with a fully supinated subtalar joint and maximal lateral rotation of the superimposed leg.



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In full subtalar joint supination, such as when the tibia is maximally laterally rotated on the weightbearing foot, supination locks not only the subtalar joint but also the transverse tarsal joint (see Fig. 12–26C). The fully supinated subtalar joint and transverse tarsal joint will tend to shift the weightbearing in the forefoot fully to the lateral border of the foot. Barring other compensatory mechanisms or when the demands of the terrain exceed the foot's ability to compensate, the entire medial border of the foot may lift and, unless the muscles on the lateral side of the foot and ankle are active, a supination (often called *inversion*) sprain of the lateral ligaments

may occur. When the locked subtalar and transverse tarsal joints are unable to absorb the rotation superimposed by the weightbearing limb or by uneven ground, the forces must be dissipated at the ankle, and excessive stresses may result in injury to the ankle joint structures. The subtalar joint of a high-arched (pes cavus) foot tends to be set in a supinated position with limited pronation motion. This supinated position also limits the ability of the transverse tarsal joint to compensate. Therefore, a high-arched foot is relatively more rigid and may be more susceptible to impact-type injuries, especially on the lateral side of the foot.[110,111]



Patient Application 12–2

Jackie Montgomery is a 65-year old woman who is complaining of right foot pain and reports that she did not always have "flat feet." Mrs. Montgomery is likely to have adult-acquired flat foot, which is defined as a progressive loss of dynamic and static medial longitudinal arch support.[112] In a flat foot (pes planus or pes valgus) deformity, the foot typically remains in a position of excessive pronation at the subtalar joint during weightbearing. Posterior tibial tendon dysfunction is usually associated with adult-acquired flat foot. Mrs. Montgomery complains of pain with active plantarflexion and inversion. As the posterior tibialis muscle loses its function, increased stress is placed on the other supporting structures of the medial longitudinal arch. This results in progressive flattening of the foot with decreased medial arch height, plantar medial rotation of the talus, hind foot valgus, and forefoot abduction. The lateral malleolus can be positioned more anteriorly than normal, resulting from medial rotation of the lower extremity. Also, there is increased loading on the first metatarsal and increased stress on the ligaments supporting the joints of the medial arch.[113–116] The deformities associated with adult-acquired flat foot were found to impact gait as stride length, cadence, and walking speed were all diminished, whereas stance duration was prolonged.[117] A classification scheme and treatment guidelines for adult-acquired flat foot are available based on the severity of the deformity.[118] This includes assessing whether the deformities are flexible or fixed. Mrs. Montgomery's deformities were able to be passively corrected to neutral and therefore defined as flexible. A number of risk factors for developing adult-

acquired flat foot have been identified. Of these, obesity seems to be the most significant factor.[119] An orthosis and exercises can be used to effectively treat early stages when the deformities are flexible.[120] However, surgical intervention may be indicated when the “flat-feet” become rigid and the deformities cannot be passively corrected to neutral.[121]

Tarsometatarsal Joints

Tarsometatarsal Axes

Each TMT joint is considered to have a unique, although not fully independent, axis of motion. Hicks examined the axes for the five rays.[59] A ray is defined as a functional unit formed by a metatarsal and (for the first through third rays) its associated cuneiform bone. The cuneiform bones are included as parts of the movement units of the TMT rays because of the small and relatively insignificant amount of motion occurring at the cuneonavicular joints. The cuneonavicular motion, therefore, becomes functionally part of the available TMT motions. The fourth and fifth rays are formed by the metatarsal alone because these metatarsals share an articulation with the cuboid bone.

According to Hicks, the first and fifth rays exhibit the most motion of the TMT joints.[59] The axes for the first and fifth rays are shown in Figure 12–15. Each axis is oblique and, therefore, triplanar. Of the TMT joints, the first has the largest ROM. The axis of the first ray is inclined so that dorsiflexion of the first ray also includes inversion and adduction, whereas plantarflexion is accompanied by eversion and abduction. The abduction/adduction components normally are minimal. Movements of the fifth ray around its axis are more restricted and occur with the opposite arrangement of components: dorsiflexion is accompanied by eversion and abduction, and plantarflexion is accompanied by inversion and adduction.

The axis for the third ray nearly coincides with a coronal axis; the predominant motion, therefore, is dorsiflexion/plantarflexion. The axes for the second and fourth rays were not determined by Hicks but were considered to be intermediate between the adjacent axes for the first and fifth rays, respectively.[59] The second ray moves around an axis that is inclined toward, but is not as oblique as, the first axis. The fourth ray moves around an axis that is similar to, but not as steep as, the fifth axis. The second ray is considered to be the least mobile of the five.

The motions of the TMT joints are interdependent, as are the motions of the CMC joints in the hand. Like the CMC joints of the hand, the TMT joints contribute to hollowing and flattening of the foot. In contrast to the hand, however, the greatest relevance of TMT joint motions is found during weightbearing. In weightbearing, the TMT joints function primarily to augment the function of the transverse tarsal joint; that is, the TMT joints attempt to regulate position of the metatarsals and phalanges (the forefoot) in relation to the weightbearing surface.[122] As long as transverse tarsal joint motion is adequate to compensate for the rearfoot position, considerable TMT joint motion is not required. However, when the rearfoot position is at an end point in its available ROM or the transverse tarsal joint is inadequate to provide full compensation, the TMT joints may rotate to provide further adjustment of forefoot position.[89]

Supination Twist

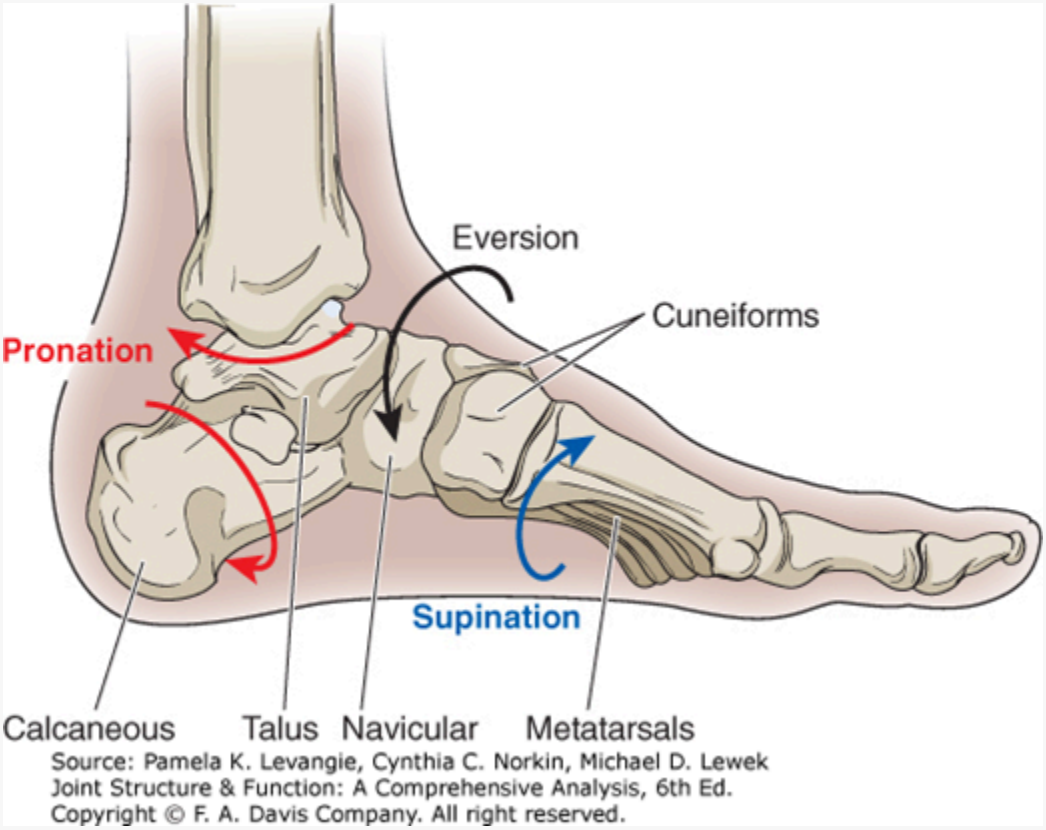
When the rearfoot pronates substantially in weightbearing, the transverse tarsal joint generally will supinate to some degree to counter-rotate the forefoot and keep the plantar aspect of the foot in contact with the ground. If the range of transverse tarsal supination is not sufficient to meet the demands of the pronating rearfoot (or if the

transverse tarsal joint is prevented from effectively serving this function), the medial forefoot will press into the ground, and the lateral forefoot will tend to lift. The first and second ray will be pushed into dorsiflexion by the ground reaction force, and the muscles controlling the fourth and fifth rays will plantarflex the TMT joints in an attempt to maintain contact with the ground. Both dorsiflexion of the first and second rays and plantarflexion of the fourth and fifth rays include the component motion of inversion of the ray. Consequently, the entire forefoot (each ray and its associated toe) undergoes an inversion rotation around a hypothetical axis at the second ray. This rotation is referred to as supination twist of the TMT joints.[59]

As an example of supination twist of the forefoot, Figure 12–27 shows the response of the segments of the foot to a strong pronation torque across the subtalar joint that may be caused either by a strong medial rotatory force from the leg or by inadequate support of the arch. In weightbearing pronation, the calcaneus everts, and the talus plantarflexes and adducts in space. With sufficient pronation, the navicular bone is pushed downward by the motion of the talar head, thus limiting the ability of the transverse tarsal joint to supinate adequately. The first and second rays will dorsiflex and invert, whereas the fourth and fifth rays will plantarflex and invert. This results in a supination (inversion) twist of the TMT joints to attempt to adequately adjust the forefoot. Because the five TMT joints have some independence, the configuration of the forefoot in a supination twist can vary according to the weightbearing needs of the foot and the terrain.

FIGURE 12-27

Extreme pronation at the subtalar joint is accompanied by adduction and plantarflexion of the head of the talus, eversion of the calcaneus, and (in some instances) pronation at the transverse tarsal joint as a result of the navicular bone being forced down by the talus. If the forefoot is to remain on the ground, the tarsometatarsal joints must undergo a counteracting supination twist.



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Pronation Twist

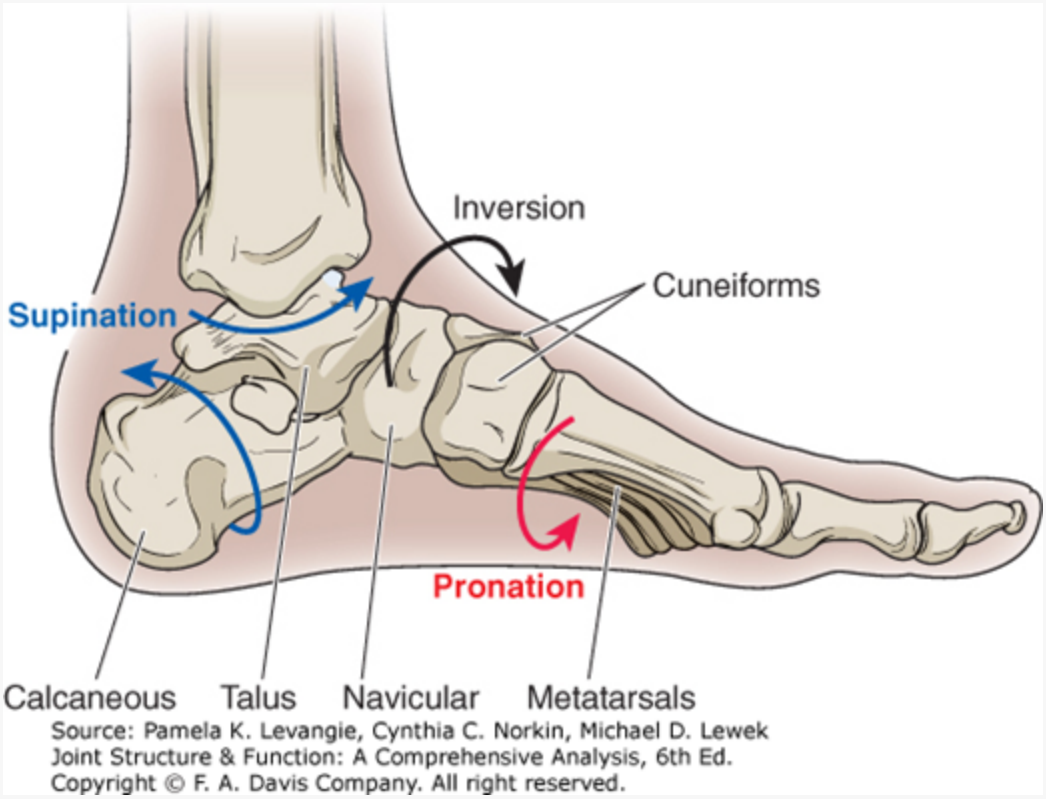
When both the rearfoot and the transverse tarsal joints are locked in supination, the adjustment of forefoot position must be left entirely to the TMT joints. With rearfoot supination, the forefoot tends to lift off the ground on its medial side and press into the

ground on its lateral side. The muscles controlling the first and second rays will cause the rays to plantarflex to maintain contact with the ground, whereas the fourth and fifth rays are forced into dorsiflexion by the ground reaction force. Because eversion accompanies both plantarflexion of the first and second rays and dorsiflexion of the fourth and fifth rays, the forefoot as a whole undergoes a pronation twist.[59]

Pronation twist, like supination twist, can vary in configuration. Although the pronation twist may provide adequate counter-rotation for moderate rearfoot supination, it may be inadequate to maintain forefoot stability in extreme supination. In Figure 12–28, subtalar supination results in calcaneal inversion, with dorsiflexion and abduction of the talus. The transverse tarsal joint will have little if any ability to pronate, inasmuch as the navicular and cuboid bones are carried along with the rearfoot motion. The first and second rays will plantarflex and evert, whereas the fourth and fifth rays will dorsiflex and evert, which results in a pronation (eversion) twist of the TMT joints in an attempt to adequately adjust the forefoot.

FIGURE 12-28

Extreme supination at the subtalar joint is accompanied by abduction and dorsiflexion of the head of the talus, inversion of the calcaneus, and forced supination of the transverse tarsal joint. If the forefoot is to remain on the ground, the tarsometatarsal joints must undergo a counteracting pronation twist.



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Pronation twist and supination twist of the TMT joints occur only when the transverse tarsal joint function is inadequate: that is, when the transverse tarsal joint is unable to counter-rotate or when the transverse tarsal joint range is insufficient to fully compensate for rearfoot position.

Foundational Concepts 12–6

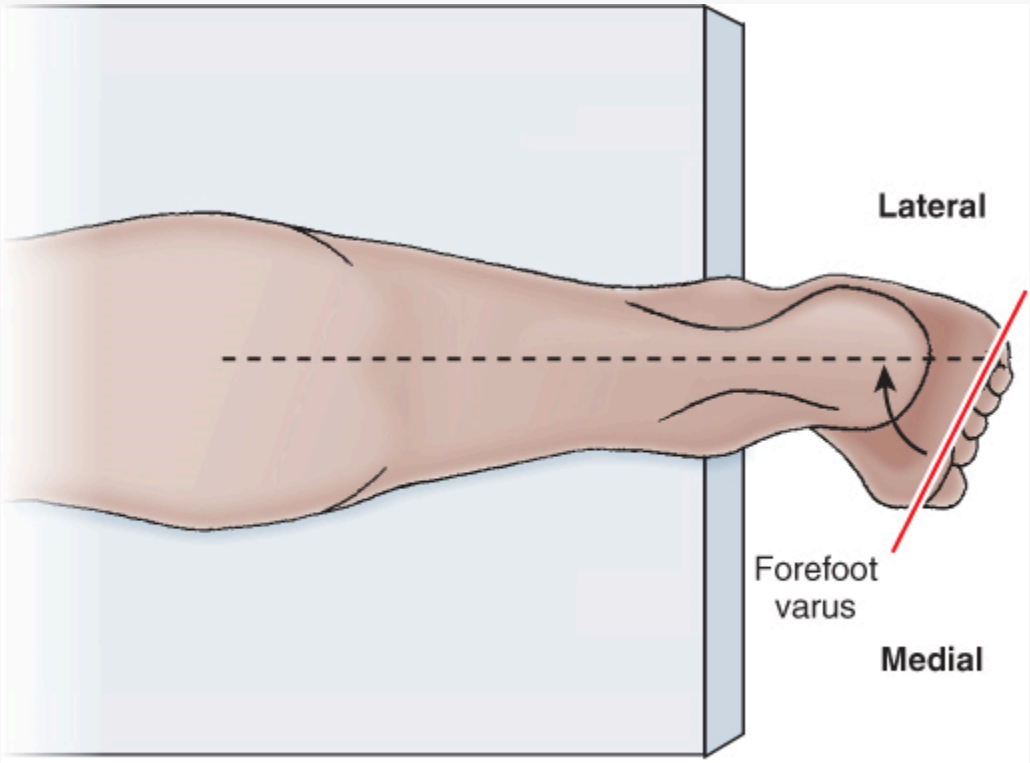
Forefoot Varus

Excessive pronation of the rearfoot has been associated with a forefoot varus deformity. With rearfoot pronation in weightbearing, the forefoot must supinate at the TMT joints to maintain appropriate weight distribution across the metatarsal heads. If adaptive tissue changes result in a sustained TMT supination, the deformity is known as a forefoot varus (effectively the same as a fixed supination twist). In chronically pronated feet, it would be wise to look for adaptive changes in the forefoot. Forefoot varus can be identified by assessing the position of the forefoot in

the frontal plane *in relation to* the subtalar neutral position of the rearfoot (typically in a non-weightbearing position). A forefoot varus deformity is considered present if the forefoot is inverted in relation to the frontal plane when the subtalar joint (calcaneus) is manually held in its neutral position (Fig. 12–29). Identifying forefoot varus can be challenging, given the problems with ascertaining subtalar neutral in the pronated rearfoot, and may best be identified visually as simply present or not present.[93,99,123]

FIGURE 12-29

A forefoot varus deformity is identified by manually placing the non-weightbearing calcaneus in subtalar neutral position (manipulating hand not shown) and determining whether the forefoot is deviated in the frontal plane from a line bisecting the calcaneus.



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Metatarsophalangeal Joints

Although multiple degrees of freedom might be useful to the MTP joints in rare instances, like when the foot participates in grasping activities, flexion and extension are the predominant functional movements at these joints. During the late stance phase of walking, toe extension at the MTP joints permits the foot to pass over the toes, whereas the metatarsal heads and toes help balance the superimposed body weight through activity of the intrinsic and extrinsic toe flexor muscles, coupled with the passive rigidity imposed within the foot. The MTP joints have two degrees of freedom, but flexion/extension motion is much greater than abduction/adduction motion, and extension exceeds flexion. Although MTP motions can occur in weightbearing or non-weightbearing, the MTP joints serve primarily to allow the weightbearing foot to rotate over the toes through MTP extension (known as the metatarsal break) when rising on the toes or during walking.

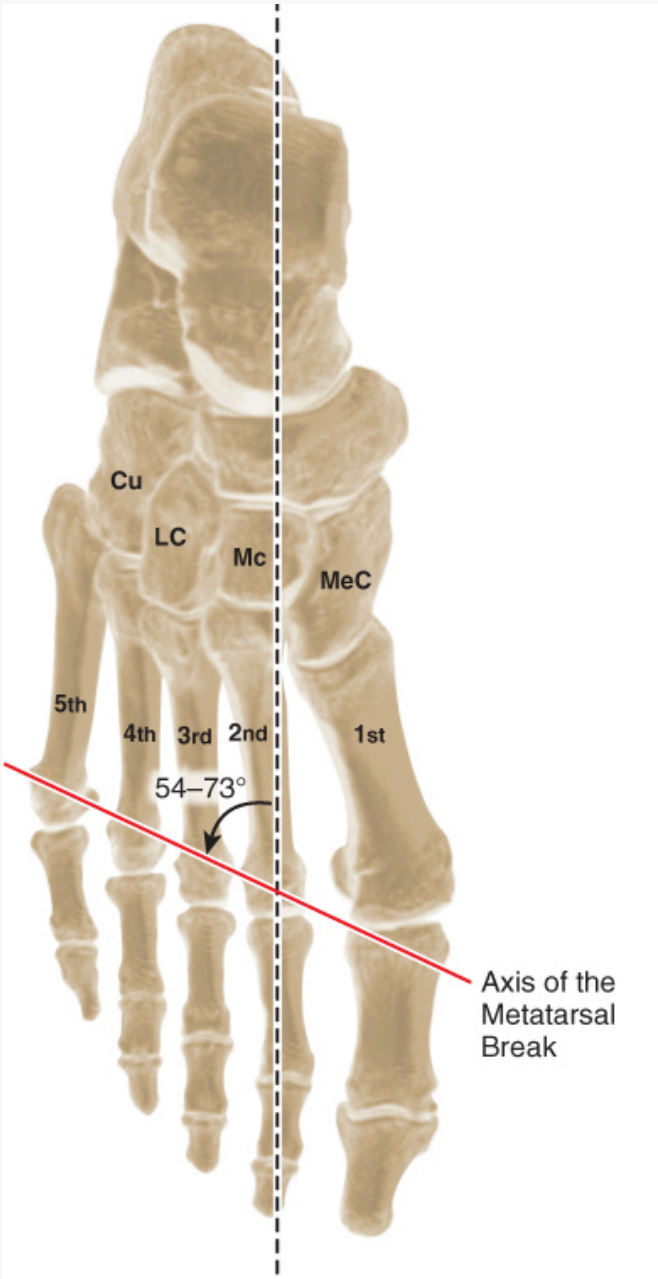
Metatarsophalangeal Extension and the Metatarsal Break

The metatarsal break derives its name from the hinge or *break* that occurs at the MTP joints as the heel rises and the metatarsal heads and toes remain weightbearing. The metatarsal break occurs as MTP extension around a single oblique axis that lies through the second to fifth metatarsal heads (**Fig. 12–30**). The inclination of the axis is produced by the diminishing lengths of the metatarsals from the second through the fifth rays and varies among individuals. The angle of the axis around which the metatarsal break occurs may range from 54° to 73° with respect to the long axis of the foot.[124] The range of MTP extension will vary depending on the amount of dorsiflexion/plantarflexion motion at the TMT joints, the age of the individual, and whether the motion is assessed in weightbearing or non-weightbearing.[125–127] One study reported that the first MTP

joint extension averaged 81° in a younger population (mean age of 21 years of age) compared with 56° in an older population (mean age of 80 years).[126] Gait studies have reported averages ranging between 36° to 65° degrees of MTP extension during walking.[126–128] Limited extension ROM at the first MTP joint will interfere with the metatarsal break and is known as hallux rigidus.

FIGURE 12-30

The metatarsal break occurs around an oblique axis that passes through the heads of the four lesser toes, at an angle to the long axis of the foot that varies widely among individuals from 54° to 73°.



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For the heel to rise while weightbearing, there must be an active contraction of ankle plantarflexor musculature. Most of the plantarflexion muscles also contribute to supination of the subtalar and transverse tarsal joints. The plantarflexion musculature normally cannot lift the heel completely unless the joints of the rearfoot and midfoot

are supinated and locked so that the foot can become a rigid lever from the calcaneus through the metatarsals. This rigid lever will then rotate ("break") around the MTP axis. As MTP joint extension occurs, the convex metatarsal heads glide in a plantar direction on the concave plantar plates and the phalanges that are stabilized by the supporting surface. The metatarsal heads and toes become the base of support, and the body's line of gravity (LoG) must move within this base to remain stable. The obliquity of the axis for the metatarsal break allows weight to be distributed across the metatarsal heads and toes more evenly than would occur if the axis were truly coronal. If the body weight passed forward through the foot and the metatarsal break occurred around a coronal MTP axis, an excessive amount of weight would be placed on the first and second metatarsal heads. These two toes would also require a disproportionately large extension range. The obliquity of the axis of the metatarsal break shifts the weight laterally, minimizing the large load on the first two digits.



Expanded Concepts 12–6

Hammer Toe Deformity

Excessive extension at the MTP joint in a resting position is called a hammer toe deformity. In one group of 20 healthy subjects averaging 56 (±11) years of age without foot problems, the resting MTP joint angle was 11° (±5°) of extension for the first MTP joint and 23° to 42° for the second through fifth MTP joints.[129] This MTP joint angle generally is higher in patients with diabetes and peripheral neuropathy (Fig. 12–31), possibly because of weakness in the intrinsic foot muscles that stabilize the MTP joint.[130] Presumably, because the toes cannot participate properly in weightbearing, hammer toe deformities have been associated with increased pressures under the metatarsal heads that can result in pain or skin breakdown.[129]

FIGURE 12-31

A. Radiographic image of a foot from a healthy subject and **B.** A foot from a subject with diabetes and peripheral neuropathy. The diabetic foot shows a hammer toe deformity (hyperextension at the MTP joint and flexion at the IP joint).



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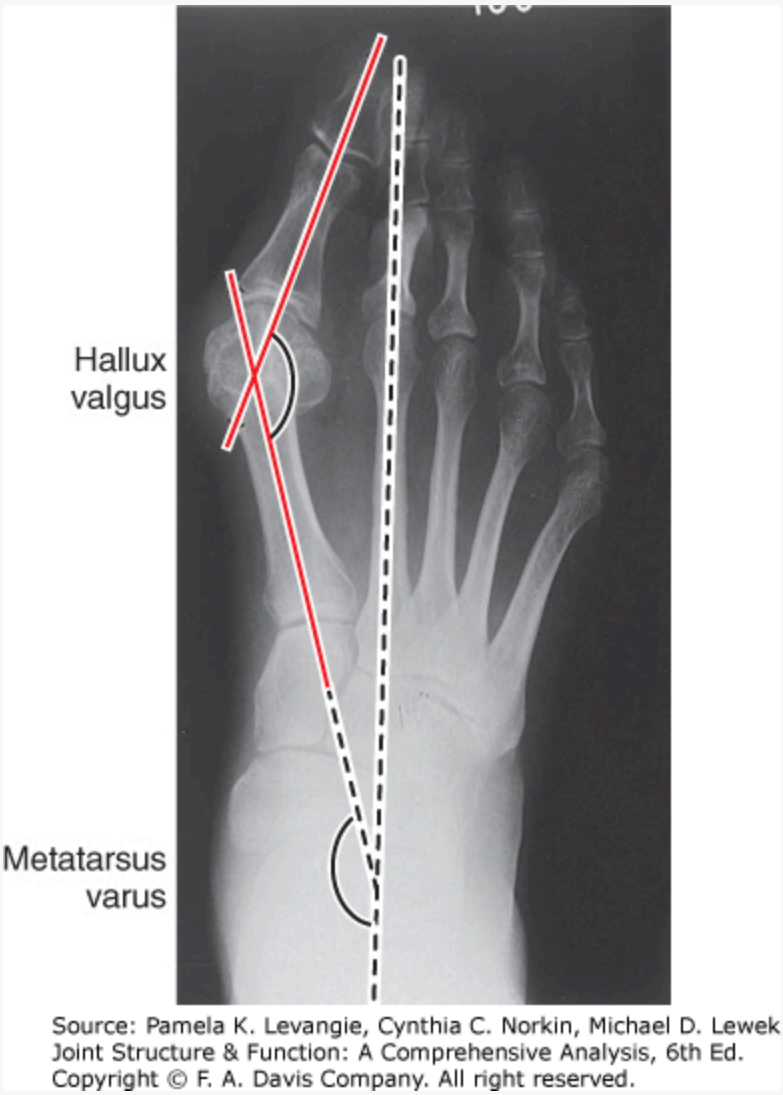
Metatarsophalangeal Flexion, Abduction, and Adduction

Flexion ROM at the MTP joints can occur to a limited degree from neutral position but has relatively little purpose in the weightbearing foot other than when the supporting

terrain drops away distal to the metatarsal heads. Most MTP flexion occurs as a return to neutral position from extension. However, the ability to generate adequate force with the toe flexor musculature is quite important and should be distinguished from the functionally less relevant MTP flexion ROM. Abduction and adduction of the MTP joint appear to be helpful in absorbing some of the force that would be imposed on the toes by the metatarsals as they move in a pronation or supination twist. The first toe normally is adducted on the first metatarsal about 15° to 19°.[46,129] An increase in this normal valgus angulation of the first MTP joint is referred to as hallux valgus and may be associated with a varus angulation of the first metatarsal at the TMT joint, known as metatarsus varus (Fig. 12–32).

FIGURE 12-32

A radiograph showing both a hallux valgus at the first MTP joint, and a metatarsus varus at the first TMT joint. Excessive bony growth at the head of the first metatarsal is due to abnormal pressures from the malalignment.



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Patient Application 12–3

Jared Brown recently visited his physician complaining of right foot pain at his first MTP joint. By inspection, he has an evident hallux valgus deformity. Hallux valgus can result in or may be associated with a reduction in first MTP joint ROM, gradual lateral subluxation of the toe flexor tendons crossing the first MTP joint, reduced weightbearing on the great toe, and increased weightbearing on the first metatarsal head.[131] These structural changes can lead to pain and difficulty during walking. People with a pronated foot may push off during walking with a greater than normal adductor moment on the great toe that pushes the toe into a valgus (MTP joint adducted) position. Localized swelling and pain at the medial or dorsal aspect of the

first MTP joint may be related to an inflamed medial bursa and is commonly called a bunion. The etiology of hallux valgus deformities is likely multifactorial and related to repetitive forces applied to the first MTP joint.[131] The person with a flat foot and excessive pronation like Mr. Brown may have instability and excessive mobility of the first ray, contributing to hallux valgus deformity.[132] Although familial disposition, narrow shoes, pes planus, and hypermobile first TMT joint have been studied, there is insufficient evidence to prove or disprove their association to hallux valgus.[131]

 Expanded Concepts 12–7

Varus/Valgus Terminology

Varus and valgus are consistently used to refer to a decrease or increase, respectively, in the medial angle. However, in the foot and ankle the reference line and the location of the angles keep changing. In the rearfoot, we already noted that varus/valgus either can be synonyms for inversion/eversion of the calcaneus (see Fig. 12–20) or may refer to fixed positioning of the subtalar joint in excessive supination or pronation (see Fig. 12–3). In both cases, the reference line was the posterior leg and a line bisecting the calcaneus. Now we refer to metatarsus varus as a deformity identified as a decrease in the medial angle between the long axis of the metatarsal and the long axis of the foot (or adduction of the metatarsal); hallux valgus is a deformity identified by an increase in the medial angle between the long axes of the metatarsal and proximal phalanx (abduction of the proximal phalanx; see Fig. 12–32). We also defined forefoot varus as a fixed supination twist of the forefoot in relation to a neutral subtalar joint. In this instance, the medial angle is formed by a line through the metatarsal heads and a line bisecting the "neutral" subtalar joint (see Fig. 12–29). Unfortunately, here the terminology may conflict.

To someone who most often assesses deviations in an otherwise normal foot (as we have with Mrs. Montgomery in Patient Application 12–2), a *forefoot varus* refers to a supinated forefoot. To someone who most often assesses paralytic or congenital deformities in the foot, the term **forefoot varus** may indicate a metatarsus varus of all the metatarsals and toes. The plane of the deviation differs in these two usages. Until the terminology is standardized, the context of the presentation will have to give the reader clues as to which usage is being applied.

Plantar Arches

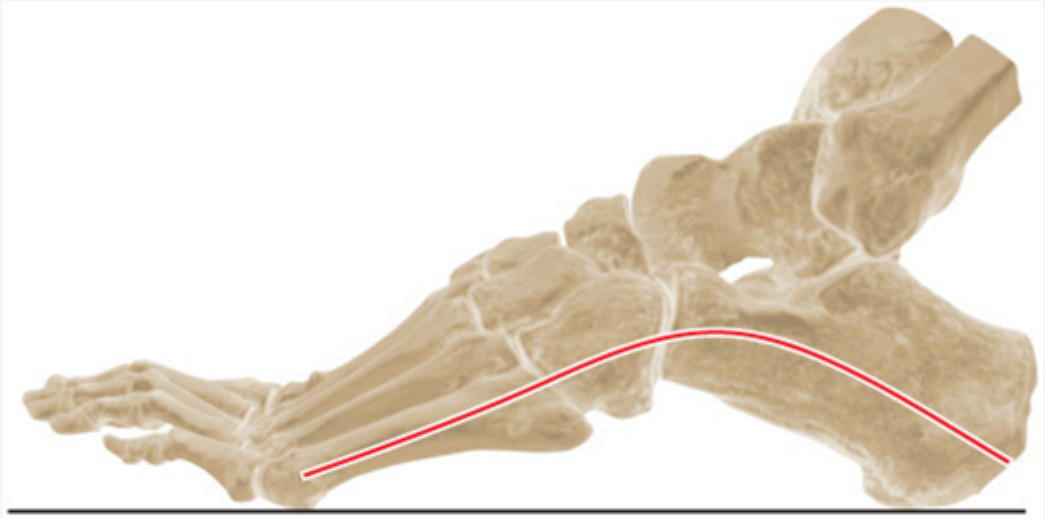
Although we have examined the function of the joints of the foot individually and discussed the effect of each joint on contiguous joints, combined function is best investigated by looking at the behavior of the arch-like structures of the foot. The foot typically is characterized as having three arches: medial and lateral longitudinal arches and a transverse arch. The medial longitudinal arch is the largest and most commonly discussed. Although we may think of and refer to the three arches as if they were separate, the arches are fully integrated with one another (being more analogous to a segmented continuous vault) to enhance the dynamic function of the foot. The arches are usually not present at birth but evolve with the progression of weightbearing. Flattened longitudinal arches are seen in younger children, less than 5 years of age. When gait parameters become similar to that of an adult (around age 5), the majority of children will develop a normal arch.[133] One study found that flat foot was prevalent in 54% of children at 3 years of age, but the prevalence decreased to 25% by the age of 6.[134] In addition to age, the prevalence of flat foot was found to be related to weight and gender.[134,135]

Structure of the Arches

The longitudinal arches are anchored posteriorly at the calcaneus and anteriorly at the metatarsal heads (Fig. 12–33). The longitudinal arch is continuous both laterally (Fig. 12–33A) and medially (Fig. 12–33B) through the foot. However, because the medial arch is higher than its lower lateral counterpart, the medial side usually is the side of reference. The talus rests at the top of the vault of the foot and is considered to be the “keystone” of the arch. All weight transferred from the body to the heel or the forefoot must pass through the talus.

FIGURE 12-33

The longitudinal arch viewed from: **A.** The lateral side of the foot is low in comparison with the view from **B.** The medial side of the foot.



A Lateral view



B Medial view

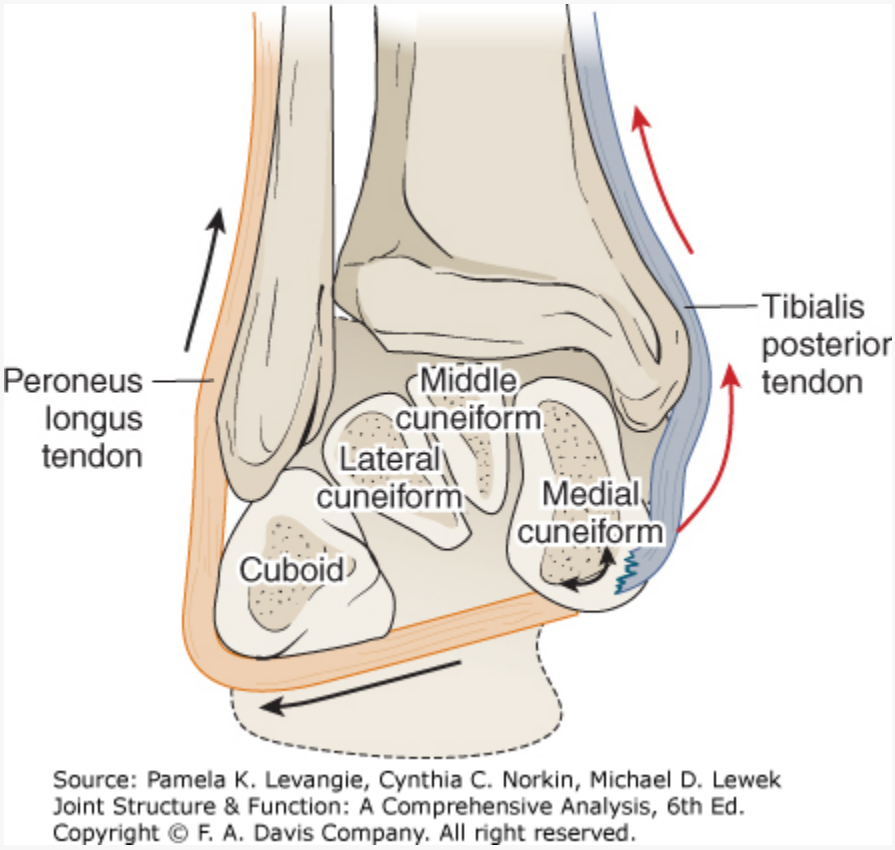
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The transverse arch, like the longitudinal arch, is a continuous structure (Fig. 12–34). The transverse arch can be visualized at the distal metatarsals where the second

metatarsal, recessed into its mortise, is at the apex of the arch. Visualized in the midfoot at the level of the TMT joints, the middle cuneiform bone forms the keystone of the arch.

FIGURE 12-34

The transverse arch at the level of the middle of the tarsals.



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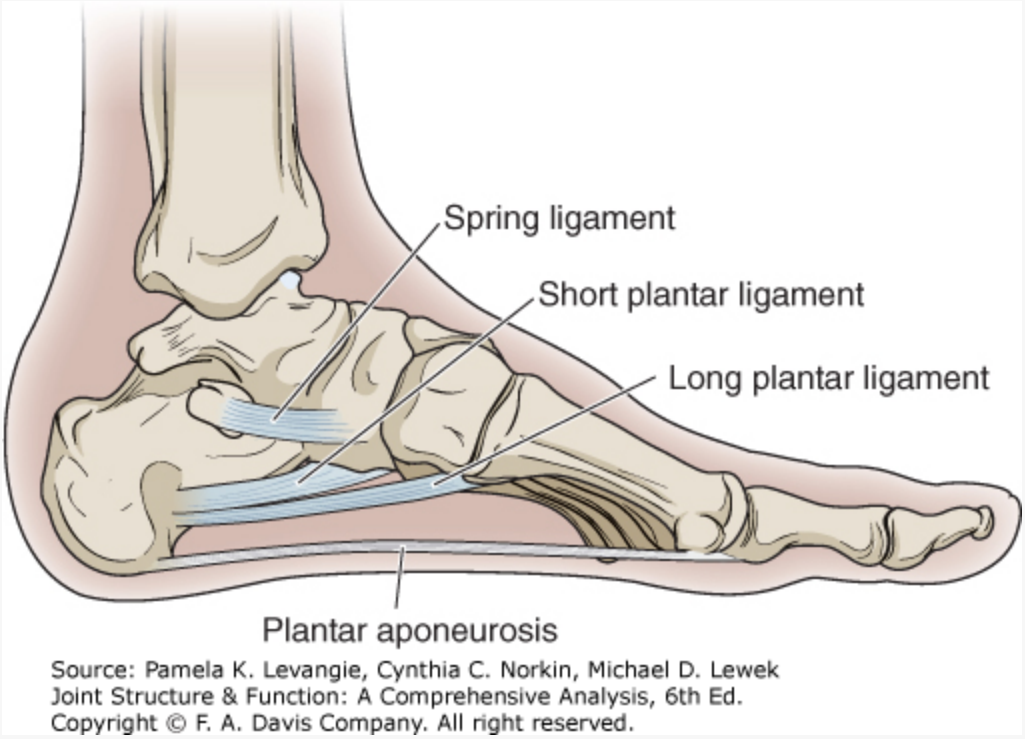
The shape and arrangement of the bones are partially responsible for stability of the plantar arches. As illustrated in Figure 12–34, the wedge–shaped midtarsal bones provide an inherent stability to the transverse arch. The inclination of the calcaneus and first metatarsal contribute to stability of the medial longitudinal arch, particularly in standing (see Fig. 12–33B). Although the structure of the tarsal bones provides a certain

inherent stability to the arches, the arches would collapse without additional support from ligaments and muscles.

Because the three arches can be thought of as a segmented vault or one continuous set of interdependent linkages, support at one point in the system contributes to support throughout the system. The plantar calcaneonavicular (spring) ligament, the interosseous talocalcaneal ligament, deltoid ligament, and the plantar aponeurosis have been credited with providing key passive support to the medial longitudinal arch (Fig. 12–35).[136,137] The "articular" (superomedial) portion of the spring ligament directly supports the head of the talus and is the most important static stabilizer.[35,115,136] The laterally located long and short plantar ligaments support the lateral longitudinal arch and may also contribute, although indirectly, in supporting the medial longitudinal arch.

FIGURE 12-35

The medial longitudinal arch with its associated ligamentous support, including the plantar aponeurosis. The more laterally located short plantar ligament would not ordinarily be seen in a medial view, but is shown as if projected "through" the foot.



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Function of the Arches

Although the arch-like structures of the foot are similar to the palmar arches of the hand, the purpose served by each of these systems is quite different. The arches of the hand are structured predominantly to facilitate grasping and manipulation, but must also assist the hand in occasional weight-bearing functions. In contrast, the foot in most individuals is rarely called on to perform any grasping activities. The plantar arches are adapted uniquely to serve two contrasting mobility and stability weightbearing functions.[138] First, the foot must accept weight during early stance phase and adapt to various surface shapes. To accomplish this weightbearing mobility function, the plantar arches must be flexible enough to allow the foot to (1) dampen the impact of weightbearing forces, (2) dampen superimposed rotational motions, and (3) adapt to changes in the supporting surface. To accomplish weightbearing stability functions, the

arches must allow (1) distribution of weight through the foot for proper weightbearing and (2) conversion of the flexible foot to a rigid lever. The mobility-stability functions of the arches of the weightbearing foot may be examined by looking at the role of the plantar aponeurosis and the distribution of weight through the foot in different activities.

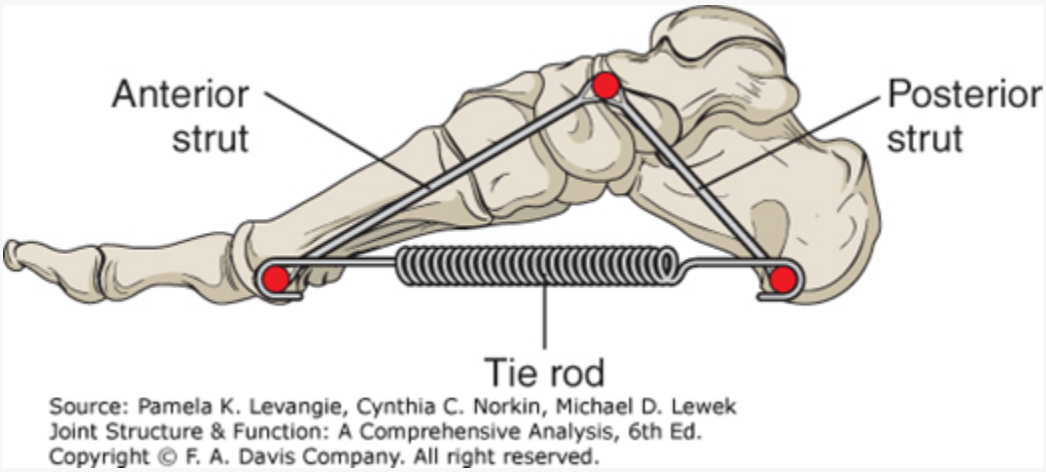
Plantar Aponeurosis

The plantar aponeurosis (plantar fascia) is a dense fascia that runs nearly the entire length of the foot. It begins posteriorly on the medial tubercle of the calcaneus and continues anteriorly to attach by digitations to the plantar plates and then, via the plates, to the proximal phalanx of each toe (see Fig. 12–35).[39,53,139] From the beginning to the end of the stance phase of gait, tension on the plantar aponeurosis increases, with maximum tension averaging 96% of body weight.[140] This increase in tension through the plantar aponeurosis arises as force is transmitted from the Achilles tendon to the forefoot.[140] In vivo experiments using radiographic fluoroscopy have shown that the plantar fascia deforms, or stretches, 9% to 12% during the stance phase of gait.[141] For this reason, the function of the aponeurosis in supporting the arches has been compared with the function of a tie-rod on a truss in classic descriptions.[142] The truss and the tie-rod form a triangle (**Fig. 12–36**); the two struts of the truss form the sides of the triangle and the tie-rod is the bottom. The talus and calcaneus form the posterior strut, and the remaining tarsal and metatarsals form the anterior strut. The plantar aponeurosis, as the tie-rod, holds together the anterior and posterior struts when the body weight is loaded on the triangle. This structural design is efficient for the weightbearing foot because the struts (bones) are subjected to compression forces, whereas the tie-rod (aponeurosis) is subjected to tension forces. Bending moments to

the bone that can cause injury are minimized. It was found that when the foot is loaded, considerable tension develops in the plantar aponeurosis whereas strain in the spring and long plantar ligament is less.[143] The fibrocartilaginous plantar plates of the MTP joints are organized to resist compressive forces from weightbearing on the metatarsal heads as well as to resist tensile stresses presumably applied through the tensed plantar aponeurosis.[53] Therefore, each biological structure is positioned to maximize its optimal loading pattern and minimize the opportunity for injury.

FIGURE 12-36

The foot can be considered to function as a truss and tie-rod, with the calcaneus and talus serving as the posterior strut, the remainder of the tarsals and the metatarsals serving as the anterior strut, and the plantar aponeurosis serving as a tensed tie-rod. Weighting the foot will compress the struts and create additional tension in the tie-rod.



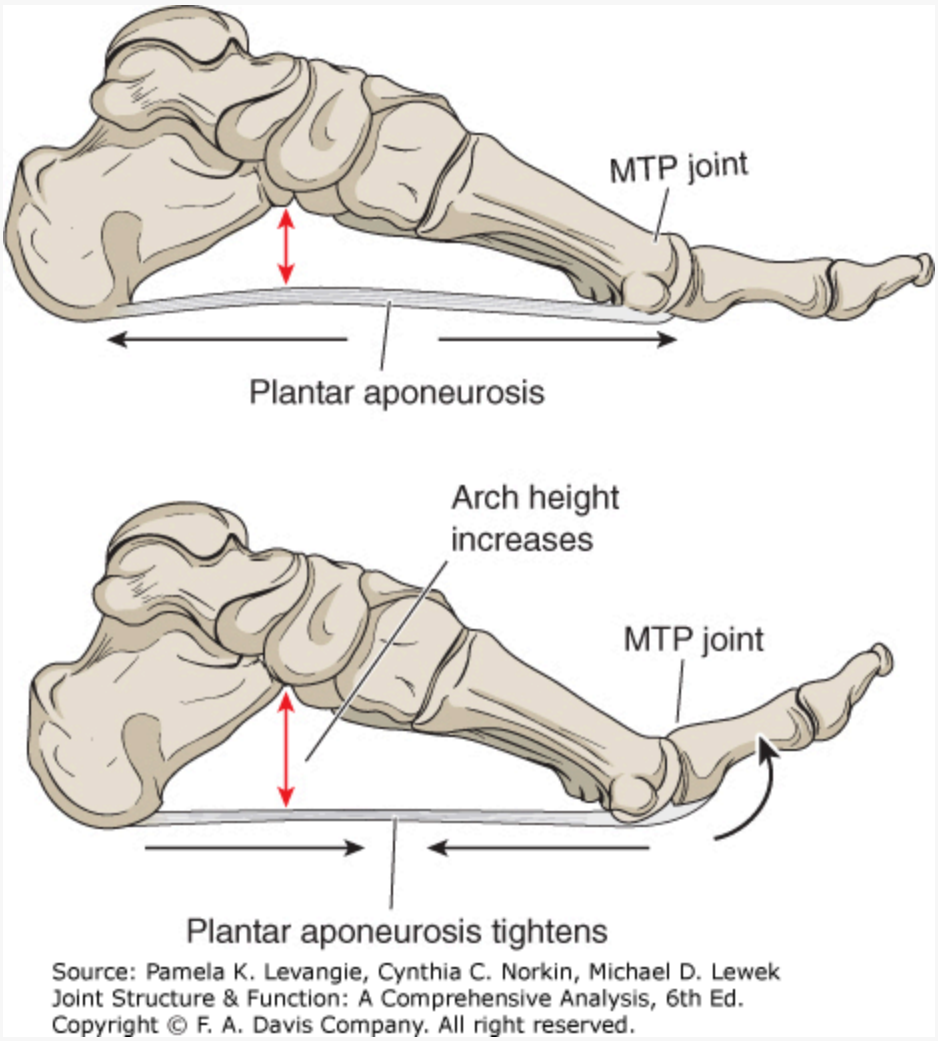
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The plantar aponeurosis and its role in arch support are linked to the relationship between the plantar aponeurosis and the MTP joint. When the toes are extended at the

MTP joints (regardless of whether the motion is active or passive, weightbearing or non-weightbearing), the plantar aponeurosis is pulled increasingly tight as the proximal phalanges glide dorsally in relation to the metatarsals or as the metatarsal heads glide in a relatively plantar direction on the fixed toes. The metatarsal heads act as pulleys around which the plantar aponeurosis is pulled and tightened (Fig. 12–37). As the plantar aponeurosis is tensed with MTP extension, the heel and MTP joint are drawn toward each other as the tie-rod is shortened, raising the arch and contributing to supination of the foot. This phenomenon, which has been referred to as the *windlass mechanism,* allows the plantar aponeurosis to increase its role in supporting the arches as the heel rises and the foot rotates around the MTP joints in weightbearing (during the metatarsal break).[144]

FIGURE 12-37

Elevation of the arch with toe extension occurs as the plantar aponeurosis winds around the metatarsal heads and draws the two ends of the aponeurosis toward each other.



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The tension in the plantar aponeurosis (the tie-rod) in the loaded foot is evident if active or passive MTP extension is attempted while the triangle is flattened (that is, when the subtalar and transverse tarsal joint are pronated). In this example, the range of MTP extension will be limited because the plantar aponeurosis is already loaded. Alternatively, raising the height of the triangle by acting on the struts can unload the tie-rod. For example, when the tibia is subjected to a lateral rotatory force, the rearfoot will supinate, the posterior strut will become more oblique, the height of the medial longitudinal arch will increase, and the plantar aponeurosis (the tie-rod) will be

relatively unloaded. The reduction in tension in the plantar aponeurosis will allow an increase in the range of MTP extension.

Through the pulley effect of the MTP joints on the plantar aponeurosis, the plantar aponeurosis acts interdependently with the joints of the rearfoot to contribute to increasing the longitudinal arch (supination of the foot) as the heel rises during the metatarsal break, thus contributing to converting the foot to a rigid lever for effective push-off. The tightened plantar aponeurosis also increases the passive flexor force at the MTP joints, preventing *excessive* toe extension that might stress the MTP joint or allow the LoG to move anterior to the toes. Finally, the passive flexor force of the tensed plantar aponeurosis also assists the active toe flexor musculature in pressing the toes into the ground to support the body weight on its limited base of support.



Foundational Concepts 12–7

Summary of Tie-Rod and Truss Relations

- Tension in the plantar aponeurosis (the tie-rod) caused by MTP joint extension can draw the rearfoot and forefoot (the struts) together to raise the longitudinal arch (supinate the foot).
- Supination of the weightbearing foot through lateral rotation of the leg or by applying a varus force to the calcaneus will decrease the angle between the struts (raise the apex of the triangle) and release tension in the tie-rod (plantar aponeurosis).
- Flattening of the triangle (pronation of the foot) in weightbearing will increase tension in the plantar aponeurosis (the tie-rod) and limit MTP joint extension.



Patient Application 12–4

Stacy Miller reports that for a week she has had pain at her right heel that is greatest when she first gets out of bed in the morning. She reports that this pain eases after a few steps but increases again when she walks more than two blocks. These

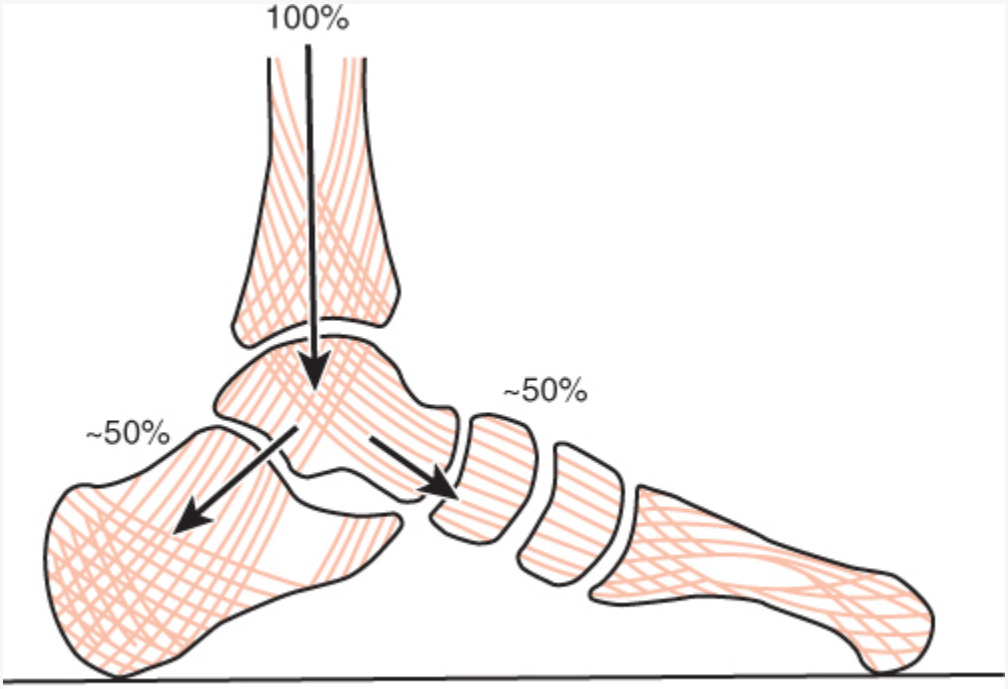
symptoms are classic for plantar fasciitis (inflammation of the plantar aponeurosis).[145] The pain typically is localized at the medial calcaneal tubercle where the plantar aponeurosis inserts. Pain can spread distally down the fascia toward the toes in more chronic conditions. Toe extension may also increase pain because extending the toes places additional tension on the fascia. The risk factors for plantar fasciitis have been identified and include limited ankle dorsiflexion ROM and a high body-mass index.[145] Arch taping or orthotic devices (either pre-fabricated or custom foot orthoses) can be used to support the medial longitudinal arch and decrease stress off the plantar aponeurosis (plantar fascia).[145]

Weight Distribution

Because the foot is a flexible rather than fixed arch, the distribution of body weight through the foot depends on many factors, including the shape of the arch and the location of the LoG at any given moment. Distribution of superimposed body weight begins with the talus, as the body of the talus receives all the weight that passes down through the leg. Therefore, in bilateral and unilateral stance each talus receives 50% and 100% of the superimposed body weight, respectively. At least 50% of the weight received by the talus passes through the large posterior subtalar articulation to the calcaneus, and 50% or less passes anteriorly through the talonavicular and calcaneocuboid joints. The pattern of weight distribution through the foot can be seen by looking at the trabeculae in the bones of the foot (Fig. 12–38). Because of the more medial location of the talar head, about twice as much weight passes through the talonavicular joint as through the calcaneocuboid joint. The somewhat lesser roles of the more laterally located long and short plantar ligaments in supporting the longitudinal arch may be attributable to the reduced weightbearing compression through the calcaneocuboid joint in comparison with the more medially located talonavicular joint.[146]

FIGURE 12-38

Trabeculae of the bones on the medial aspect of the foot illustrating transfer of 100% of the force through the talus, with 50% passing posteriorly to the calcaneus and 50% anteriorly to the forefoot through the talonavicular and calcaneocuboid joints.



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Generally during quiet standing the rearfoot and forefoot bear a majority of the force. When comparing these two regions, a larger load was found under the rearfoot, with peak pressures almost twice as much as the forefoot.[147] The distribution of weightbearing, however, can be variable and largely depends on foot type, whether pes planus, pes cavus, or “normal.”

Plantar pressures are much greater during walking than during standing, with the

highest pressures typically under the metatarsal heads and occurring during the push-off phase of walking (~80% of stance), when only the forefoot is in contact with the ground and is pushing to accelerate the body forward.[148] Plantar pressure under the foot during walking arises from a complex interaction of multiple factors.[149] These factors include physical characteristics such as age, body weight, and height, as well as foot structure, such as arch height, hallux length, and joint motion.[149–153] Gait style and muscle action will also influence plantar pressures.[149] As walking progresses to running, plantar force and peak pressures increase. Maximum plantar force increased from 1.11 to 2.14 times body weight when walking was compared with running.[154] Excessive plantar pressures can contribute to pain and injury in otherwise healthy people or contribute to skin breakdown in patients with diabetes and peripheral neuropathy. Structural and functional factors such as hammer toe deformity, soft tissue thickness, hallux valgus, foot type, and walking speed have been shown to be important predictors of forefoot plantar pressures during walking in people with diabetes.[129]

Muscular Contribution to the Arches

The role of muscle activity in maintaining and supporting the arches of the foot has not been well defined. Extrinsic muscles that include the posterior tibialis, anterior tibialis, fibularis longus, flexor digitorum longus, and fibularis tertius have all been found to play a role in dynamic arch support.[136,155,156] The posterior tibialis has been shown to have the most consistent function in medial longitudinal arch support.[136] The plantar intrinsic muscles that include the abductor hallucis, flexor hallucis brevis, flexor digitorum brevis, abductor digiti minimi, and dorsal interossei are all active during walking.[157,158] Historically, muscle activity was thought to contribute little to arch

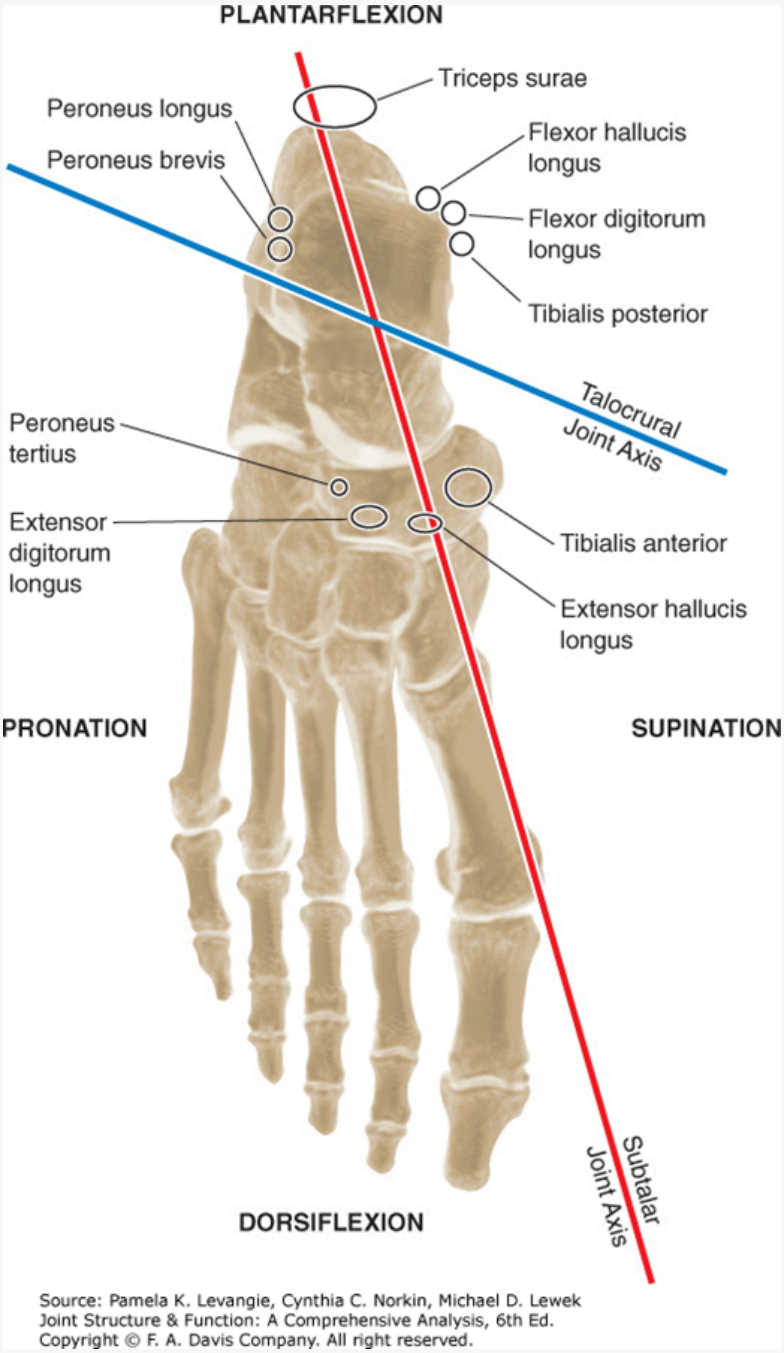
support in the normal *static* foot. However, recent evidence suggests that these intrinsic muscles do provide substantial support to the arch in static standing.[159,160]

Muscles of the Ankle and Foot

As discussed throughout this chapter, muscle activity is critical for the dynamic stability and integration of movement at multiple joints of the foot. There are no muscles in the ankle or foot that cross and act on one joint in isolation; all these muscles act on at least two joints or joint complexes. Muscle function is dependent upon the muscle's structure and, of course, where the muscle passes in relation to each joint axis the muscle crosses. The position of the ankle/foot muscles with respect to the talocrural joint axis and subtalar joint axis is represented in Figure 12–39. As illustrated in this figure, all muscles that pass anterior to the talocrural (ankle) joint will cause dorsiflexion torques or moments, while those that pass posterior to the axis will cause plantarflexion moments. Muscles that pass medial to the subtalar axis will create supination moments at the subtalar joint, whereas those that pass lateral to the subtalar axis will create pronation moments. A muscle, of course, can (and will) create both an ankle joint and a subtalar joint moment simultaneously. For example, the tibialis anterior muscle passes anterior to the talocrural axis *and* medial to the subtalar joint axis, and so it will create a simultaneous dorsiflexion moment at the ankle and a supination moment at the subtalar joint. An understanding of the position of the muscle with respect to the axis is critical for understanding its function.

FIGURE 12-39

Location of muscle insertions in relation to ankle (talocrural) and subtalar joint axes. Muscles that cross anterior to the ankle joint axis will cause dorsiflexion torques at the ankle joint, whereas those that cross posterior to the axis will cause plantarflexion torques. Muscles that cross medial to the subtalar joint axis will cause supination torques, whereas those that cross laterally will cause pronation torques.



A brief overview of muscle function is presented in this chapter with a more comprehensive description of the muscles described in Chapters 13 and 14. Extrinsic ankle/foot muscles are those that arise proximal to the ankle and insert onto the foot. Intrinsic foot muscles arise from within the foot (do not cross the ankle) and insert on

the foot. Extrinsic muscles will be divided further into the three compartments of the lower leg: the posterior, lateral, and anterior compartments.

Extrinsic Musculature

Posterior Compartment Muscles

The muscles of the posterior compartment all pass posterior to the talocrural joint axis and, therefore, are all plantarflexors. The muscles in the posterior compartment are the gastrocnemius, soleus, plantaris, tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles. The gastrocnemius muscle arises from two heads of origin on the condyles of the femur and inserts via the Achilles tendon into the most posterior aspect of the calcaneus. The soleus muscle is deep to the gastrocnemius, originating on the posterior tibia and fibula and inserting with the gastrocnemius into the posterior calcaneus. The two heads of the gastrocnemius and soleus muscles together are known as the triceps surae and are the strongest plantarflexors of the ankle. The large volume of the triceps surae is strongly associated with its ability to generate torque (r^2 = .69).[161] The Achilles tendon inserts perpendicularly on the calcaneus relatively far from the ankle joint axis (see Fig. 12–39). This efficient attachment provides a large moment arm to generate plantarflexion torque. The Achilles tendon has been referred to as a supinator; however, recent evidence suggests that the function of the triceps surae in the frontal plane may be variable and dependent on the position of the subtalar joint with respect to being in inversion or eversion.[162,163] When the subtalar joint was everted the triceps surae produced an inversion moment, and when the subtalar joint was inverted it produced as eversion moment.[162] Because the foot is everted near midstance, activity of the gastrocnemius and soleus on the weightbearing foot helps to invert and lock the foot into a rigid lever both through direct supination of the subtalar

joint and through indirect supination of the transverse tarsal joint. Continued plantarflexion force will raise the heel and cause elevation of the arch (potentially assisted by the increased tension in the plantar aponeurosis as the MTP joints extend). Elevation of the arch by the triceps surae when the heel is lifted off the ground is observable in most people when they actively plantarflex the weightbearing foot.

The soleus and the gastrocnemius together eccentrically control dorsiflexion of the ankle while also supinating the subtalar joint after the foot is loaded in stance. These muscles provide supination torque, which contributes to making the foot a rigid lever for push-off, and continue to provide plantarflexion torque throughout heel rise and plantarflexion of the ankle as the ground reaction force moves to the metatarsal heads and toes.



Expanded Concepts 12–8

Shortening of the Gastrocnemius and Soleus Muscles

Because the gastrocnemius and soleus muscles pass posterior to the ankle joint, a limitation in the length of the muscles (i.e., tightness) results in limited dorsiflexion ROM. Furthermore, the gastrocnemius also passes behind the knee joint, so shortness in the gastrocnemius may further limit dorsiflexion ROM when the knee is extended. Limited dorsiflexion ROM as a result of a short gastrocnemius muscle may cause a pulling sensation behind the knee when sitting and simultaneously extending the knee and dorsiflexing the ankle. Tight hamstring muscles also may contribute to this type of pulling sensation behind the knee and could be distinguished from a short gastrocnemius muscle by extending the hip (which relieves tension on the hamstrings but not the gastrocnemius muscle). Limited dorsiflexion ROM as a result of a short triceps surae muscle group is thought to contribute to excessive pronation at the subtalar joint and has been associated with midfoot and forefoot pain.[74]

The other ankle plantarflexion muscles are the plantaris, the tibialis posterior, the flexor hallucis longus, and the flexor digitorum longus. Although each of these muscles passes posterior to the ankle axis, the moment arm for plantarflexion for these muscles is so small that they provide only 5% of the total plantarflexor force at the ankle.[164] The plantaris muscle is so small that its function can essentially be disregarded.

The tendon of tibialis posterior muscle passes just behind the medial malleolus, medial to the subtalar joint (see Fig. 12–39), to insert into the navicular bone and plantar medial arch. The tibialis posterior muscle is the largest extrinsic foot muscle after the triceps surae and has a relatively large moment arm for both subtalar joint and transverse tarsal joint supination.[162] The tibialis posterior muscle is an important dynamic contributor to arch support and has a significant role in controlling and reversing pronation of the foot that occurs during gait.[136,155,162] When the foot is being loaded early in the stance phase of walking, the tibialis posterior muscle activates eccentrically to control subtalar and transverse tarsal pronation. Tibialis posterior muscle activity continues to work concentrically as the foot moves toward supination and plantarflexion. Because of its insertion along the plantar medial longitudinal arch, tibialis posterior dysfunction is a key problem associated with acquired pes planus, or flat foot.[136,155,162]

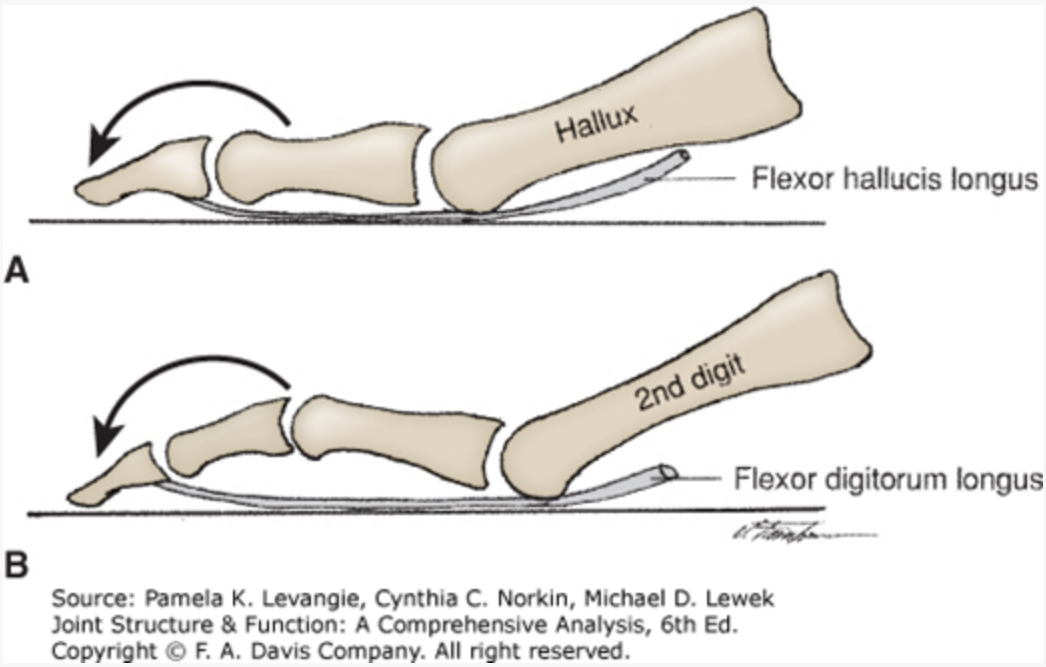
The flexor hallucis longus and the flexor digitorum longus muscles pass posterior to the tibialis posterior muscles and the medial malleolus, spanning the medial longitudinal arch and helping support the arch during gait. Because the tendons pass medial to the subtalar joint, the extrinsic toe flexors also assist in subtalar joint supination. These muscles attach to the distal phalanges of each digit and, through their actions, cause

the toes to flex. The flexor digitorum longus tendon courses around the medial malleolus before splitting and passing to the distal phalanx of the four lesser toes. The line of pull of the tendon is oblique and, without assistance, would cause the toes to simultaneously flex and deviate toward the medial aspect of the foot. The **quadratus plantae** muscle is an intrinsic muscle arising from either side of the inferior calcaneus that inserts into the lateral border and plantar surface of the flexor digitorum longus tendon. This intrinsic muscle and the long toe flexors together form a concurrent force system with a resultant line of pull that flexes the toes with minimal deviation.

Although there is relatively little need for the toes to actually go into flexion, the toe flexors play an important role in balance when the LoG moves toward the metatarsal heads and toes. The toe flexors actively reinforce the passive role of the plantar aponeurosis during gait by eccentrically controlling the MTP extension (metatarsal break) at the end of stance phase, preventing the LoG from passing too far forward in the foot. In more static activities, the toes effectively lengthen the base of support for postural sway and during activities such as leaning forward to reach for or pick up objects, as long as the toe flexors are strong enough to resist MTP extension and press firmly into the ground (Fig. 12–40).

FIGURE 12-40

A. Action of the flexor hallucis longus causes the distal phalanx of the hallux to press against the ground. **B.** Activity of the flexor digitorum longus causes the four lesser toes to grip the ground.



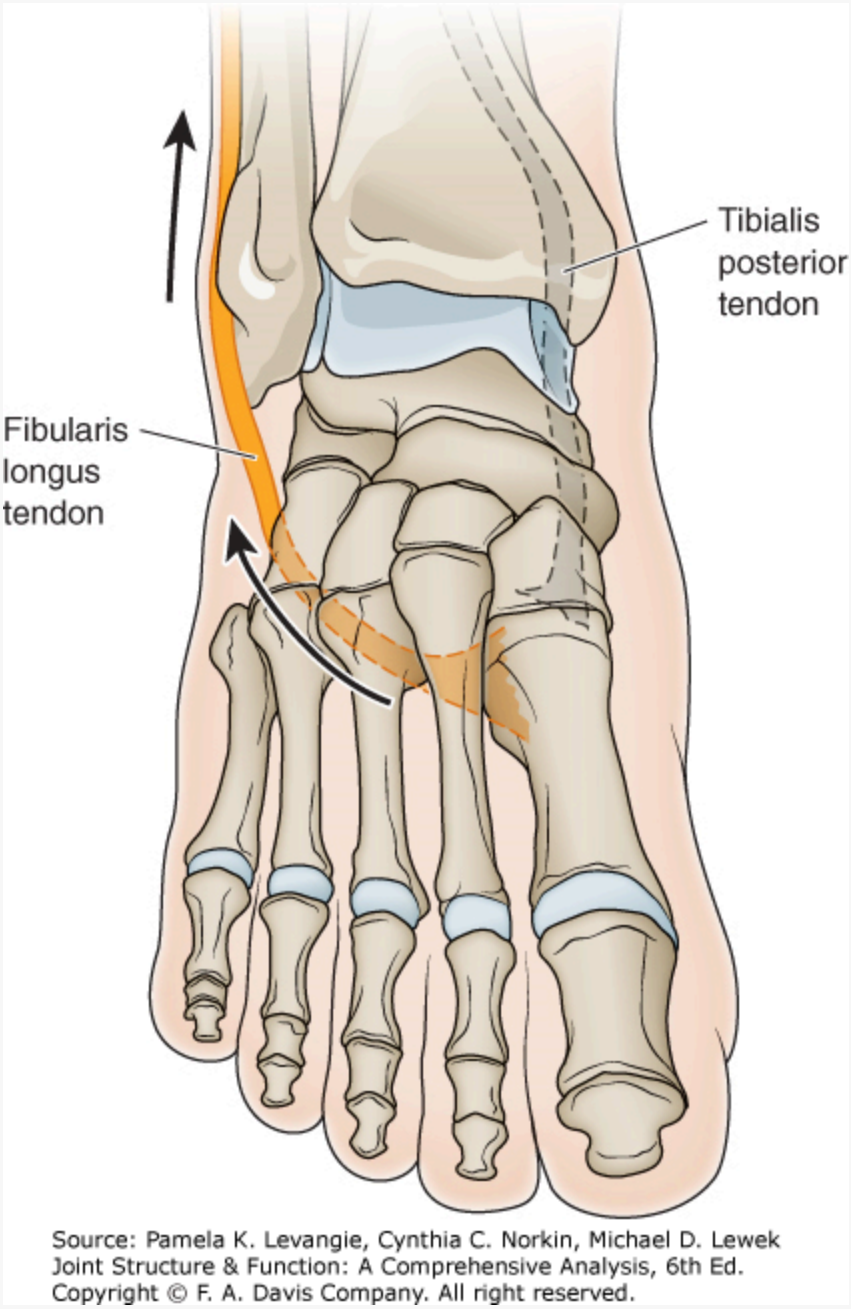
Flexion of the IP joint of the hallux by the flexor hallucis longus muscle presses the toe against the ground (Fig. 12–40A). Flexion of the distal and proximal IP joints of the four lesser toes by the flexor digitorum longus causes clawing (MTP extension with IP flexion) similar to what occurs in the fingers when the proximal phalanx is not stabilized by intrinsic musculature (Fig. 12–40B). As is true in the hand, activity of the interossei muscles can stabilize the MTP joint and prevent MTP hyperextension. Pathologies (such as peripheral neuropathy) that cause weakness of the interossei muscles can contribute to destabilization of the MTP joint, hammer toe deformity (hyperextension at the MTP joint), and excessive stresses under the metatarsal heads.[130] These excessive stresses can contribute to pain under the metatarsal heads (i.e. **metatarsalgia**) or skin breakdown in persons who lack protective sensation (i.e. those with peripheral neuropathy).

Lateral Compartment Muscles

The fibularis (peroneus) longus and brevis muscles pass lateral to the subtalar joint and, because of their significant moment arms, are the primary pronators (everters) at the subtalar joint.[162] Their tendons pass posterior but close to the ankle axis and thus are weak plantar flexors. The tendon of the fibularis longus muscle passes around the lateral malleolus, under the cuboid bone, and across the transverse arch and inserts into the middle cuneiform bone and base of the first metatarsal (Fig. 12–41). Muscle contraction during late stance phase of gait facilitates transfer of weight from the lateral to the medial side of the foot and stabilizes the first ray as the ground reaction force attempts to dorsiflex it, actively facilitating pronation twist of the TMT joints while the rearfoot moves into increased supination.[102] Because of its path across the arches, the fibularis longus tendon is credited with support of the transverse and lateral longitudinal arches as it causes eversion after heel contact, and stabilizes the forefoot after heel rise.[165]

FIGURE 12-41

The tendon of the fibularis longus passes transversely beneath the foot to insert into the base of the first metatarsal. An active contraction of the muscle can support the transverse arch and the first ray of the foot. The tibialis posterior tendon passes medial to the joint and produces supination and eccentric control of pronation.



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The stability of each of the fibularis tendons at the lateral malleolus depends on integrity of the superior and inferior peroneal retinacula located just superior and inferior to the ankle joint respectively (see Fig. 12–9). Sprains of the lateral ankle structures may affect the peroneal retinacula that contribute to lateral ankle and

subtalar support. Laxity of the superior retinaculum can lead to acute or chronic subluxation of the fibularis tendons, causing lateral ankle pain and possibly a sense of instability.[166]

Anterior Compartment Muscles

The muscles of the anterior compartment of the leg are the tibialis anterior, the extensor hallucis longus, the extensor digitorum longus, and the fibularis tertius muscles. All muscles in the anterior compartment of the lower leg pass under the extensor retinaculum (see Fig. 12–9) and insert well anterior to the talocrural joint axis (see Fig. 12–39). Besides being a strong dorsiflexor muscle at the ankle joint, the tibialis anterior muscle passes medial to the subtalar axis and is a key supinator of the subtalar and transverse tarsal joints. Tibialis anterior controls rearfoot plantarflexion from heel contact to 10% stance, and pronation between 10% stance and foot flat.[165] The tendon of the extensor hallucis longus muscle crosses near the subtalar joint axis and is, at best, a weak supinator of the foot. Additionally, the extensor hallucis longus muscle is active in gait when the heel first contacts the ground to control the strong plantarflexion moment at the ankle created by the ground reaction force. Both the tibialis anterior and extensor hallucis longus muscles are active in dorsiflexing the ankle as the foot leaves the ground and in holding the foot up against the plantarflexion torque of gravity.

The tendons of the extensor digitorum longus and the fibularis tertius muscles pass beneath the extensor retinaculum and insert anterior to the ankle joint axis and lateral to the subtalar joint axis; consequently, these muscles are dorsiflexors of the ankle and pronators of the rearfoot. The extensor digitorum longus muscle also extends the MTP

joints of the lesser toes, working with the extensor hallucis longus muscle to hold the toes up when the foot is off the ground. The structure and function of the extensor digitorum longus muscle at the MTP and IP joints are identical to those of the extensor digitorum communis of the hand.

The extrinsic musculature that contributes to supination of the foot is stronger than those that produce pronation. This phenomenon is likely due to the fact that the LoG in weightbearing most often falls medial to the subtalar joint, creating a strong external pronation torque that must be controlled.[167] Similarly, the plantarflexors are stronger than the dorsiflexors because the LoG in weightbearing is most often anterior to the ankle joint axis.

Intrinsic Musculature

The most important functions of the intrinsic muscles of the foot are their roles as (1) stabilizers of the toes and (2) dynamic supporters of the transverse and longitudinal arches during gait. The intrinsic muscles of the hallux attach either directly or indirectly to the sesamoid bones and contribute to the stabilization of these weightbearing bones.[44] The extensor mechanism of the toes is similar to that of the fingers. Activity in the lumbricals, and the dorsal and plantar interossei muscles flex the MTP joints while producing IP extension. This helps to stabilize the MTP joints during walking to allow the toes to remain weightbearing and reduce loading on the metatarsal heads. The flexors act together to resist toe extension during the stance phase of locomotion while the abductors affect the mediolateral pressure by positioning the forefoot.[168] Furthermore, many of the intrinsic muscles arise on the posterior strut (calcaneus) and insert on the anterior strut (metatarsals) of the longitudinal arch, thereby serving to

actively augment the tie-rod function of the plantar aponeurosis.[158] Periodic contraction during standing and consistent contraction during the stance phase of walking dynamically help to relieve stress on the passive connective tissue structures supporting the longitudinal arch.

Patient Application 12–5

Achy, Flat Feet

As in the case of Mrs. Montgomery (Patient Application 12–2), Wyatt Michaels has flat feet, and he complains that his feet often ache at the end of the day. Although there can be several reasons for this type of generalized foot pain, a primary reason may be that the intrinsic muscles have to work harder and longer to stabilize the arches of the flat foot in comparison with a normal or high-arched foot. The high-arched foot in particular receives substantial passive support (i.e., bony alignment and ligaments), whereas the flat foot may rely more on the active contraction of the intrinsic muscles, which results in overuse, fatigue, and an "achy feeling" at the end of the day that could lead to an inflammatory response over time.

The specific function of the intrinsic muscles of the foot can be understood and appreciated by comparing each foot muscle with its corresponding hand muscle. Although most people are not able to use the muscles of the foot with the same ability that they use the muscles in the hand, the potential for similar function is limited only by the unopposable hallux and the length of the digits. Table 12–2 summarizes the specific functions of the intrinsic muscles of the foot.

Table 12-2

Intrinsic Muscles of the Foot

Life Span and Clinical Considerations

The complex interdependency of the foot and ankle joints makes it almost impossible to have dysfunction or abnormality in only one joint or structure. Although foot and ankle disorders can occur across the life span, they are less common in the pediatric population. As children become more active in sports and move to adolescence and young adulthood, traumatic ankle injuries, such as sprains, become more common. These injuries can cause biomechanical abnormalities and deviations that may affect both proximal and distal joints. Over time, if not treated effectively, these abnormalities can cause limitations and progressive deterioration of musculoskeletal structures as the patient ages. As an example, an individual with posttraumatic ankle arthritis may require a total ankle replacement to restore function. As the human life span continues to increase, a rise in geriatric population presenting with foot and ankle injuries will likely become more prevalent. The decline of musculoskeletal function, consisting of decreased ROM and muscle performance, along with increased pain and stiffness, is associated with limitations in gait and balance.[169] Arthritis and bony deformities in the forefoot, midfoot, and rearfoot are prevalent in this population because of pes planus, muscle imbalances, and age-related trauma.[170] These pathologies can have a significant effect on long-term ankle issues seen in the geriatric population.

Foot and Ankle Problems Revisited

The large number of congenital and acquired ankle/foot problems cannot each be described, although several have already been referenced in the chapter. The ankle (specifically the talocrural joint) is a synovial hinge joint with a capsule and associated ligaments that contribute to stability. These connective tissue structures are the most likely to be injured and cause pain in patients. The ligaments that support the proximal and distal tibiofibular joints (the crural tibiofibular interosseous ligament, the anterior and posterior tibiofibular ligaments, and the tibiofibular interosseous membrane) are important for stability of the mortise and, therefore, for stability of the ankle. An injury that disrupts the ligaments supporting the distal tibiofibular joint can cause increased motion of the fibula.[78] Although the distal tibiofibular joint is an extremely strong articulation, injuries can occur when the talus is forcibly dorsiflexed and externally rotated within the ankle mortise.[8] The capsule of the ankle joint is fairly thin and especially weak anteriorly and posteriorly. Therefore, the stability of the ankle depends on an intact ligamentous structure. Excessive eversion or pronation of the ankle and talus can injure the deltoid ligament.[14,15] However, these forces may actually fracture and displace (avulse) the tibial malleolus before the deltoid ligament tears. The anterior and posterior talofibular ligaments run in a fairly horizontal direction, whereas the longer calcaneofibular ligament is nearly vertical.[16] In contrast to the deltoid ligaments, the lateral ligamentous structures help control inversion or supination of the ankle and talus, and are weaker and more susceptible to injury.[14,17]

In contrast with injuries that cause excessive motion, a limitation in dorsiflexion motion can be seen after long periods of immobilization or when the distal tibiofibular joint is fixated surgically with open reduction internal fixation (ORIF). Contractures, particularly

of the gastrocnemius/soleus complex, can contribute to reduced dorsiflexion passive ROM. This impairment is particularly prevalent after many neuromuscular injuries or disorders, such as stroke or spinal cord injury. Physical trauma in the ankle can lead to ankle arthritis because of direct cartilage damage as well as structural changes that lead to biomechanical abnormalities. Therefore posttraumatic arthritis is the most common type of arthritis occurring at the ankle.[75]

Subtalar joint supination is a normal foot motion; a foot that appears fixed in this position is called a “supinated” or pes cavus foot. Similarly, subtalar joint pronation is also a common foot motion; a foot that appears fixed in this position often is called “pronated,” pes planus, or flat foot. Reports have noted approximately 60% of individuals have an arch height considered normal, with 20% having a low arch (pes planus), and 20% having a high arch (pes cavus).[171,172] With pes cavus, the high medial longitudinal arch is associated with a calcaneus that is noticeably inverted. This inverted calcaneus allows the medial heel pad to be observed when the patient stands with the foot straight ahead and has been referred to as the “peek-a-boo” sign.[173] The subtalar and transverse tarsal joints are excessively supinated, which prohibits these joints from participating in shock absorption or in adapting to uneven terrain. Rearfoot supination often is associated with a lateral rotation stress on the leg and lateral weight shift. Recurrent ankle sprains, peroneal tendon pathology, metatarsalgia, and fifth metatarsal stress fractures have been attributed to this lateral weight shift.[174] Although the pes cavus deformity can result from Charcot-Marie-Tooth Disease (CMT), it can also be idiopathic in nature. Ultimately, this deformity can lead to contractures, increased pressure under the metatarsal heads, and pain associated with metatarsalgia.[175]

In a flat foot (pes planus or pes valgus) deformity, the foot typically remains in a position of excessive pronation at the subtalar joint during weightbearing. Posterior tibial tendon dysfunction is usually associated with adult-acquired flat foot. A weightbearing subtalar joint maintained in pronation (e.g., pes planus/flat feet) can create a medial rotation force on the leg that may influence the knee and hip joints. Excessive pronation of the rearfoot has also been associated with a forefoot varus deformity. A forefoot varus deformity is considered present if the forefoot is inverted in the frontal plane when the subtalar joint is manually held in its neutral position (Fig. 12–29).

The structure of the foot can alter the force distribution through the foot. For example, a Morton's type structure has been hypothesized to cause an increased force through the second metatarsal, predisposing that metatarsal to injury, such as a stress fracture. Sesamoiditis is an inflammatory condition of the sesamoid bones and the supporting structures. These structures can become traumatized with excessive loading, leading to acute fracture, stress fracture, osteonecrosis, or chrondromalacia.[47]

Flexion ROM at the MTP joints can occur to a limited degree from neutral position, but has relatively little purpose in the weightbearing foot other than when the supporting terrain drops away distal to the metatarsal heads. Excessive extension at the MTP joint in a resting position is called a hammer toe deformity, resulting in the toe's inability to participate properly in weightbearing. Hammer toe deformities have been associated with increased pressures under the metatarsal heads that can result in pain or skin breakdown.[129] An increase in the normal valgus angulation of the first MTP joint is referred to as hallux valgus and may be associated with a varus angulation of the first

metatarsal at the TMT joint, known as metatarsus varus (Fig. 12–32). Hallux valgus can result in or may be associated with a reduction in first MTP joint ROM, gradual lateral subluxation of the toe flexor tendons crossing the first MTP joint, reduced weightbearing on the great toe, and increased weightbearing on the first metatarsal head.[131] Plantar fasciitis (inflammation of the plantar aponeurosis) is usually identified with pain localized at the medial calcaneal tubercle where the plantar aponeurosis inserts.[145] Pain can spread distally down the fascia toward the toes in more chronic conditions. Toe extension may also increase pain because extending the toes places additional tension on the fascia. The risk factors for plantar fasciitis include limited ankle dorsiflexion ROM and a high body–mass index.[145]

Extrinsic muscles are divided into the three compartments of the lower leg: the posterior, lateral, and anterior compartments. Each compartment is susceptible to injury, which can limit function. In the posterior compartment, limited dorsiflexion ROM as a result of a short triceps surae muscle group is thought to contribute to excessive pronation at the subtalar joint and has been associated with midfoot and forefoot pain.[74] The tibialis posterior muscle works concentrically as the foot moves toward supination and plantarflexion. Because of its insertion along the plantar medial longitudinal arch, tibialis posterior dysfunction is a key problem associated with acquired pes planus, or flat foot.[136,155,162] As is true in the hand, activity of the interossei muscles can stabilize the MTP joint and prevent MTP hyperextension. Pathologies (such as peripheral neuropathy) that cause weakness of the interossei muscles can contribute to destabilization of the MTP joint, hammer toe deformity (hyperextension at the MTP joint), and excessive stresses under the metatarsal heads.[130] These excessive stresses can contribute to pain under the metatarsal heads (i.e. metatarsalgia) or skin

breakdown in persons who lack protective sensation (i.e. those with peripheral neuropathy). In the lateral compartment, the fibularis (peroneus) longus and brevis muscles pass lateral to the subtalar joint and, because of their significant moment arms, are the primary pronators at the subtalar joint.[162] Sprains of the lateral ankle structures may affect the peroneal retinacula that contribute to lateral ankle and subtalar support. Laxity of the superior retinaculum can lead to acute or chronic subluxation of the fibularis tendons, causing lateral ankle pain and possibly a sense of instability.[166]

Summary

- The foot and ankle consists of a complex arrangement of structures and joints that allow the foot to be flexible during the midstance phase of gait, yet relatively rigid during late stance phase. The complexity of the interrelationships makes it easy to disrupt normal function or exceed the limitations of the active and passive tissues that make up the ankle/foot complex.

- Studies that have investigated the relationship between various foot types or alignments and lower extremity injury often show little or no correlation. This should not be surprising, given the many factors that may contribute to health or injury of a given tissue.[1]

- An important point of this chapter, however, has been to consider how various structures and structural deviations can affect movement and stresses on adjacent structures and tissues. Although group studies may minimize the apparent influence of a given structural problem in dysfunction, the problem should not be

ruled out as a factor in, or even primary cause of, pain or dysfunction in an individual case.

- The kinesiology impact of a given set of alignments and apparent forces in producing maladaptive stresses should be placed in the full context of the individual person, his or her health status, and his or her activity level and activity goals.

Study Questions

1. Identify the proximal and distal articular surfaces that constitute the ankle (talocrural) joint. What is the joint classification?
2. Describe the proximal and distal tibiofibular joints, including classification and their composite function.
3. Identify the ligaments that support the tibiofibular joints.
4. Describe the ligaments that support the ankle joint, including the names of components when relevant.
5. Why is ankle joint motion considered triplanar?
6. Why does the fibula move during dorsiflexion/plantarflexion of the ankle?
7. What are the primary checks of ankle joint motion?
8. Which muscles crossing the ankle are single-joint muscles?

9. Describe the three articular surfaces of the subtalar joint, including the capsular arrangement.

10. Which ligaments support the subtalar joint?

11. Describe the axis for subtalar motion. What movements take place around that axis, and how are these motions defined?

12. When the foot is weightbearing, the calcaneus (the distal segment) of the subtalar joint is not free to move in all directions. Describe the movements that take place during weightbearing subtalar supination/pronation.

13. What is the close-packed position for the subtalar joint? Which motion of the tibia will lock the weightbearing subtalar joint?

14. Describe the relationship between the subtalar and the talonavicular joint with regard to articular surfaces, axes, and available motion.

15. Describe the articulations of the transverse tarsal joint.

16. What is the general function of the transverse tarsal joint in relation to the subtalar joint?

17. What are the TMT rays? Describe the axis for the first and fifth ray and the movements that occur around each axis.

18. What is the function of the TMT joints in relation to the subtalar and the transverse tarsal joints?

19. How does pronation twist of the TMT joints relate to supination of the subtalar joint?

20. What ligaments contribute to support of the medial longitudinal arch of the foot?

21. What is the weight distribution through the various joints from the ankle through the metatarsal heads in unilateral stance?

22. How does extension of the MTP joints contribute to stability of the foot?

23. In terms of structure, compare the MTP joints of the foot with the MCP joints of the fingers.

24. What is the metatarsal break? When does this occur, and how is it related to support of the longitudinal arch?

25. What is the role of the triceps surae muscle group at each joint it crosses?

26. What is the non-weightbearing posture of the subtalar and transverse tarsal joints?

27. What other muscles besides the triceps surae exert a plantarflexion influence at the ankle? What is the primary function of each of these muscles?

28. Which muscles may contribute to support of the arches of the foot?

29. What is the function of the quadratus plantae? What is the analog of this muscle in the hand?

30. Drawing an analogy between the foot and the hand, describe the function of each of the intrinsic and extrinsic foot muscles.

31. If a person has a pes planus, describe two possible causes for this condition.

32. Identify at least three possible effects of pes planus.

33. Identify three primary signs to identify an excessively supinated or pronated foot.

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Chapter 13: Posture

Lee N. Marinko; Cynthia C. Norkin

Introduction

The focus of this chapter is to explore how various body structures work together to enable the body as a whole to either maintain a particular posture or to transition from one posture (or alignment) to another during movement. A brief review of the integrated mechanisms associated with postural control is included, and knowledge of individual joint and muscle structure and function is used as the basis for determining how each contributes to equilibrium and stability in an ideal posture. The internal and external forces acting on the body in relation to various postural orientations are considered, as well as how deviations in initial alignment can alter joint movement and function. This chapter begins with what is considered ideal alignment for various static positions before considering some common postural deviations, and discusses how these deviations may contribute to, or be the result of, various conditions encountered in physical therapy practice. Life span differences in postural control and balance will also be addressed.

Posture and Balance Defined

Posture

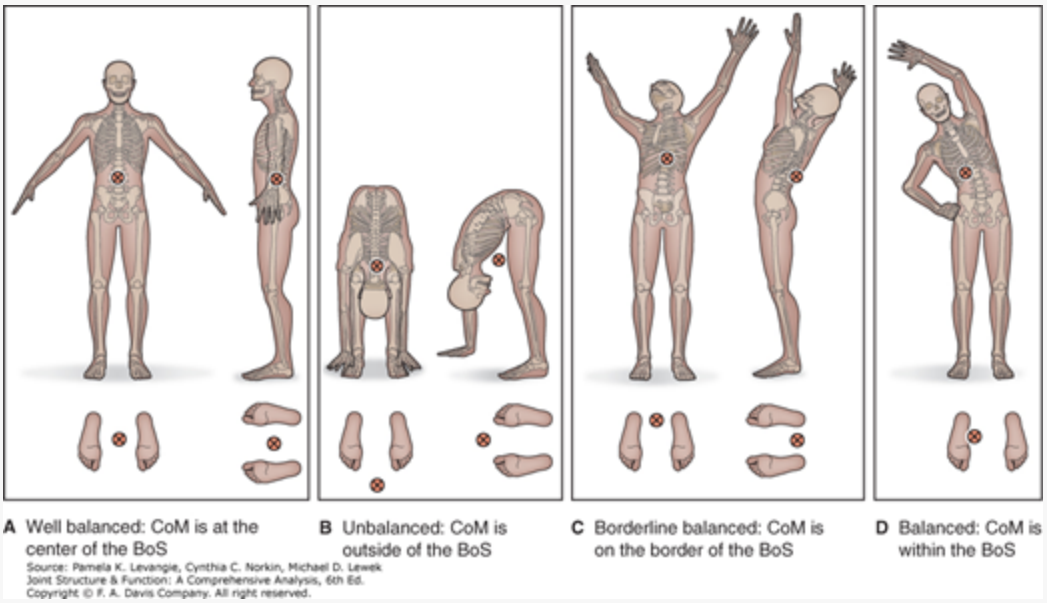
Posture is the orientation, or alignment, of the human body, and can be either static or dynamic. In **static posture,** the body and its segments are distributed in a manner that maintains the body in equilibrium. Standing, sitting, or lying are all examples of static

postures with the body at rest. **Dynamic posture** refers to postures in which the body or its segments are moving—walking, running, jumping, throwing, and lifting are all examples of dynamic postures. All successful human movement is initiated from a static posture and is maintained through the integration of multiple systems.

The force of gravity directly impacts a person's stability. In order for you to remain stable, your center of mass (CoM) and the vertical projection from this point, referred to as the line of gravity (LoG), must be maintained within your base of support (BoS; see Chapter 1). Stability is directly related to how easy or difficult it is to displace the CoM outside of your BoS, as once your LoG moves beyond your BoS, you may fall. You may increase your stability to avoid a fall by adopting a larger BoS or by lowering your CoM closer to the BoS. The multi-articulating components of the human skeleton can be arranged in an infinite number of positions. This rearrangement of body segments can influence the position of the body's CoM and LoG and hence influence its stability (Figure 13–1).[1,2]

FIGURE 13-1

The center of mass (CoM) and base of support (BoS) impact stability. When the CoM is outside of the BoS, stability is affected.



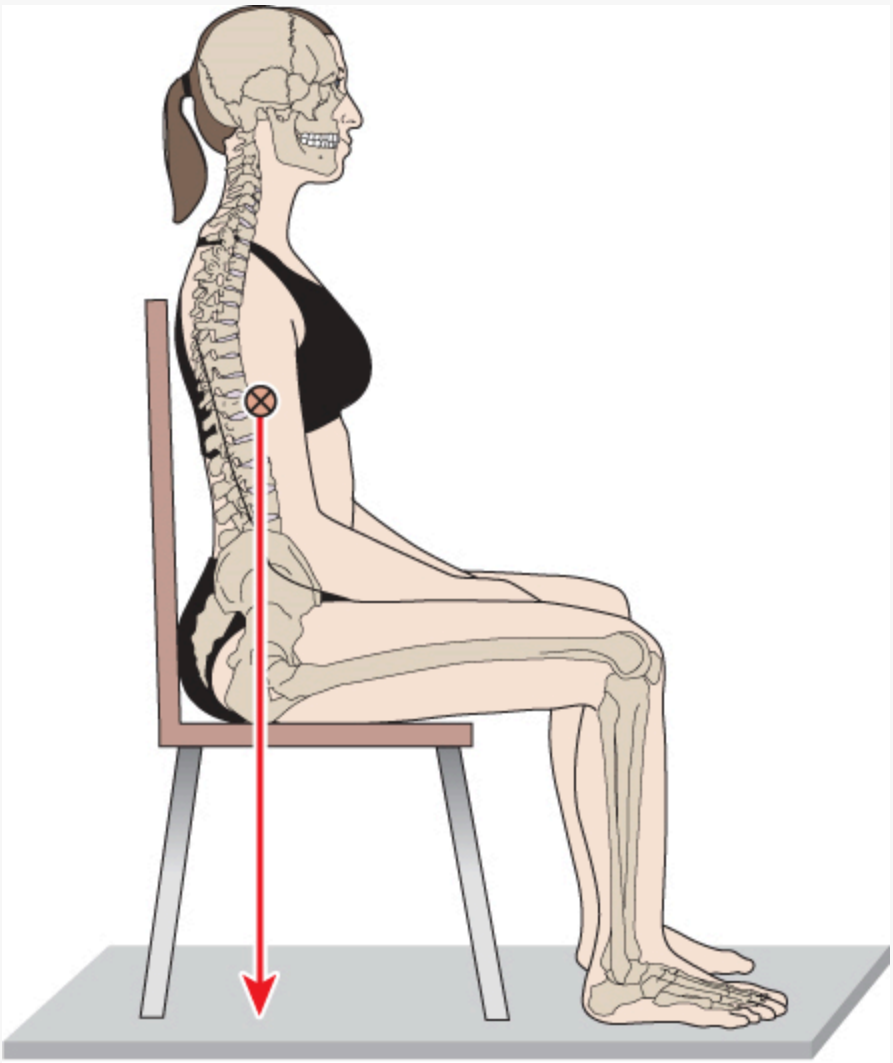
Balance

Balance is the process by which upright posture is maintained. Postural control is responsible for achieving, maintaining, or restoring appropriate balance.[3] When forces are in equilibrium, the body remains at rest, or static, and is considered stable. Balance is more than simply a mechanical issue, and it requires multiple systems to ensure that the body maintains equilibrium. Maintaining the body's COM within the boundaries of its BoS is a requirement for maintaining stability, or balance, and is the ultimate goal of postural control. In standing anatomical position, the human's BoS is bounded by the area from the back of the heels circling the tips of the toes, and the CoM is located at approximately the level of the second sacral segment in the midsagittal plane (see Figure 13–1). In a seated posture, the BoS now includes the boundaries of the feet as well as the outline/footprint of the item that the individual is seated upon. When seated, balance is dependent on the CoM of the head, arms, and trunk because the pelvis and lower extremities are supported. Therefore, when seated, the CoM shifts

superiorly to a region just below the axilla (**Figure 13–2**). Redistribution of the limbs as depicted in Figure 13–1 will alter the position of the individual's CoM and LoG. Characteristics such as age, height, and weight may alter the CoM and, therefore, vary the requirements for stability.[4] Human balance also requires a complex interplay between the mechanical properties of the musculoskeletal system and the integrity of the central nervous system (CNS).

FIGURE 13-2

When seated, the CoM is just below the axilla and the BoS includes the boundaries of the feet and the outline of the chair.



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
Joint Structure & Function: A Comprehensive Analysis, 6th Ed.
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Patient Application 13–1

The Romberg and the Sharpened Romberg tests are clinical measurements of an individual's balance that differ by the position of the feet.[5] In the Romberg test, the individual stands with feet together; in the Sharpened Romberg test, the individual places the heel of one foot directly in front of the toe of the other (tandem stance). Both positions reduce the BoS in standing compared with the anatomical position (feet approximately shoulder width apart). In the Romberg test, the BoS in the frontal plane has been reduced from the anatomical position. In the Sharpened Romberg test the BoS in the frontal plane is reduced even further; however, the BoS in the

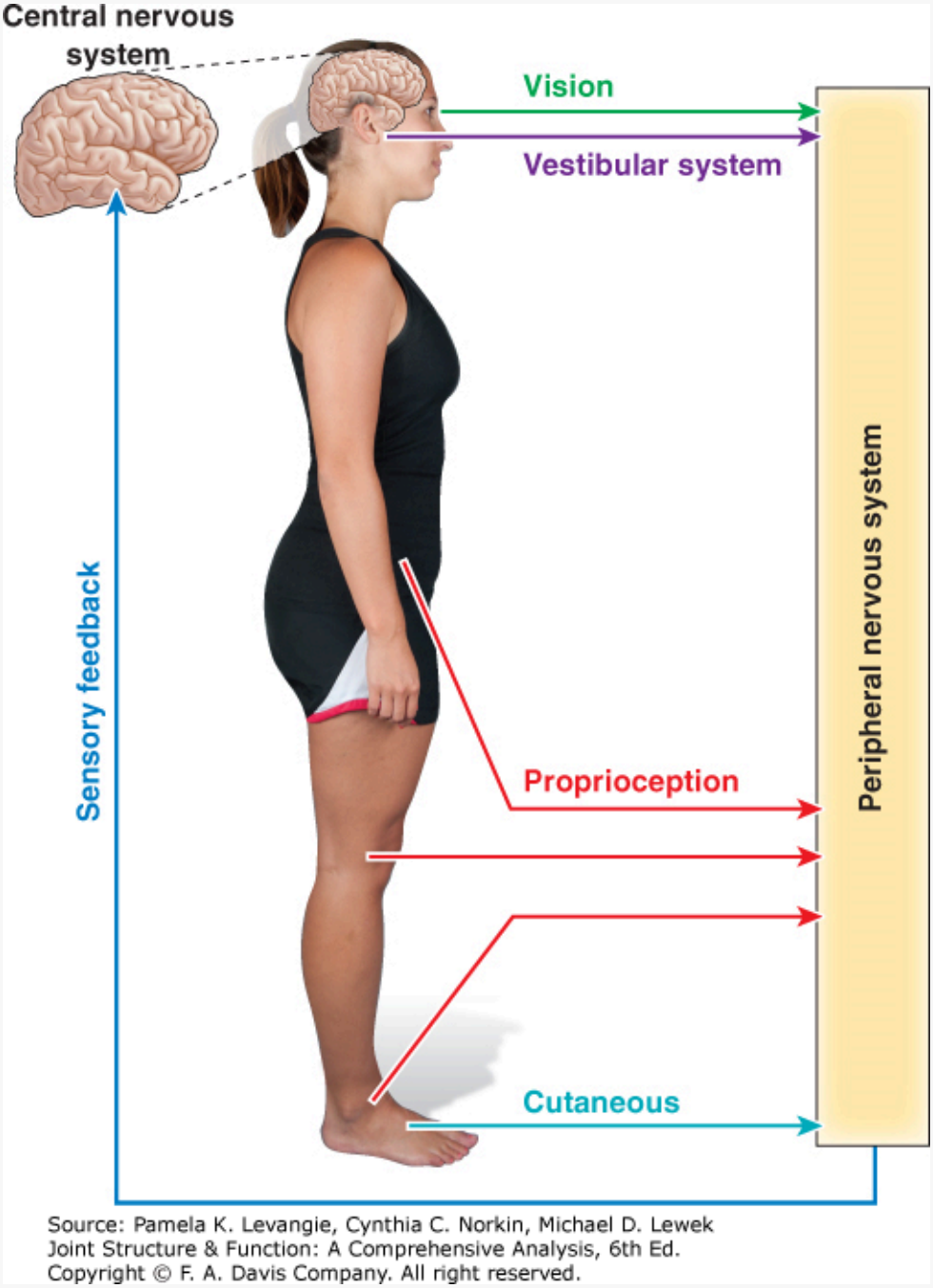
sagittal plane has been increased. Therefore, someone standing in Sharpened Romberg may have greater stability in the anteroposterior direction compared with normal standing, but would have significantly diminished stability in the medial–lateral direction, requiring greater effort to maintain stability. This test progression is an example of how altering the BoS can impact an individual's stability.

The Role of Sensorimotor Motor Control in Posture and Balance

Postural control is the act of maintaining balance at rest, during movement, or with positional changes given the constraints of the individual, the environment, or the task. All successful movement requires postural control. In the past, clinicians believed that postural control was simply a reflexive function manifested by the CNS. Today it is understood to be a complex motor skill that incorporates multiple processes across many systems and can be learned or acquired.[6] In experimental studies challenging postural control, Scholz and colleagues were able to demonstrate that when stability was disturbed under varying conditions, individuals consistently realigned their CoM and body segments into the pre-perturbation position, supporting the concept that the primary goal of postural control is maintenance of the body's CoM and LoG within the BoS.[2] Although only a relatively small amount of muscular activation is required to maintain a stable, erect standing posture, the maintenance of posture is complex and part of the body's motor control system.[5] Postural control requires intact and appropriate neuromuscular integration of the peripheral and central nervous systems. Specifically, information is incorporated from the visual system, the vestibular system, the somatosensory system (proprioceptive, cutaneous, and joint receptors), and the musculoskeletal system (joints and muscles; Figure 13–3).[5,7]

FIGURE 13-3

Postural control requires integration of the peripheral and central nervous systems.



Humans use two primary mechanisms to maintain stability: **anticipatory postural**

adjustments (APA) and **compensatory postural adjustments (CPA).**[8] APAs have been described as feedforward activations of trunk and limb muscles prior to a self-imposed balance perturbation or movement of the CoM. Feedforward indicates that the muscles contract in anticipation of movement rather than as the result of movement.

The role of APAs is to reduce any negative known consequences of the intended movement; in Example 13–1, it would be falling forward. Therefore, there has to be some prior knowledge of the task and the environment for the APAs to be successful.[9] CPAs are activated motor compensations (muscle contractions) that occur when balance is disturbed (also known as feedback motor strategies). CPAs respond to sensory feedback information in the presence of either predicted or unpredicted perturbations.[10] If posture is perturbed unexpectedly, CPAs are the primary strategy used by the CNS to restore equilibrium. In the presence of known perturbation or planned movement, APAs and CPAs work together. In simplistic terms, APAs prepare the body for movement and CPAs adjust during and after the movement.



Example 13–1

As a simple example, imagine raising your arms straight out in front of you while standing. Prior to lifting your arms, the ankle plantarflexors should contract to draw the tibia and rest of the body posteriorly, which will shift the CoM slightly posterior. Shifting the CoM slightly posterior in preparation for the forward arm raise will allow the CoM to return to the middle of the BoS when the arms are raised. Thus, the APA allows the body to prepare for an upcoming movement to keep the CoM centered within the BoS at the end of the intended movement.

Sensory information is critical to postural control. The primary sources of sensory information that contribute to postural control are the visual, vestibular, and

somatosensory systems. Vision, for example, plays an important role in regulating APAs. To demonstrate the importance of vision, Mohapatra and Aruin measured APAs during trunk perturbations under varying visual conditions.[11] They observed a significant increase in APAs when subjects received visual cues, but saw no APAs when vision was completely obstructed, supporting the fact that vision is a requirement that allows APAs to prepare the body for perturbations. The vestibular system is also involved in triggering postural adjustments and is necessary when there is conflicting somatosensory or visual information.[12] The vestibular system functions as the spatial reference system to gravity. In this role, the vestibular system responds to changes in the orientation of the head and trunk in space, but it is unaffected by changes in the support surface.[13,14] Finally, somatosensory information provides the CNS with information about the body's position with respect to a support surface, as well as the inter-relationship between all the body segment's positions and movement within space. Specific sensory cells (mechanoreceptors) are located within the skin, joint, and muscle structures to provide information regarding touch, joint motion, and tendon and muscle stretch, as well as how quickly the limb is moving (see Figure 13–3). When there is adequate visual information and a stable surface, the majority of balance control is the result of somatosensory information from the joints of the lower extremities. In the presence of unstable surfaces, the CNS will rely less on somatosensory information and re-weight information from the visual and vestibular systems, demonstrating the coordinated integration of sensory information in postural control.[6] Overall, it is important to understand the integrated roles of these multiple systems when observing resting posture and movement, because deviations from ideal alignment may be indicative of underlying sensory, visual, or vestibular disorders.

Recognizing these disorders may assist in predicting static and dynamic postural deficits. Table 13–1 provides a summary of the elements of postural control.

Table 13-1

Postural Control

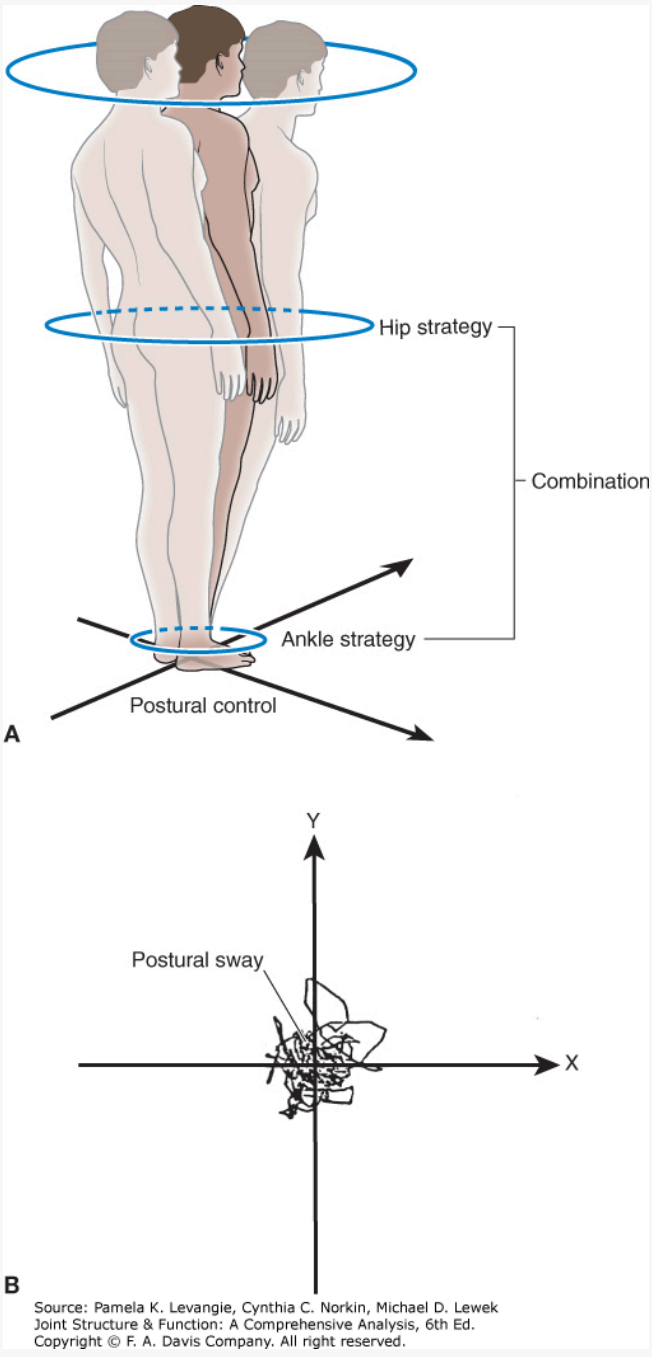
Control of Posture in Static Positions

Stability depends, in part, on the individual's BoS. Although static posture is emphasized in this chapter, the term *static* can be misleading. Unless every part of the body is supported on a fixed surface, the influence of gravity and inertia create constant external forces to produce movement within the limits of stability. These small amounts of movement represent a subtle and often visually imperceptible motion known as *postural sway*.[5] Static posture, therefore, is not one single point, but is instead an area within which individuals can maintain their CoM and LoG within their BoS without requiring a step or change in BoS (**Figure 13–4**).[6] Maintenance of static posture is dependent upon the orientation and integrity of body segments, resting muscle tone, and small magnitudes of muscle activation. In a static upright posture, the head remains very stable despite the fact that the upper cervical spine is the most mobile region of the vertebral column (see Chapter 4, The Vertebral Column). This is likely due to the abundance of muscle spindles and their direct connection to both the visual and vestibular systems within the cerebellum.[15] In an upright standing position, the distance between the relatively small BoS and high CoM results in a need for postural

control mechanisms that are influenced by the support surface, available joint mobility, muscular control, and intact sensory information.

FIGURE 13-4

Postural sway and limits of stability. **A.** Stability is controlled by the ankles, hips, and a combination of the two. **B.** Recording of the movement of the center of pressure for 60 seconds in a subject standing on a balance platform.



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
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Postural Control Strategies

Three basic motor strategies can maintain stability during quiet stance: an ankle strategy, a hip strategy, and a combination of the two. The *ankle strategy* is often the first strategy employed and is used to manage small perturbations. As the simplest

strategy, the ankle strategy describes the body's movement as an inverted pendulum about the ankle.[16] When postural sway occurs in the anterior to posterior direction, discrete distal to proximal muscle activation patterns are observed that are consistent with the direction of sway. Muscle activity begins in the ankle joint, then in the thigh, and finally in the trunk. For example, when translation of the body is in the posterior direction, muscle activity begins with the tibialis anterior, followed by the rectus femoris, and then in the rectus abdominis.[17] When translation is in the anterior direction, muscle activity begins with the gastrocnemius, followed by the biceps femoris, and finally the erector spinae.

The second strategy is called the *hip strategy* and is incorporated when either the perturbation is larger or the base of support is smaller.[1,17] This strategy functions as a multilink pendulum, with motion occurring primarily at the hip such that muscle activation occurs in a proximal to distal direction.[13,17] During this strategy, when postural sway occurs in the anterior to posterior direction, muscle activity begins in the abdominal muscles followed by the quadriceps. When the sway is in the posterior to anterior direction, activation is initiated in the erector spinae, followed by the hamstrings.[17]

Although the hip and ankle strategies have been described as independent and separate events based upon the varying demands placed on the body, Creath and colleagues suggest that these strategies are always present to a certain extent, therefore inferring a third motor strategy, which is a combination of both ankle and hip occurring concurrently.[13] Muscle activation patterns in all of these strategies appear dependent upon the task or demands of the environment so that people can demonstrate varying

degrees of both ankle and hip strategies.

Patient Application 13–2

Pathologies associated with the central or peripheral nervous system that interfere with normal somatosensory integration, such as multiple sclerosis or peripheral vascular disease, are associated with balance deficits and an increased risk for falls. These pathologies disrupt the normal sensory processing at the ankle and can therefore limit postural stability from an ankle strategy. Rehabilitation that targets balance training using a hip strategy may be incorporated to accommodate these distal impairments.

Supported and Unsupported Upright Sitting

Sitting on a chair will create a more stable situation than standing. Sitting on a chair provides a large BoS that encompasses the person's feet as well as the feet of the chair. Likewise, the CoM is lower in a seated position compared with standing. Thus the lower CoM with the larger BoS creates a more stable circumstance. Sitting can be made relatively less stable, however, by reducing the BoS. Lifting one or both feet from the floor or reducing the BoS of the chair can create an unstable environment. As an example, sitting on a gym ball creates a more unstable environment than sitting in a chair.

Sitting on a fixed surface is inherently more stable than upright standing. Although unsupported sitting (without the trunk supported) also requires greater postural demand than supported sitting, standing still requires greater postural demands than even unsupported sitting. In seated positions most of the sway is observed in the anterior-posterior direction. The system is modeled as a single inverted pendulum with the axis of rotation at the hip, such that the trunk and upper torso are considered a single rigid unit.[18,19] Recent evidence on seated postural control demonstrates that

sitting has significantly less postural sway, and compensatory responses are delayed compared with quiet standing.[18–20] The reduced postural sway in a seated position may be attributed to several factors: (1) greater cutaneous feedback from the body segments in contact with the chair, (2) the larger BoS during sitting, and (3) the relatively smaller distance between the COM and pendulum axis. When standing, the distance for determining stability is between the CoM (around the midsacrum) and the axis at the ankle. During sitting, the distance for determining stability is between the CoM at midsternum and the axis at the hip joint.

Additionally, critical vestibular and somatosensory information is perceived in sitting through perturbations to the head and neck. The direct connection to both the visual and vestibular systems and rapid relay of information regarding position and movement of the head in relation to the body may result in more rapid responses to both internal and external moments.[21]



Patient Application 13–3

The relationship between the visual, vestibular, and somatosensory systems within the cervical spine is a relatively new area of research in the presence of chronic neck pain, concussion, cervicogenic dizziness, and whiplash associated disorder (WAD). Individuals who have sustained injuries to the cervical spine demonstrate balance deficits and impaired proprioception in the cervical spine. They often complain of a sensation of dizziness and deficits in balance or postural control. Treatment that incorporates specific muscle training to the postural control muscles of the neck has been found to improve these deficits and symptom complaints.[15]

Control of Posture During Movement

Sometimes, movement of the body is anticipated (planned and controlled), such as when an individual reaches up to a shelf, bends over to pick something up, or walks to

the kitchen. At other times, movements are unplanned, such as when an individual is pushed from behind, trips on a crack in the sidewalk, or experiences sudden acceleration while standing on a subway train. As with control of static posture, postural control is required during both planned and unplanned movements. Recent evidence suggests that anticipated and planned movement strategies involve multiple stages of motor adaptations and muscle synergies that are directly related to the pre-planned movement and are more complex than just maintaining an upright posture.[22–24] APAs are designed to maintain postural stability and occur approximately 100 ms prior to the planned movement.[22–24]

Anticipatory synergy adjustments (ASAs) are another motor strategy that are present during planned movements, and they occur even earlier than APAs.[25] These synergies generally have two distinct patterns. One is designed to maintain the motor output that is predictive of the intended task or action, and the other allows for disruption of the movement. ASAs are motor synergies or outputs that occur about 250–300 msec prior to the planned movement.[25] Various neurological conditions will impact this discrete control of motor synergies and may result in poor postural control or visible motor coordination deficits.



Expanded Concepts 13–1

Quiet Stance

Although quiet stance is considered automatic, newer research has demonstrated that cortical activity precedes postural (feedforward) muscle reactivity.[26] In examining an individual's posture during movement from rest, deviation from ideal and increased muscle activation may indicate a deficit in postural control and may require subsequent examination.

Expanded Concepts 13–2

Postural Strategies for Protecting Injured Tissue

Acute musculoskeletal injuries are associated with altered muscle activation patterns such that muscles are activated earlier to protect the injured tissue. If these motor strategies are maintained for prolonged periods of time they can become problematic to the joint because of the increased load through the joint and decreased movement and motor variability. This has been demonstrated in individuals with chronic musculoskeletal conditions like low back pain, neck pain, shoulder impingement, lateral epicondylitis, and hip and knee osteoarthritis.[27]

Foundational Concepts 13–1

Summary of Posture and Balance

- *Posture:* The orientation or alignment of the human body in space. Posture can be static, (i.e., the body staying still such as in standing or sitting) or dynamic (i.e., the body is in motion such as in walking or running).
- *Balance:* The maintenance of the body in an upright posture, requiring sensorimotor, visual, or vestibular feedback.
- *Postural control:* The act of maintaining, achieving, and restoring the body's balance.
- *APAs:* Specific trunk and limb postural muscle activations that occur prior to planned or anticipated movements, such as reaching for a cup on a table. APAs prepare the body for the planned motion and occur approximately 100 msec before the movement occurs.
- *CPAs:* CPAs are muscle activations that occur when balance is disturbed to restore balance or reduce movement errors in the direction of a movement. CPAs respond to feedback (e.g., visual cues, and joint and muscle sensory receptors) for accuracy.
- *Anticipatory synergy adjustments (ASAs):* A motor strategy that occurs prior to APAs when the movement is known to the individual. These muscle activations seem to have two purposes: (1) maintain the movement that is intended for the task or action, and (2) provide muscle activation that allows for movement disruption (prepare for perturbations). These occur approximately 250 to 300 msec ahead of the planned movement.

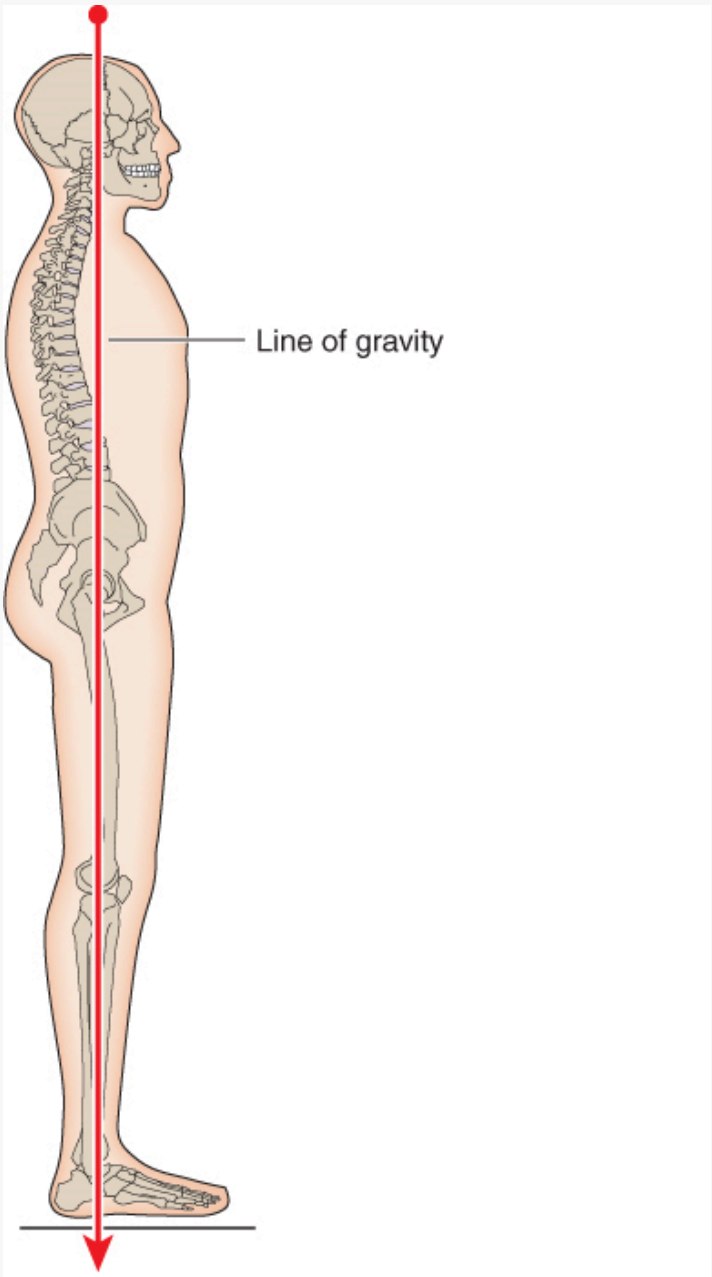
Kinetics and Kinematics of Posture and Postural Control

When examining posture and postural control we need to consider the concepts of kinetics and kinematics and their influence on the musculoskeletal system. Forces acting on the human body are generally described as occurring internally or externally. The muscle responses previously described for balance and postural control are examples of internally generated forces. As a reminder, internal forces are those generated from within the body and can be produced by active muscle contraction or passive tissue tension (e.g., resting muscle tone, passive muscle stiffness, and tension in joint capsules and ligaments).[28] External forces are those that arise from outside the body or body segments such as gravity (Figure 13–5). Other examples of external forces include objects carried in the hand, the application of physical contact from the environment (e.g., floor, bed), and physical interaction with another individual. During normal day-to-day activities, small perturbations and postural sway will result from the application of gravity as a constant external force that acts at the CoM of the body or its segments. In a static standing or sitting posture, the net forces and moments of the internal forces from active muscle and passive connective tissue are responsible for offsetting these external forces and moments imposed by gravity and other objects to maintain an upright position with the head and eyes oriented to the horizon.

FIGURE 13-5

Location of combined action line formed by ground reaction force vector (GRFV) and the line of gravity (LoG) in the optimal standing

posture.



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Patient Application 13–4

Individuals who have sustained a cerebrovascular event (i.e., stroke) and subsequent brain damage with loss of muscle control on one side of the body often demonstrate

an inability to maintain upright posture. This lack of postural control is because of the inability to generate active internal muscle forces sufficient to offset the external gravitational forces. Whenever a therapist examines an individual, all the potential factors that may contribute to postural abnormality should be considered.

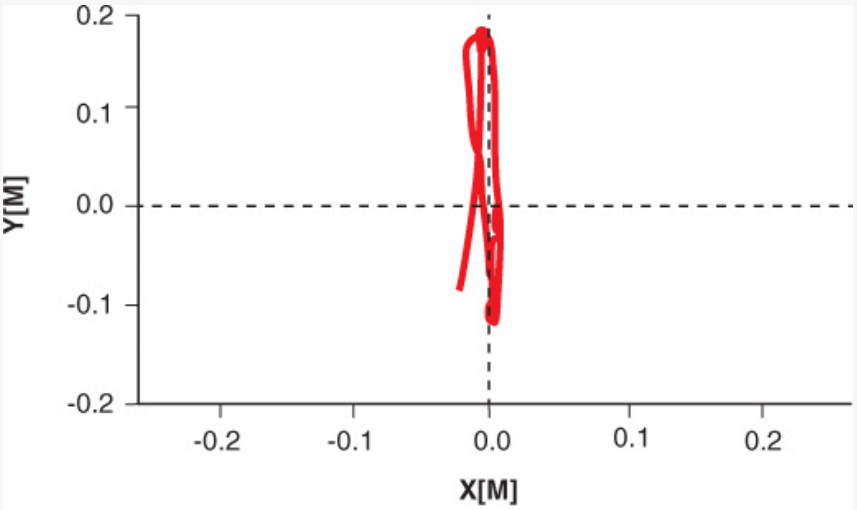
Ground Reaction Forces

When the body comes into contact with the ground, or a muscle acts across a joint to produce joint compression, there is an equal and opposing force generated. At the joint, the force is called a **joint reaction force (JRF),** whereas the force produced by the ground in stance or during gait is called the **ground reaction force (GRF).** This force has three directional components corresponding to each of the cardinal planes.

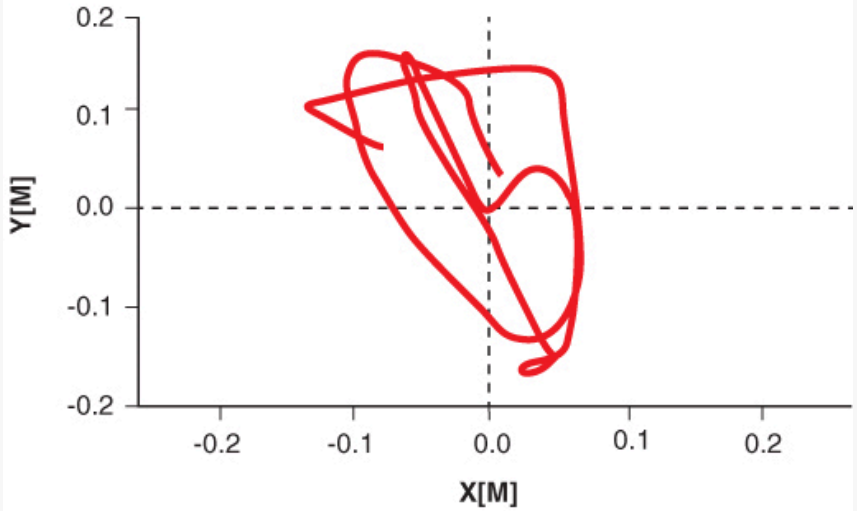
Measurement of the ground reaction force can be done through three-dimensional plotting of the forces produced on a force transducer or force plate.[29] Many of the studies on postural control and balance use force plate measurements related to the **center of pressure (CoP).** Although the entire foot is making contact with the ground, we can estimate a single point of application that represents the sum of all the contact pressures. The CoP is therefore a calculated (theoretical) point of application on the contact surface. The CoP measurement has been used to imply the relative position of the **ground reaction force vector (GRFV)** and position of the CoM in upright posture. The path or map of the CoP in a time series is used to represent the movement of the CoM during postural sway (Figure 13–6). In quiet stance, the GRFV and the LoG have coincident action lines that create equal and opposite external forces on the joints of the human body (see Chapter 1).

FIGURE 13-6

Path of the center of pressure (CoP) in erect stance. **A.** A CoP tracing plotted for a person standing on a force plate. The tracing shows a normal rhythmic anteroposterior sway envelope during approximately 30 seconds of stance. **B.** A CoP tracing showing relatively uncontrolled postural sway.



A Controlled A-P sway



B Uncontrolled postural sway

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Joint reaction forces occur at the joint as a result of the combined internal and external forces imparted across the joint. Muscle activation, and in particular, co-activation by antagonist muscles, is the largest internal force, whereas gravity is the largest external force influencing JRFs. An overall increase in JRFs may contribute to common pain

complaints in conditions involving the lower extremity, such as osteoarthritis.[30–32]

External and Internal Moments

The overall influence of the internal and external forces acting on the body segments during standing or sitting is determined by the location of the LoG from the segment's CoM, in relation to the joint axis of rotation. When the LoG passes directly through a joint, no moment is created. However, if the LoG passes at a distance from the axis of rotation, an external moment is created. Rotation of that joint will occur unless it is opposed by an internal moment created by passive tissue tension or muscle contraction. The magnitude of the external or internal moment is dependent upon the magnitude of the applied force and the moment arm. As a reminder, the moment arm is the perpendicular distance between the joint axis of rotation and the applied force vector (see Chapter 1). The direction of the external moment depends on the location of the LoG with respect to the joint axis.



Example 13–2

If the LoG is posterior to the knee joint axis, then gravity is pulling the femur posteriorly on the tibia (flexion), creating an external knee flexion moment. To maintain equilibrium, the body must generate an equal and opposite internal extension moment (arising from a quadriceps contraction). This is easily experienced by performing a partial squat, in which the LoG is now posterior to the knee joint axis (i.e., external knee flexion moment). If you try this yourself, you should be able to feel the quadriceps become active to maintain this position. In such a static posture, the sum of the internal and external moments is zero.



Foundational Concepts 13–2

Key Points Related to Postural Kinetics and Kinematics

- GRFs are the equal and opposing forces acting on the body or body segments during contact with the ground.
- CoP measurements are theoretical points of application of the ground reaction force and the GRFV that are used to measure postural control and sway.
- LoG and GRFV are coincidental vectors that are equal and opposing in static upright posture.
- The moment arm is the perpendicular distance from the joint axis of rotation to the force vector.

Role of Postural Assessment/Initial Alignment in Physical Therapy

The clinical examination of individuals in any health-care setting generally begins with a visual inspection. In physical therapy this visualization often begins with an observation of the patient's posture. For instance, the therapist should take note of how a patient is lying in bed or seated in a chair when the therapist approaches, or how the patient moves from a chair to approach the therapist. This initial inspection is used early in the examination process to assist in formulating a clinical hypothesis.[33] In particular, therapists often use visual inspection of posture to ask three primary questions: what can the individual do, how does he/she do it, and why does he/she do it that way?[33] The answers to these questions come from observing the individual's alignment as well as the movements of body segments relative to each other. Comparing what is observed with what is expected for normal alignment and movement strategies can inform clinical decision making.

Postural control is also used as a predictor for motor and cognitive development in maturing infants and children.[34] Assessing movement variability during various postural tasks can assist in determining interventions for infants and children.[34] An

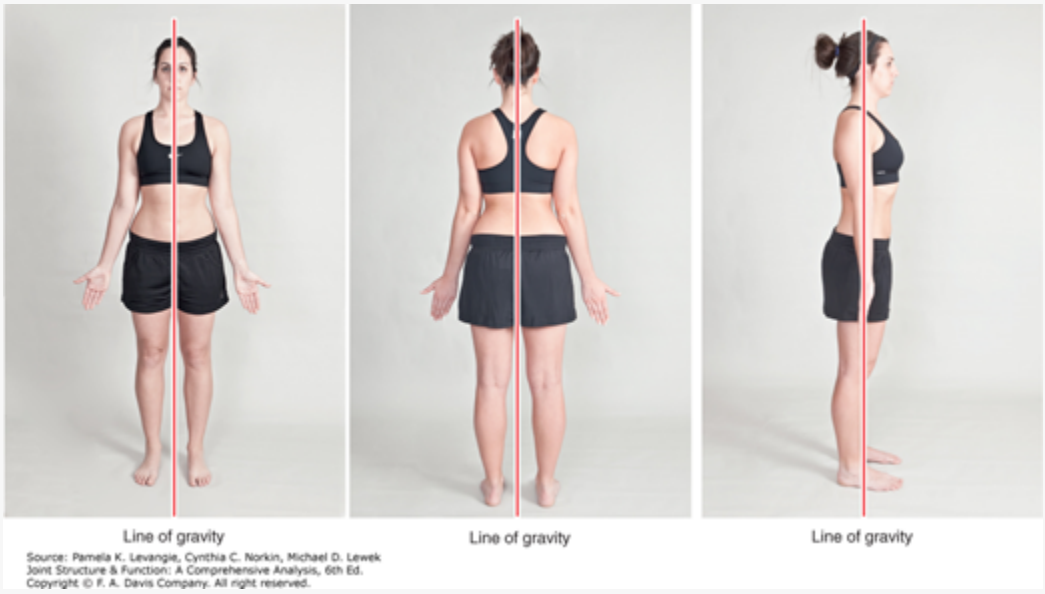
assessment of postural sway and CoP measurements can also be used as determinants for all risks in various populations.[35, 36] Furthermore, an assessment of resting posture can help identify potential causes for painful musculoskeletal conditions.[37,38] Observation of postural control and resting alignment is a fundamental skill that all clinicians interested in human movement should develop in their practice with a thorough understanding of ideal alignment as an important first step.

Ideal Standing Alignment

Ideal upright posture is a position in which all the body segments are aligned relatively vertically, and the LoG passes through or as close to the joint axis as possible (Figure 13–7). The closer the LoG lies to the joint axis of rotation, the smaller the external moment will be and hence the smaller the opposing internal moment will need to be. This is important for maintaining an upright posture with as little energy expenditure as possible. A position in which the LoG passes directly through *all* joint axes, however, is essentially impossible to attain because of the inherent variability in joint shape and structure. Optimal posture, therefore, is the position in which internal moments are minimized by having the external moments be as small as possible. This position can vary based upon anthropometric characteristics such as height, weight, age, and gender.[39,40] Because large deviations from optimal alignment may increase strain on passive supporting structures or require high levels of muscle activation, it is important to identify and remediate these deviations.

FIGURE 13-7

Ideal upright posture. All body segments are aligned relatively vertically and the LoG passes through or as close to the joint axis as possible.



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Analysis of Standing Posture

The most clinically used postural assessment is visual inspection of the body with respect to a vertical line corresponding to the LoG or a plumb line (a line with a weight on one end). This observation is typically done from both sagittal and frontal views. Use of visual assessment has been shown to have limited inter-rater reliability so in clinical practice it may be best to use photographic assessment whenever possible.[41] Such measurements are particularly helpful for determining angular orientation of the spine and extremities.[8,42] Photographs may also be useful to demonstrate changes in posture either with progression of disease, maturation, or treatment effect.

In general, when viewing an individual from the side, the LoG should bisect the body

into relatively equal anterior and posterior parts. The visualization of certain anatomical bony landmarks will assist in observation of postural orientation relative to this LoG.[43,44] Although normal postural sway means that the relationship of the LoG to the body segments is not fixed, this text will use a single description to identify ideal orientation of body segments. From the side, the LoG will fall (1) just anterior to the ear or be aligned with the mastoid process of the temporal bone, (2) just anterior to the acromion, (3) through the midline of the ilium bisecting the anterior and posterior superior iliac spines (ASIS, PSIS), (4) through the greater trochanter, (5) slightly anterior to the femoral condyle (posterior to the patella), and (6) anterior to the lateral malleoli. When viewing the individual from the front or back, the LoG should bisect the body into equal and symmetric left and right halves.[39] The head should be vertical with eyes level, and the shoulders (clavicles), pelvis (iliac crests), hips (greater trochanters), and knees (patella) should be at equal heights, with the weight distributed evenly through both feet.

Analysis of Standing Posture: Side View

Orientation and angulation of the head and spinal column is often observed in a sagittal view. Mean sagittal plane angles of the spine by age group are shown in Table 13–2. Important measurements include the following, which are shown in Figure 13–8:

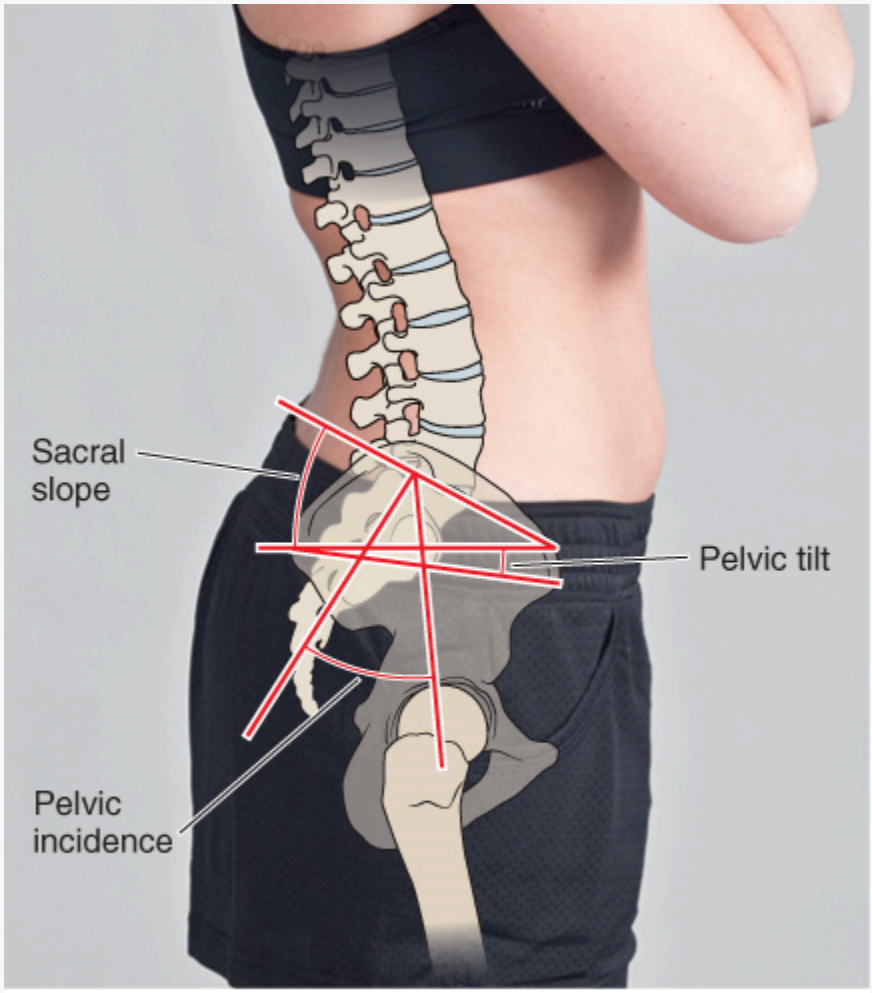
Pelvic incidence: A line drawn from the hip axis to the midpoint of the sacral endplate, and a line perpendicular to the center of the sacral endplate

Sacral slope: The angle created by a line drawn parallel to the sacral endplate and a line from the horizontal

Pelvic tilt: The angle between the horizontal and a line drawn between the PSIS and ASIS

FIGURE 13-8

Pelvis incidence angle, sacral slope, and pelvic tilt are illustrated to demonstrate various methods of measuring the orientation of the pelvis.



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Table 13-2

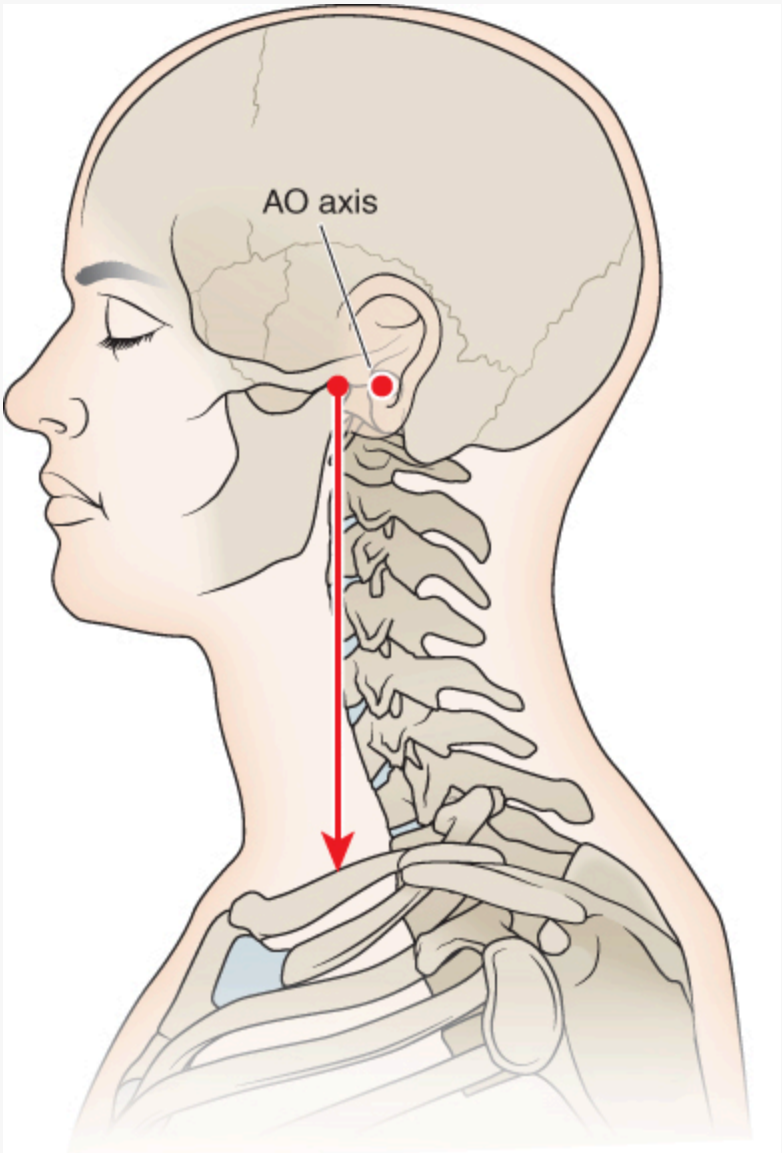
Mean Sagittal Plane Angles of the Spine by Age Group[45,46]

Head

In ideal alignment, the head should be positioned over the shoulders so that the ear is in line with the clavicle. The LoG will pass just anterior to, or directly through, the external auditory meatus (ear canal), approximating the mastoid process of the temporal bone (Figure 13–9). This is slightly anterior to the axis of rotation at C1 to C2, creating an external flexion moment of the cranium on the upper cervical spine. The eyes should be angled slightly above the ear, horizontally. This angle is known as the *sagittal head angle* and is calculated by a horizontal line passing directly through the external auditory meatus and a line that passes from the lateral corner of the eye (Figure 13–10).[47] The head should be aligned directly over the CoM of the body, which is just anterior to the S2 spinal segment.[45] Maintenance of the head in this position is provided by a balance between the external flexion moment produced by gravity and the active muscle force of the deep cranio-cervical extensors.

FIGURE 13-9

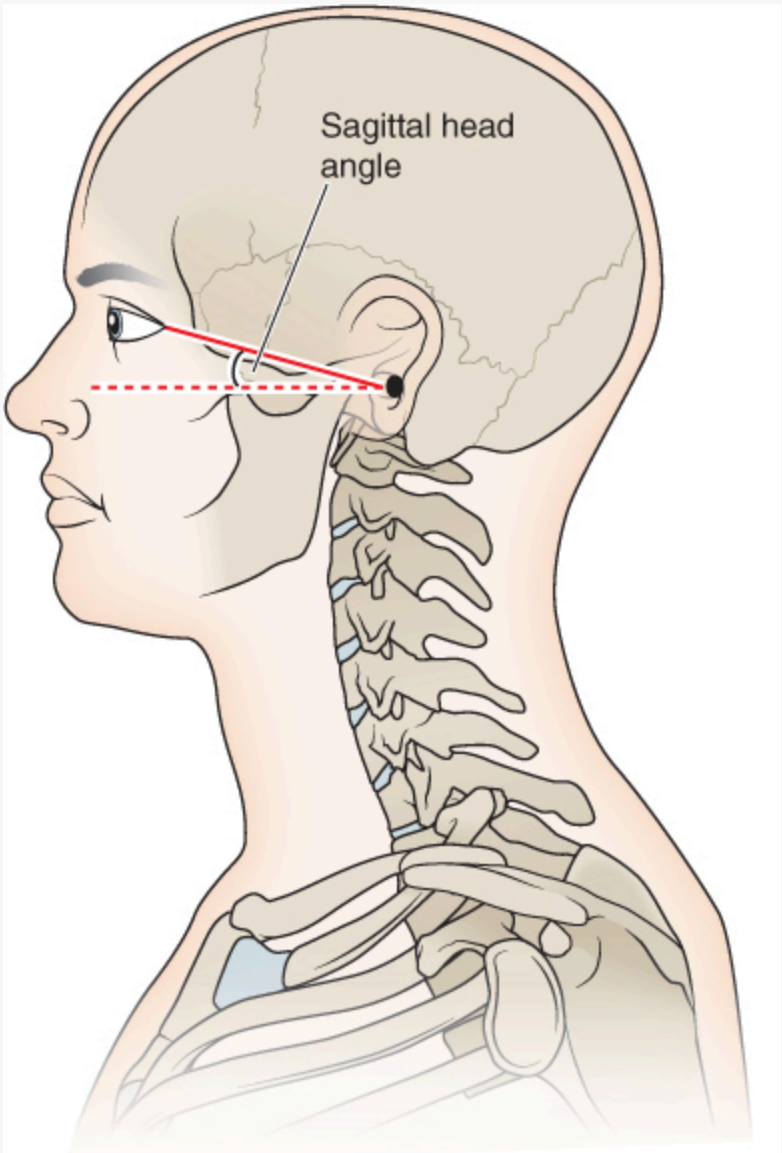
Ideal head alignment showing the ear aligned with the clavicle.



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
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FIGURE 13-10

Sagittal head angle gives an indication of alignment of the head in space.



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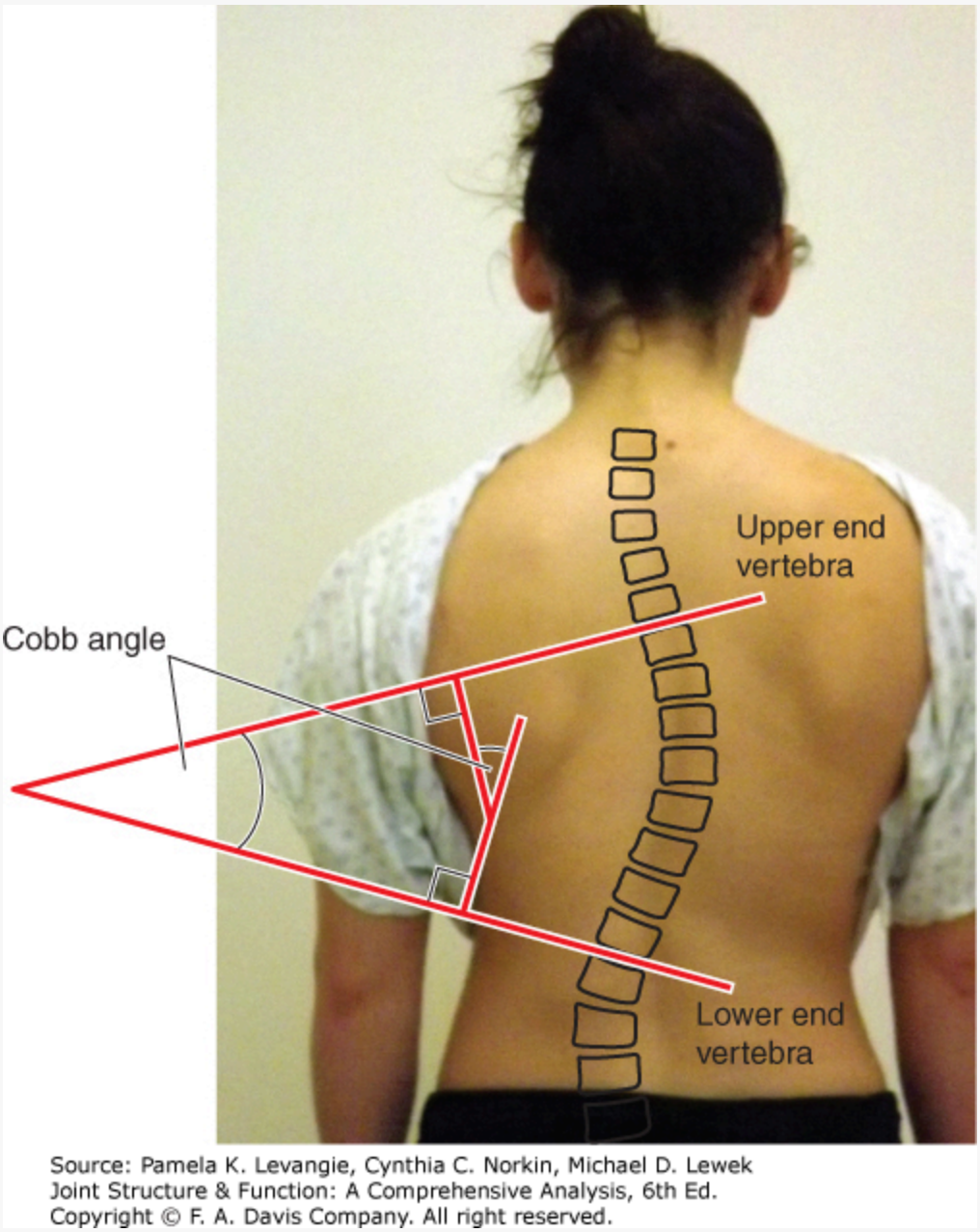
Vertebral Column

The vertebral column should demonstrate varying degrees of lordosis and kyphosis from the cervical spine to the lumbar spine (see Fig. 4–1 in Chapter 4, The Vertebral Column). Quantification of these spinal angles is commonly described using **Cobb angles,** which are determined by measuring region-specific angulations between

vertebral endplates on radiographic images (**Figure 13–11**).[48] Mean averages of spinal region angles are listed in Table 13–2. The degree of spinal curvature at each spinal region is interdependent. For example, an increase in the angle at the T1 vertebral endplate will increase the degree of cervical lordosis. An increase or decrease in the inclination of the pelvis or sacrum will also increase or decrease lumbar lordosis.[49,50] Deviation from normative values outlined in Table 13–2 has been demonstrated in many painful spinal conditions and may be associated with deficits in health-related quality of life.[51] Visual deviations of curvature may therefore warrant further radiographic testing, as the restoration of normal angles is the goal of many spinal surgical interventions.

FIGURE 13-11

Cobb angle.



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In the frontal plane, the LoG should pass directly midline of the trunk. In the cervical and lumbar spine the LoG of the head and trunk passes slightly posterior to the joint axes with the spine in lordosis, and in the thoracic spine it passes just anterior to the joint axes with the spine in kyphosis. Table 13–3 shows the change in stress on the intervertebral disc and force from muscle activation between a neutral and anteriorly

translated posture.

Table 13-3

Percent Increase in Stress of the Intervertebral Disc and Muscle Activation Between Neutral and Anterior Translated Posture[52]

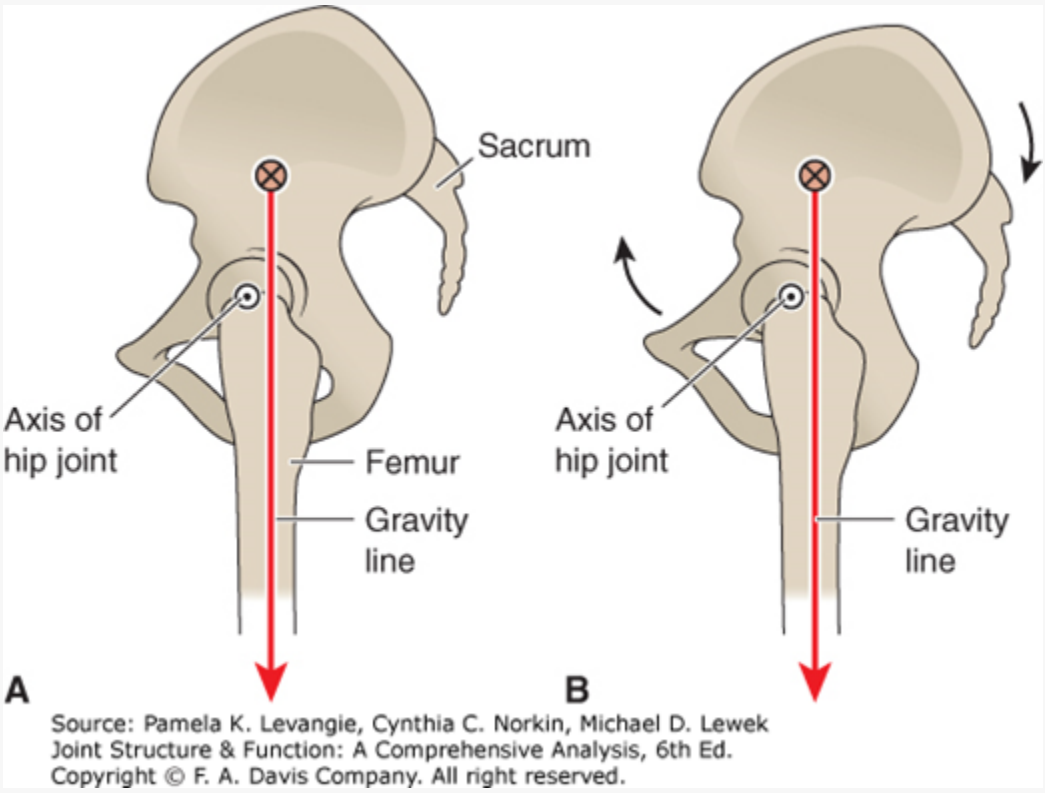
Pelvis and Hip

Sagittal alignment of the pelvis and sacral slope have been shown to predict orientation of the cervical, thoracic, and lumbar spine.[3,45,50,53] In an ideal upright posture the LoG should pass just anterior to the sacrum (S1 on spinal radiograph) and slightly posterior to the axis of rotation (femoral head) of the hip joint.[39] Although often difficult to palpate and visualize, the anterior superior iliac spine (ASIS) of the pelvis is described as being slightly lower than the posterior superior iliac spine (PSIS), positioning the pelvis in a slight anterior tilt.[54] The position of the LoG, therefore creates an external flexion moment on the sacrum (nutation) and external extension moment at the hip.[55] In normal quiet standing, this external hip moment is offset by muscle activity of the hip flexors (Figure 13–12).[56]

FIGURE 13-12

Ideal alignment, line of gravity, and internal moments at the hip joint. **A.** The LoG passes through the greater trochanter and posterior to the axis of the hip joint. **B.** The posterior location of the LoG creates an

external extension moment at the hip, which tends to rotate the pelvis posteriorly on the femoral heads. The arrows indicate the direction of the gravitational moment.



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Knee

At the knee, the LoG passes slightly anterior to the joint axis (femoral condyle), and posterior to the patella, creating an external knee extension moment.[55] The small external moment arm requires very little activation of hamstring muscle activity, but may be controlled by the muscle activation of the gastrocnemius given its proximal insertion.

Ankle

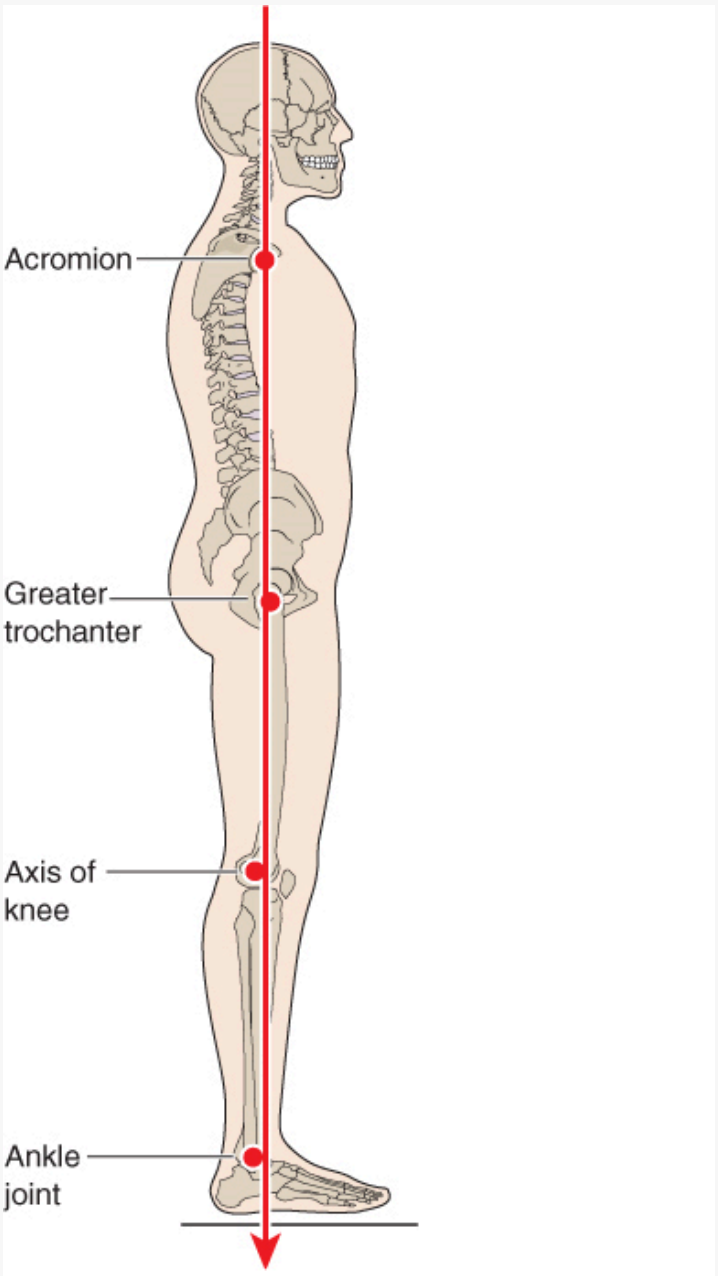
In optimal upright stance, the ankle joint is in neutral position between plantarflexion

and dorsiflexion. The LoG passes just anterior to the ankle joint axis of rotation (lateral malleolus).[57] This external moment arm creates an external dorsiflexion moment, which is offset by the internal muscular force of the gastrocnemius/soleus muscle group.[55,56] The soleus demonstrates consistent activity, and contains many Type 1 muscle fibers, indicating its role as a postural stabilizer. In addition, the biarticular design of the gastrocnemius, with its proximal attachment originating proximal to the knee joint axis, allows the gastrocnemius to facilitate an internal plantarflexion moment at the ankle and internal knee flexion moment simultaneously.[58]

Figure 13–13 shows the ideal upright posture (side view) with bony landmarks. Table 13–4 outlines the alignment of the sagittal plane in upright standing.

FIGURE 13-13

Ideal upright posture with bony landmarks.



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Table 13-4

Alignment in Sagittal Plane in Upright Standing[3]

Analysis of Standing Posture: Frontal Plane

In viewing posture in the frontal plane from either an anterior or posterior view, the LoG should divide the body into two equal symmetrical halves with equal height and position of body segments. In both the anterior and posterior views, the head should appear straight with no tilting or rotation evident and the face should appear to be bisected into equal halves. The eyes, ears, clavicles, and shoulders should be level and horizontal.

From the posterior view, the scapulae should rest flush to the thorax, between the level of the second through seventh ribs, with the inferior angles at equal heights and equidistant from the vertebral column. The iliac crests should be level and parallel to the floor. Both the anterior and posterior superior iliac spines should be level and equidistant from the LoG as well as the joint axes of the hips, knees, and ankles. When viewing each body segment, the femurs and tibia should appear to be vertical, the patellae at equal heights and facing anteriorly, and medial and lateral malleoli at equal heights to each other, with the feet directed to the front. In the frontal plane, stability is maintained by a balance between the left and right sides as well as muscle forces on the medial and lateral sides of the limbs.[58,59]

Tables 13–5 and 13–6 outline the alignment of standing posture in the anterior and posterior views.

Table 13-5

Alignment of Standing Posture: Anterior View

Table 13-6

Alignment of Standing Posture: Posterior View

Foundational Concepts 13–3

Postural Assessment

Postural assessment in the clinic is done from the side and from both the front and back. In a side view the LoG should bisect the body into equal anterior and posterior divisions of the body. In both the front and back views, the LoG should separate the body into two symmetric halves. Using a photograph increases the reliability and accuracy of postural measurements as well as allows for tracking change over time. Radiographic images are more accurate and provide for use of quantifiable measures of spinal curvatures. However, exposure to the radiation from radiographs may not be desirable.

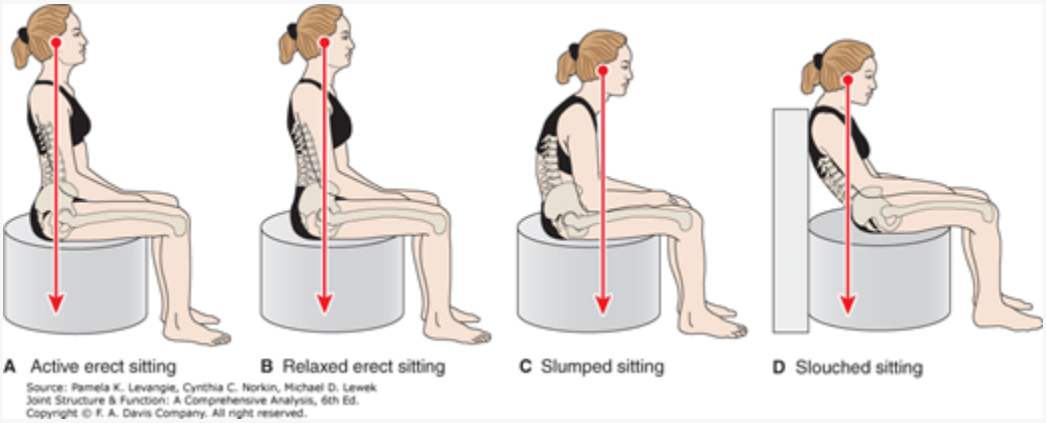
Ideal Sitting Postures

Sitting posture is influenced by the surface on which the individual is seated. The overall goal of sitting posture is to attain a stable alignment of the body that can be

maintained while minimizing energy expenditure and stress to body structures. There are many different sitting postures; four common ones are shown in Figure 13–14. **Active erect sitting posture,** which is unsupported posture in which a person attempts to sit up as straight as possible, is often considered the ideal seated posture. This posture affects forces differently than **relaxed erect, slumped,** and **slouched** sitting postures. In a way, sitting postures are more complex than standing postures. The same gravitational moments as in a standing posture must be considered, but in addition, we must consider the contact forces that are created when various portions of the body, such as the head and back, interface with various parts of the chair, such as the foot rest and seat back. The location and amount of support provided to various portions of the body by the chair or stool may change the position of the body parts and thus the magnitude of the stresses on body structures.

FIGURE 13-14

Common seated postures.



Although the ideal seated posture is often advocated, there is limited quantitative data to support this position.[60] The LoG passes close to the joint axes of the head and spine

in active erect sitting posture. In the slumped posture, the LoG is more anterior to the joint axes of the cervical, thoracic, and lumbar spines than it is in either active or relaxed erect sitting. Therefore, it might be assumed that more muscle activity is required in the slumped posture than in the other sitting postures. In contrast to these expectations, researchers have found that maintaining an active erect sitting posture requires a greater number of trunk muscles as well as an increased level of activity in some of these muscles compared to both relaxed erect and unsupported slumped postures.[61] The **flexion relaxation (FR) phenomenon** may provide a possible reason why the slumped sitting posture requires less muscle activity than does the active erect sitting posture. Flexion relaxation is a cessation of muscular activity, as manifested by electrical silence of the back extensors during trunk flexion in either sitting or standing postures.

Seated posture in a standard office chair with back support has been shown to result in an increase in posterior pelvic tilt posture with a flattening of the lumbar lordosis when compared with unsupported sitting on an anteriorly tilted seat.[62] Interestingly, despite the presence of a back support, sitting in the office chair resulted in an increased amount of postural sway and muscle activation. This finding of increased muscle activation in the back-supported position is interesting, as most other studies on seated posture and pain have examined the influence of unsupported sitting and therefore may not capture the demands placed on the spine in a standard office chair.[61,63,64]



Patient Application 13–5

Low back pain is one of the leading causes of disability in the United States.[65] Many individuals with low back pain report increased symptoms in the seated position. The increase in pain may arise from the altered posture of the lumbar spine and pelvis in

the seated posture compared with a standing posture. For example, a patient who states that sitting in a standard chair increases his/her low back pain may be exacerbating the pain because of a decrease in lordosis. Asking patients about postures that aggravate and ease their pain may lend insight into the underlying cause of mechanical low back pain.

Common Deviations from Ideal Alignment

The Role of Pain

There is an enormous amount of literature on the role of pain in posture and movement both in pathology and in experimental studies performed on healthy individuals. The presence of pain can change the behavior of muscles and lead to altered postures or movements. These altered postures in the presence of pain may be viewed as positive (protective) effects immediately following acute injury or detrimental (compensatory) effects in chronic conditions.[66] For example, it is often easy to identify the painful limb or segment just by observing a person's posture. Pain in a weightbearing limb is often observed as a weight shift away from the painful side. In the spine, pain may be observed as changes in the normal spinal curves. Harrison and colleagues compared the lordosis in the cervical spine between subjects who were healthy (n=72), had acute neck pain (n=52), and had chronic neck pain (n=70).[67] They found that compared with the healthy group, the subjects with pain had reduced lordosis, with the chronic pain group demonstrating the least lordosis.

Changes in the angular alignment of the vertebral column, as in a forward head or slouched posture, are associated with changes in the stress and strain to the lumbar intervertebral disc as well as the muscle activity required to maintain this position.[52,68] Maintaining this abnormal spinal position requires balance between the external

moment produced by gravity, and the internal moment produced by connective tissue structures (passive) or muscle support (active). Importantly, greater increases in the internal moment from muscle activation are associated with increased stress to the intra- and extra-articular structures. Other experimental pain studies have demonstrated an increase in muscle activation of the spinal stabilizers as well as increased postural sway in the presence of low back and neck pain.[69–72] Experimental muscle pain in the lower extremities also alters postural control and challenges postural stability, which may manifest as use of upper extremity support or difficulty transitioning between positions.[73,74]

Scoliosis

A common postural deviation seen in the vertebral column is **scoliosis,** a condition that causes excessive curvature of the vertebral column. Scoliosis is most easily observed in the frontal plane (see Figure 13–10); however, it must be recognized that the curvature is three-dimensional. For example, scoliosis often occurs in the transverse (rotary), sagittal (flexion/extension), and frontal (lateral bending) planes. These cross-planar curvatures occur because spinal facet joints don't lie in a single plane. Thus, a curve in the frontal plane will cause a concomitant change in the transverse plane. This is the basis for the classically described *rib hump* that is often used for detecting the initial presence of scoliosis (**Figure 13–15**). Curves are conventionally named by the direction of the convexity and the location of the abnormality, such that a right thoracic scoliosis would indicate that the peak of the convexity occurs on the right side of the vertebral column in the thoracic region (**Figure 13–16**). Scoliosis may be structural or functional in nature and can occur at any age, although adult onset is less common. Structural scoliosis is characterized by bony and soft tissue changes that are not reversible.

Structural scoliosis most commonly affects the thoracic spine and results in an ipsilateral rib hump (see Figure 13–15), as well as compensatory opposite curves in the other regions of the spine. Functional scoliosis is associated with some underlying cause, such as a discrepancy in leg length, and can be reversed once the cause is corrected. There are a number of categories of scoliosis, with the distinction made primarily based upon age of onset (Table 13–7). Scoliosis may be idiopathic (no known cause), or because of underlying causes such as neuromuscular, genetic, environmental, or metabolic secondary to growth-modulating chemical factors, with limited evidence to support any one factor.[75]

FIGURE 13-15

Right rib hump on a young woman with scoliosis.



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FIGURE 13-16

Scoliotic S-shaped curve, with a left lumbar curve and a right thoracic curve.



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Table 13-7

Categories of Scoliosis

Spinal Abnormalities

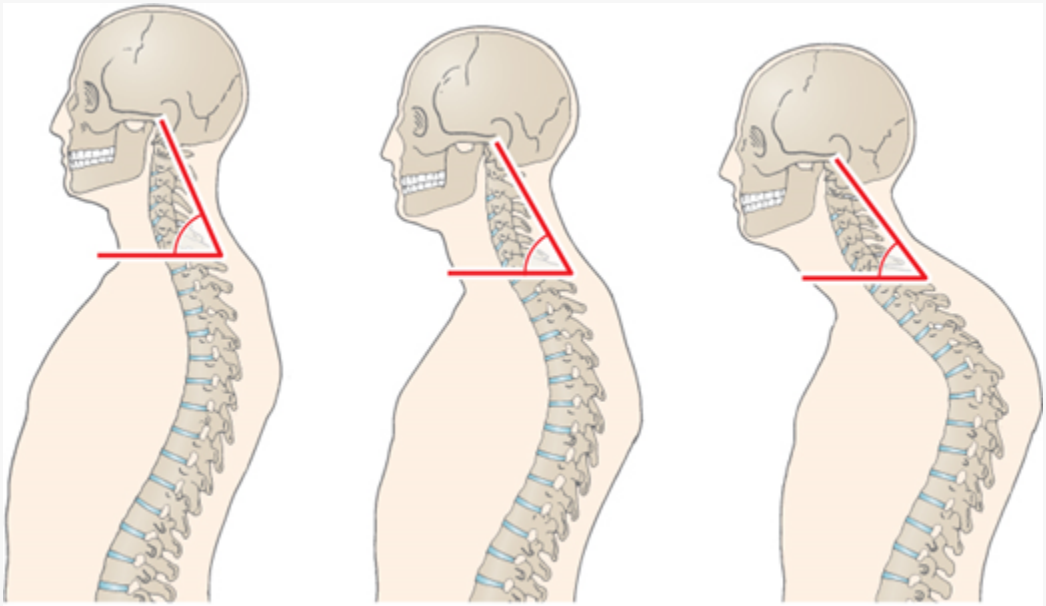
Other common vertebral column disorders are excessive kyphosis, spondylosis, and spondylolisthesis. Although kyphosis is the normal curvature of the thoracic spine, older adults may present with conditions in which the curvature in the thoracic spine is excessive. This condition is called hyperkyphosis and can be associated with or is a result from vertebral compression fractures.[76] **Spondylosis** is a reduction in the intervertebral disc heights and hypertrophy of the zygapophyseal joints, capsule, and ligaments that are commonly associated with aging. Painful disorders, such as myelopathy or radiculopathy, can be associated with spondylosis.[77] Any acute muscular condition associated with the neck or back may also present with a loss of lordosis in either the cervical or lumbar region; however, this is usually short term and not considered to be spondylosis. **Spondylolisthesis** is a condition in which a superior vertebral body slips anterior to the vertebra below it. It may be caused by degeneration of the segments, bilateral fracture of the pedicles of the vertebra, or occur as the result of trauma. Excessive lumbar extension (e.g., as experienced by gymnasts and football lineman) is a common mechanism of such injuries. Typically, this is seen in the lower lumbar segments. This condition can result in the adoption of various lumbar postures, ranging from excessive lumbar lordosis to flattening of the lumbar spine.[78]

Forward head posture in the cervical spine is also very common and tends to be related to prolonged positioning with poor muscular control. It is characterized by the head

being positioned anterior to the shoulder girdle and involves a combination of craniocervical extension, lower cervical flexion, and forward position of the shoulder girdles (Figure 13–17). Measurement of this position has been validated using digital photography, using the angle formed by a line drawn from the most posterior point of C7 to the horizon, and a line from C7 to the posterior aspect of the tragus of the ear.[79,80] Many painful musculoskeletal conditions, such as temporomandibular dysfunction, cervicogenic headache, and mechanical shoulder pain have been associated with this posture.[81–83]

FIGURE 13-17

Forward head posture.



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Shoulder Girdle

Malposition or abnormal movement of the scapula is a common finding among all age groups.[84] This is not surprising given that stabilization of the scapulothoracic joint is

dependent on suitable muscle strength (see Chapter 7). Winging of the scapula is defined as the entire medial border of the scapula not being positioned flush to the thoracic wall, with the scapula in a position of excessive internal rotation (**Figure 13–18**).[85] This position may result from serratus anterior or rhomboid muscle weakness. Excessive anterior tilt of the scapula is identified by the inferior angle of the scapula resting away from the thorax and rib cage, which is often associated with shortened length in the pectoralis minor muscle.[84,86] A number of painful shoulder conditions are related to poor scapular alignment and positioning, so a thorough clinical examination of this region is critical.

FIGURE 13-18

Winging scapula.



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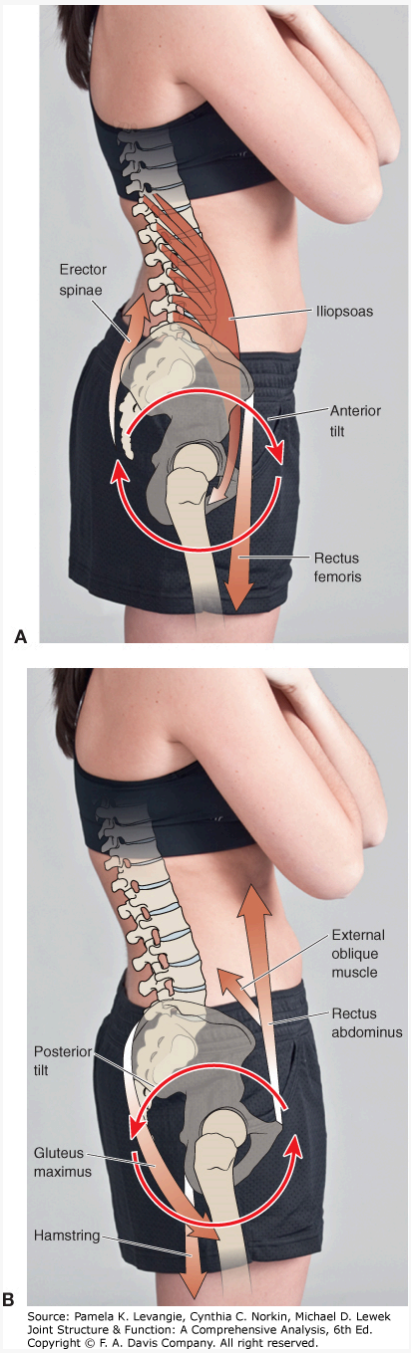
Lumbopelvic and Hip Abnormalities

Excessive anterior or posterior pelvic tilt is often visible in the sagittal plane. Both of these postures can be the result of muscle length deficits, with an anterior tilt associated with reduced length in hip flexor musculature and posterior pelvic tilt

associated with reduced length in the hip extensor musculature (e.g., hamstrings). An anterior pelvic tilt will typically coincide with an increase in lumbar lordosis and hip flexion, placing increased compressive forces on the zygapophyseal joints of the lumbar spine (Figure 13–19).[87] In contrast, an anterior displacement of the pelvis with a concomitant posterior displacement (swaying back) of the upper trunk is often referred to as **swayback posture** (Figure 13–20). Such a posture is marked by an increase in lumbar lordosis and thoracic kyphosis. The corresponding increase in hip extension allows the person to maintain the GRF posterior to the hip joint and use the anterior hip ligaments for support. Swayback posture is often associated with an increase in anterior hip pain and results in increasing compressive forces on the intervertebral disc of the lumbar spine.[37,87]

FIGURE 13-19

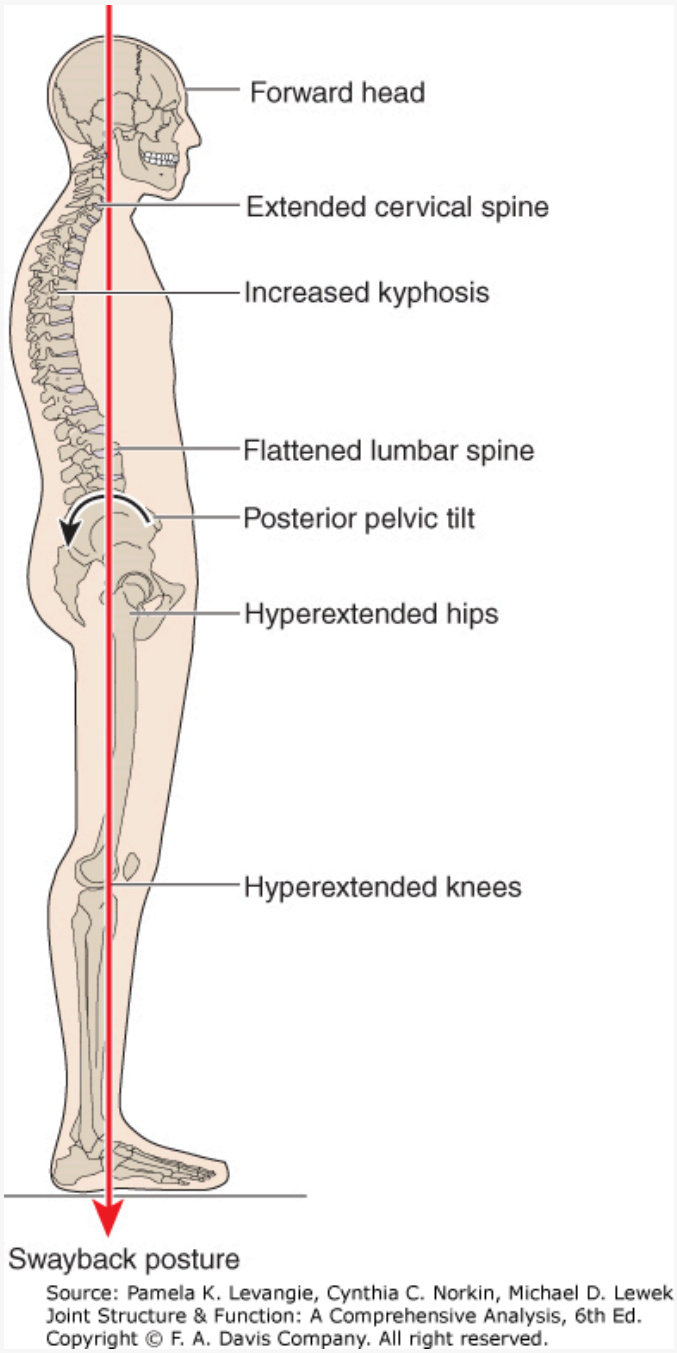
A. Anterior and **B.** posterior pelvic tilt. *(From Houglum P, Bertoti D. Brunnstroms Clinical Kinesiology, 6E. Philadelphia, PA: F. A. Davis; 2012, with permission. From Samuels V. Foundation in Kinesiology and Biomechanics. Philadelphia, PA: F. A. Davis; 2018, with permission.)*



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FIGURE 13-20

Swayback posture.



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Knee

Genu recurvatum (hyperextension of the knee in stance) is a position in which the knee is in ≥10 ° extension (Figure 13–21).[88] In this position, the LoG passes further anterior to the knee joint axis than typical, which places increased tensile stress on the posterior

capsule and ligamentous structures of the knee. There is some evidence that increased laxity of the knee into hyperextension may increase the risk of anterior cruciate ligament (ACL) injuries.[89] Common deviations of the knee in the frontal plane include genu varum and genu valgum (Figure 13–22). **Genu varum** (bow-legs) is a medial angulation of the distal tibia in relation to the femur when the feet are together. It usually involves both the femur and the tibia. Genu varum is a normal finding in children from birth to about age 3.[90,91] in the adult, however, the presence of genu varum is a predictor for increasing risk of the development or progression of medial compartment knee osteoarthritis.[38,92] In this alignment, the LoG passes medial to the knee joint axis, placing greater compression on the medial joint structures. This increased joint loading to the medial meniscus and tibiofemoral joint surfaces may result in physical stresses that exceed tolerance levels, resulting in tissue breakdown as demonstrated by the development or progression of osteoarthritis.[38] **Genu valgum** (knock-knees) is a relative lateral angulation of the distal tibia in relation to the femur. An individual with genu valgum may stand such that the knees are touching each other, but the feet are unable to come together. This angulation is considered normal in children from 3 to 7 years, but should resolve by adolescence.[90,91] Increased valgus angle of the knee places the LoG on the lateral side of the knee joint axis, increasing compression to the lateral joint structures. In individuals with knee osteoarthritis, genu valgum is associated with reduced development and progression of medial compartment cartilage degeneration and more compressive loading on the lateral compartment.[38,93] Valgus angulation during static and dynamic tasks may be associated with increased stress and pain in the patellofemoral joint, as well as increased incidence of ACL injuries.[94–96]

FIGURE 13-21

Genu recurvattum.



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FIGURE 13-22

Common postural deviations at the knee. **A.** Genu varum of the right leg. **B.** Genu valgus.



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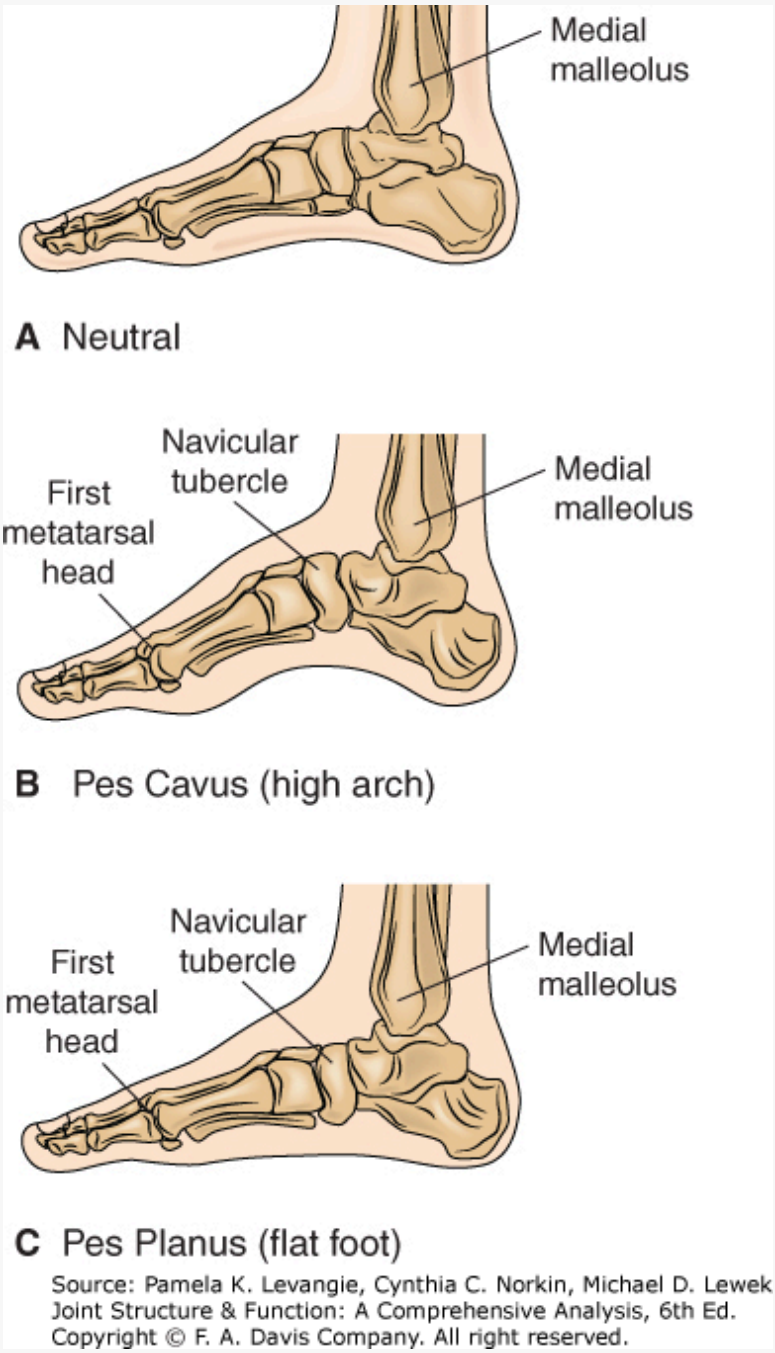
Ankle, Feet, and Toes

Because of the complexity of the foot, postural alignment can be extremely variable. The most common descriptors for foot posture are **neutral, pes planus,** or **pes cavus** (Figure 13–23).[97] There are a number of different methods for classifying these different foot types, but for a postural analysis, only visual inspection is required.[98] A neutral foot is described as a foot that is well aligned both in the rearfoot (calcaneus and talus) and the forefoot. In the neutral position, lines drawn down the midline of the posterior tibia and the posterior calcaneus should be parallel. Pes planus is the most commonly

observed postural abnormality.[98,99] A pes planus foot, also called pronated or flat foot, can consist of either a low arch with a valgus angulated hindfoot (calcaneus), or a low arch with a varus angulated forefoot, or both. Research in the area of foot abnormalities and posture has shown that excessive pronation is associated with lower extremity injuries, increased incidence of low back pain, altered kinematics in gait, and reduced walking speeds and quality of life scores.[98,100–102] These gait deviations may result in increased stress, and subsequent injury or pain to structures further up the kinetic chain. Furthermore, Molines-Barroso and colleagues found in a cross-sectional study of individuals with diabetes and sensory disruption in the foot that those individuals with pronated feet had a higher incidence of ulceration than those with supinated or neutral feet.[103] These studies suggest that visualization of pronated feet may assist in identifying individuals at risk for certain impairments or painful conditions, which may increase their level of disability.

FIGURE 13-23

Common foot postures.



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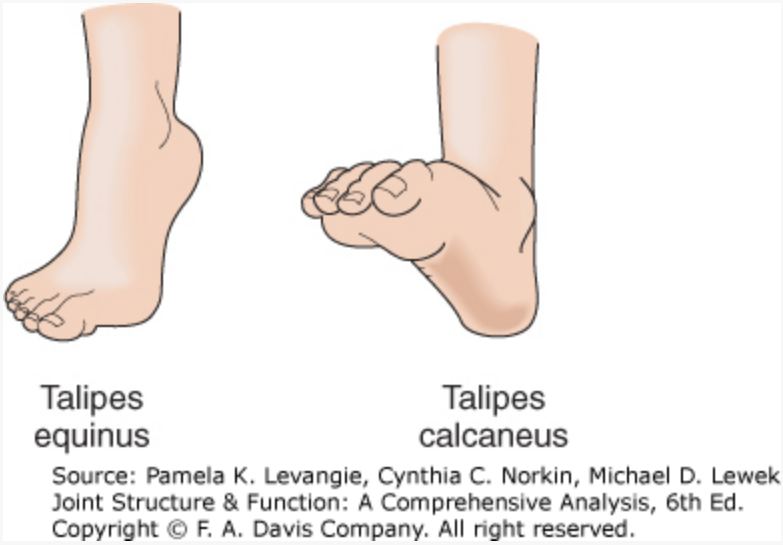
Pes cavus feet are often called supinated, or high arched. The foot structure consists of either a high arch with a varus hindfoot, a high arch with a valgus forefoot, or both (see Chapter 12). Far less literature exists regarding cavus feet than pronated feet; however, there is some evidence to suggest the presence of an increased incidence of ankle

instability and stress fracture with pes cavus feet.[104,105] Kinematic studies of gait have demonstrated that cavus feet in stance demonstrate less motion during loading response and midstance than planus or neutral feet.[101,106] This reduction in motion may result in reduced absorption of ground reaction forces and increased stress to the foot, ankle, and lower limb.

Equinus deformities of the foot are often associated with various neurological conditions and hemiparesis (**Figure 13–24**).[107,108] In children with cerebral palsy, these deformities are associated with significant gait deviations and proximal limb compensations like excessive pelvic rotation, increased hip flexion, and internal rotation.[109] In this population, these compensations result in increased mechanical work and energy expenditure compared with healthy controls.[110] In 49 adults with hemiparesis and equinus foot deformities, Manca and colleagues found that subjects presented with a variety of different gait deviations and speeds.[108] The severity of ankle and foot deformities was associated with slower walking speeds and greater compensatory upper limb motion. The speeds demonstrated by this group of patients were associated with those of individuals who were restricted to household and limited community ambulation. Although the complexity of neurological conditions contributes to these velocity differences, it is useful for a clinician to understand that equinus foot positioning may inhibit gait progression.

FIGURE 13-24

Equinus deformities.



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Abnormal toe alignment in stance is often indicative of bony and soft tissue disorders of the foot (Figure 13–25). Hallux valgus is a common progressive deformity of the first toe, in which the toe abducts or deviates laterally while the first metatarsal deviates medially (Figure 13–25A).[111] It is more common in women than men, but men with pes planus and higher body mass index are at a higher risk than men without pes planus.[112] It is often associated with bunions, which are bony bumps at the base of the first toe that can be painful and often require surgical correction. Other deformities affecting more than just the first toe are mallet toe, hammer toe (Figure 13–25B), and claw toe. These deformities are usually the result of abnormal tension between the intrinsic and extrinsic muscles of the feet resulting in altered joint positions (Table 13–8). Deformities such as these may be the result of trauma, ill-fitting footwear, connective tissue diseases, neuromuscular conditions, or metabolic diseases.[113] Observation of these deformities should be noted as they can result in impaired postural control and may result in abnormal compensatory stresses on the proximal limb.[114] Older individuals with painful foot conditions have a higher risk for falls compared with those without

foot pain.[114]

FIGURE 13-25

Abnormal toe alignments.

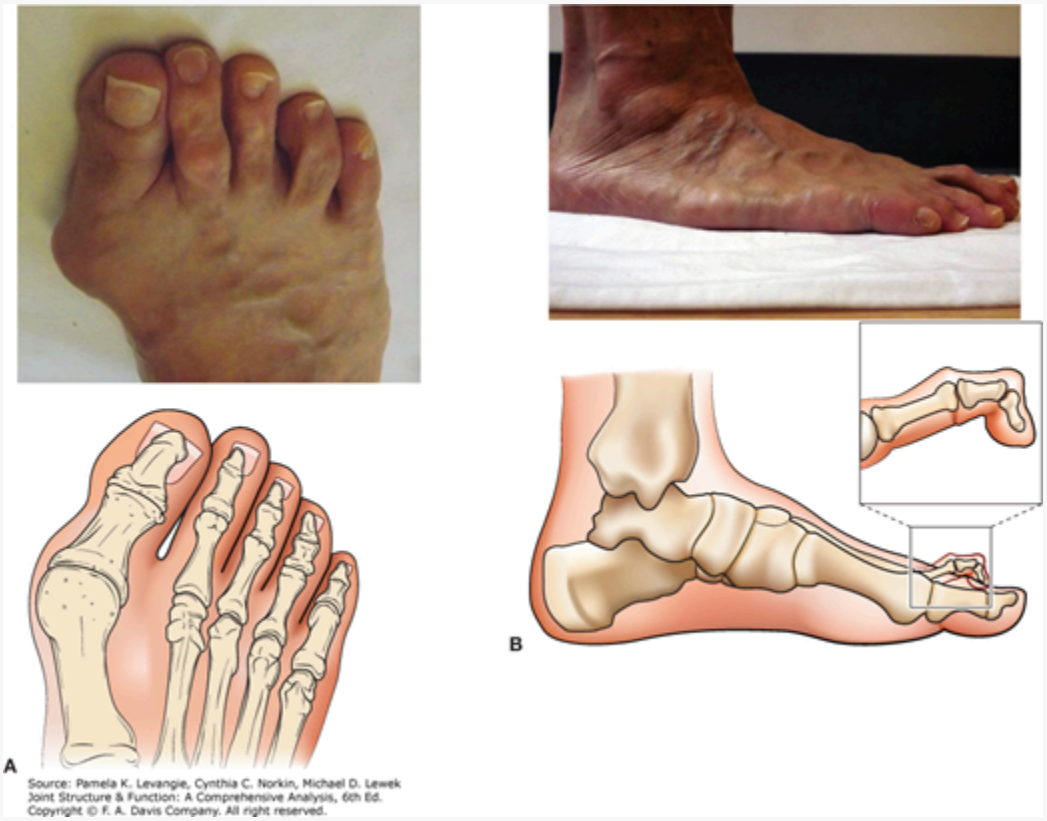


Table 13-8

Abnormal Toe Postures

Neurological Conditions That Influence Posture

Maintaining upright posture relies on the interplay between the mechanical properties

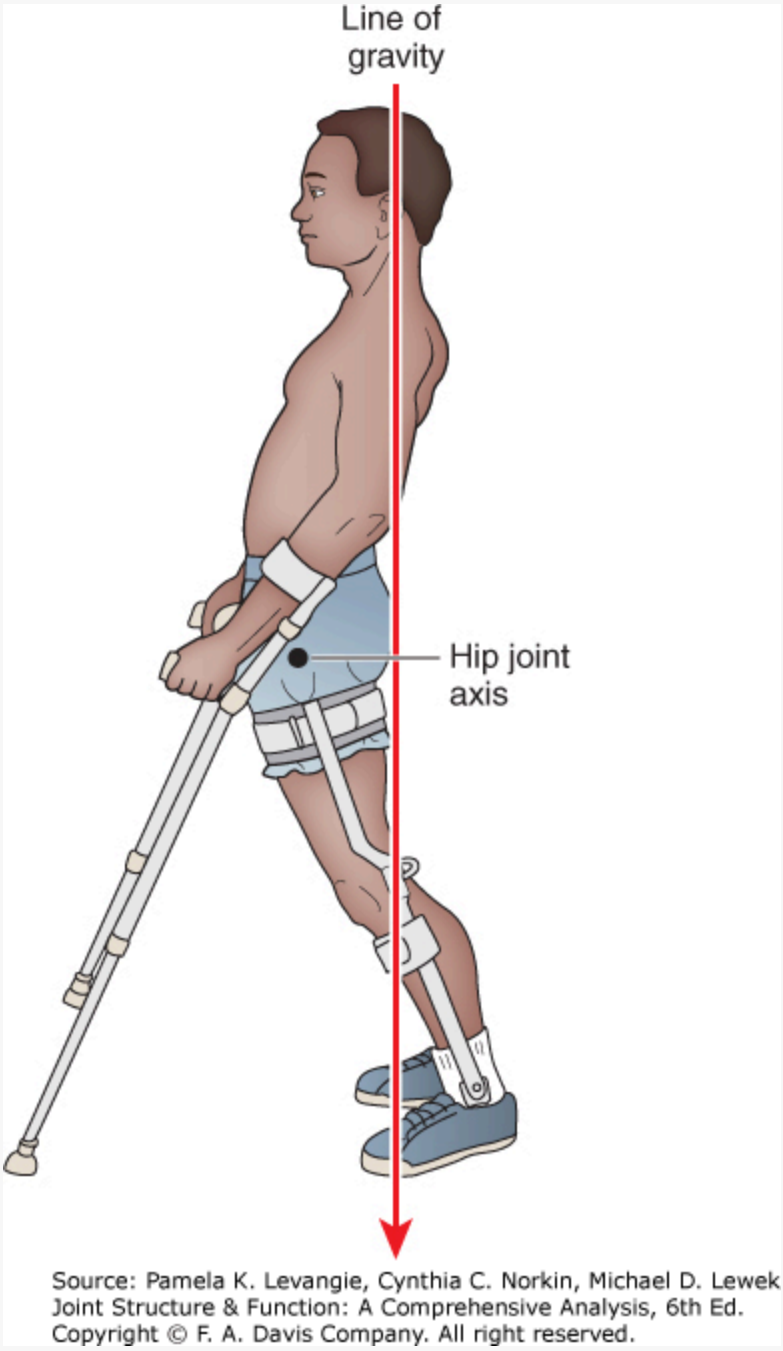
of the musculoskeletal system and integrity of the CNS. Conditions that impact either of these systems will result in altered postural control and abnormal alignment. Hemiparesis is a condition where one side of the body is weaker because of a neurological impairment within the brain, brain stem, or spinal cord. Individuals with either left- or right-sided hemiparesis will find upright alignment and trunk control difficult. Postural alignment may present as a lean or a total body collapse toward the paretic (weak) side. This lean to the paretic side is attributed to muscle weakness because the weakened muscles are unable to counteract the external moment produced by gravity. The lean may be confounded by additional impairments in the sensory perception of the body's position in space.

Injuries or conditions that affect the spinal cord may also result in complete or partial loss of motor or sensory deficits distal to the level of the lesion and reorganization of their visual and somatosensory systems.[115] These individuals may rely on the passive supportive structures of the musculoskeletal system to maintain an upright position. Following an incomplete spinal cord injury, upright stance is often maintained by shifting the pelvis anteriorly such that the LoG passes farther posterior to the hip and farther anterior to the knee joint than normal, creating external extension moments in both (Figure 13–26). This enables the individual to rely on passive internal moments to maintain an upright position. Specifically, an individual may use the large anterior hip ligaments to resist the external extension moment at the hip, whereas the posterior knee capsule and ligaments can be used to resist the external extension moment at the knee. With the LoG passing anterior to the ankle joint, an external dorsiflexion moment is created to allow the passive tension in the Achilles tendon and posterior ankle structures to maintain upright standing. If necessary, additional bracing can be used to

maintain these joint positions to allow standing in the absence of muscle activity. In the seated position, injuries above the thoracic region will often result in a flexed trunk posture, such that stability arises from the passive stiffness of the posterior spinal ligaments and joint capsules, and anterior compression of the vertebral bodies.

FIGURE 13-26

Individuals with paraplegia often maintain an upright stance by shifting the pelvis anteriorly, which puts the LoG posterior to the hip joint axis. The external extension moment is counteracted by the internal flexion moment of the anterior hip ligaments (passive resistance).



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Postural Changes Across the Life Span

Loss of postural control and balance can have a negative impact on the functional capacity of individuals at any age.[114,116] Postural stability varies with age. In a study of

1,724 individuals between the ages of 6 and 97, Schwesig and colleagues found a significant nonlinear relationship between age and postural stability.[117] In this population they found postural stability was reduced in the young, peaked between adolescence and the mid-30s, and showed the greatest decline between ages 40 and 70, with significant limitations after the eighth decade.[117] This information is valuable to the clinician as decline in postural stability is associated with increased risk for falls.[114,116,118]

Adults and Older Adults

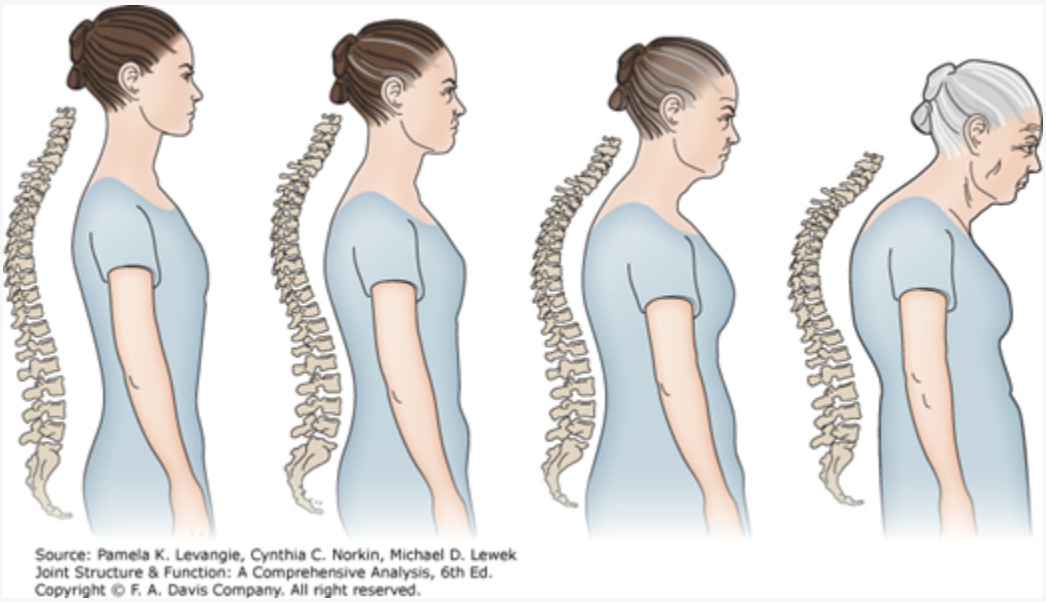
Deviations in resting alignment may be the result of compensatory mechanisms that are incorporated by the individual to facilitate greater postural stability. Examples of this in older adults may be seen by forward leaning or reaching for support with their upper extremities.

Age-related changes in sagittal alignment of the spine, pelvis, and lower extremities are common (**Figure 13–27**).[119–124] Alterations in cervical lordosis can influence, or be influenced by, the amount of thoracic kyphosis in adults.[50,125,126] In fact, individuals with reduced cervical lordosis have demonstrated ~10° reduction in their thoracic kyphosis compared with those with normal cervical lordosis.[125] Similarly, Lee and colleagues used radiographic measurements to demonstrate that the slope of the T1 vertebral body was the key factor to determine the sagittal alignment of the cervical spine.[50] Maintenance of this alignment is integral to upright posture as well as to maintaining horizontal gaze. Many studies have demonstrated increased anterior translation of the spine relative to the pelvis with increasing age.[87,119,124,127] This anterior translation increases rapidly from the seventh to ninth decade of life.[124]

Changes in the anterior translation of the spine relative to the pelvis can be compensated for by alterations in the position of the lumbopelvic spine or the lower extremities.[77,121,122,128,129] Common compensations include reduced lumbar lordosis, increased thoracic kyphosis, and a reduction in the sacral slope, with an increase in posterior tilt of the pelvis.[77,121,122,129,130] Without these compensations the move of an individual's CoM anteriorly within the BoS requires greater amounts of muscular effort to maintain upright posture. Concomitant loss of muscle in the aging population may increase the risk of falling and correlate to the rapid decline in postural stability between 40 and 70 years. These postural changes are also related to a reduction in health-related quality of life measures, suggesting the significant increase in postural deviation may impact overall well-being.

FIGURE 13-27

Postural changes across the life span.



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Pregnancy

Pregnancy is associated with a number of physiological changes, including increased weight in the abdomen and breasts and relaxing of the ligaments and connective tissue.[131] These physiological changes are often associated with alterations in postural alignment and subsequent complaints of low back, pelvic girdle, and leg pain (Figure 13–28).[131–133] Compared with non-pregnant women, pregnant women demonstrate significant increases in thoracic kyphosis, reduced lumbar lordosis, and sacral posterior inclination.[133,134] With increasing abdominal size, maintenance of the CoM over the BoS may result in compensatory adjustments of the craniocervical region as well as the lower extremities.[135] Musculoskeletal complaints of pain in the low back and pelvic girdle are likely associated with increased stresses placed on the lumbar spine and pelvis through increased axial load and anterior shear. Distortion of the abdominal and pelvic floor musculature from the expanding uterus may also result in changes in motor control necessary for postural stability.[136,137] McCrory and colleagues found that women who are pregnant walk with greater frontal plane mobility, increased step width, and greater thoracic extension at initial contact compared with non-pregnant women.[138] These gait deviations may vary dependent upon the characteristics of the individual such as weight gain, distribution, and height.

FIGURE 13-28

Postural changes associated with pregnancy.



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Foundational Concepts 13–4

Posture Changes Across the Life Span

Postural stability changes across the life span, with the peak of stability occurring from 18 to 30 but rapidly declining between 40 and 70 years.

- Interestingly, the peak age for ACL injuries in females is between ages 14 and 18, prior to peak postural stability.
- Pregnancy causes an upward and anteriorly directed shift in the CoM that may be accommodated by an increase in lumbar lordosis (posterior lean) to re-center the CoM over the BoS.
- The highest rate of hip fractures in males and females occurs over the age of 50, during the period of rapid decline in postural stability.
- Assessment of upright posture and balance control in the young female or in an adult over 50 years may be a great clinical screening tool to help identify individuals at risk for musculoskeletal injury.

Dynamic Postural Changes: Kinematics and Kinetics

Control of body alignment during transfers is essential for functional mobility in daily life. When examining dynamic activities, knowledge of normal kinematic and kinetic function is necessary.

Sit to Stand

Successful transition from a seated position to upright stance requires sufficient muscle strength and joint mobility, and the ability to perform this transition can be the difference between independence and disability. Elderly individuals, or individuals with musculoskeletal or neurological impairments, often experience a great deal of difficulty performing sit to stand transitions without use of upper extremity support or assistance. Yoshioka and colleagues assessed the lower extremity kinematics for sit to stand in healthy young adults and found that for successful accomplishment of this task the hip angle moves from about 100° of flexion to 0, the knee from 120° of flexion to 0, and the ankle from 30° of dorsiflexion to 0°.[139] The combined hip-knee peak joint moment necessary for success needed to be sufficient to raise the weight of the body. In

a more recent study, the same authors used mathematical modeling to determine that successful sit to stand cannot occur without adequate joint range of motion (ROM) or combined lower extremity muscle force.[140] Any disorder that disrupts adequate joint motion or muscle force will impact the sit to stand transfer. Individuals with knee osteoarthritis, for example, often exhibit deficits in quadriceps strength. As a result, they often demonstrate deviations during sit to stand, including increased weightbearing through the contralateral limb, increased trunk flexion, and reduced knee extension moments and impaired mechanical efficiency.[141,142]

Returning to sitting from standing requires eccentric control of the leg extensors. Individuals with paresis secondary to spinal cord injury with limited strength in the lower extremities will demonstrate increased use of the upper extremities, greater trunk lean, longer time to complete the task, or a higher impact force on the seating surface compared with healthy controls.[143] Seat height, the presence of arm rests, and foot position during the task are all critical components that will influence an individual's ability to successfully perform a sit to stand task.[144,145]



Patient Application 13–6

In the presence of weakness in the quadriceps muscles, an individual may have difficulty in rising from a lower seat height, such as a sofa with soft cushions, but no difficulty at all in rising from a higher seat or standard height seat with a firmer surface, such as a kitchen chair. Patients with weak quadriceps when seated on a sofa often use momentum or rocking to achieve an upright stance as this helps to compensate for limited strength in their limbs. Furthermore, a total hip arthroplasty done via a posterior surgical approach has a risk of dislocation when the hip flexes greater than 90° during the first few weeks of recovery. Use of raised toilet seats or seat cushions is common practice to limit the amount of hip flexion required to achieve the seated position.

Lunging

A lunge is a forward movement of the body with one leg in front of the other. It is a motion that may be necessary to reach into a lower cabinet or to get down to the floor. Lunges are exercises that are commonly used to help develop strength, power, and balance in the lower extremity. Inability to perform a lunge or maintain optimal alignment during a lunge is often a clinical indicator of lower extremity pathology or muscle weakness. Lunges require knee flexion greater than 90° and ankle dorsiflexion in both the front and back legs, with hip flexion in the forward limb and extension in the rear limb. Hale and colleagues found different muscle activation patterns between males and females who were performing a lunging movement.[146] Specifically, males demonstrated greater activation in biceps femoris, whereas females demonstrated greater semitendinosus activation. Both demonstrated similar activation patterns in the quadriceps throughout the task. Increased body mass has been shown to increase the hip extensor moment during lunging and therefore may be a reason that an individual is having difficulty performing this task.[147] When observing a lunge from the front, assessing the frontal plane stability of the femur is essential. An inability to maintain a neutral femur may be indicative of ankle joint limitations or weakness in the hips and trunk.[148,149]

Patient Application 13–7

Anterior cruciate ligament tears and patellofemoral pain syndromes are both painful conditions that result in activity and participation limitations. Some evidence suggests that individuals with poor control of frontal plane motion of the femur during functional tasks such as squatting, lunging, or stepping down are at a higher risk for these painful conditions. Observation of these functions may help guide further examination.

Jumping

Jumping or landing from a jump is a common task in sporting events and requires a great deal of strength and motor control. A number of injuries sustained in the lower extremity, including anterior cruciate ligament tears, commonly occur during jumping. There has been extensive research in the area of jumping with an effort to identify possible variables that may correlate to higher risk for injury. Landing from a jump requires the lower extremity to attenuate ground reaction forces while simultaneously decelerating the body. Landing techniques can impact the stability requirements of the knee. During a soft landing, which is described as a peak knee flexion angle of > 90°, the forces attenuated through the knee are less than when landing on a stiff knee.[150–152] Observing an individual's ability to maintain the knee in frontal and transverse plane alignment as knee flexion is controlled may assist a clinician in recognizing potential impairments that may lead to risk of injury.

Summary

- The maintenance of both static and dynamic balance occurs through the integration of sensory information from visual, vestibular, and somatosensory systems. Postural control maintains the CoM over the BoS using feedforward and feedback response systems.
- Optimal standing and sitting posture are often achieved by having the LoG pass as close to the joint axes as possible to minimize the need for internal joint moments.
- Observation of seated and standing posture is accomplished by the examination of multiple landmarks from multiple viewpoints (i.e., anterior, posterior, lateral).

- During seated and standing postures, the internal and external moments about each joint should maintain equilibrium.
- Assessment of static posture and ideal alignment were introduced with specific reference to various regions of the body, including the extremities and vertebral column. Common postural abnormalities were described introducing various conditions that may affect ideal posture and life-span variabilities.
- The role of postural alignment during dynamic functional movement was introduced.

Study Questions

1. What is the difference between posture and balance?
2. What is the role of each of the following systems for postural control: visual, vestibular, and somatosensory?
3. What is postural sway and what are the different strategies used to maintain upright posture?
4. How do we maintain our posture in the presence of known and unknown postural perturbations?
5. Describe the moments that would be acting on all body segments as a result of an unexpected forward movement of a supporting surface in a fixed support strategy. Describe the muscle activity that would be necessary to bring the

body's LoG over the BoS.

6. What is the role of postural assessment in clinical practice?

7. In ideal upright posture, where is the LoG relative to each of the following regions: head, vertebral column, hip, knee, and ankle? How does this influence muscle activity in those areas?

8. What is the relationship between the GRFV, LoG, and CoM in the erect static posture?

9. For the erect standing posture, identify the type of stresses that would be affecting the following structures: apophyseal joints in the lumbar region, apophyseal joint capsules in the thoracic region, annulus fibrosus in L5 to S1, anterior longitudinal ligament in the thoracic region, and the sacroiliac joints.

10. What effect might tight hamstrings have on the alignment of the following structures during erect stance: pelvis, lumbosacral angle, hip joint, knee joint, and the lumbar region of the vertebral column?

11. How would you describe a typical idiopathic lateral curvature of the vertebral column?

12. Identify the changes in body segments that are commonly used in scoliosis screening programs.

13. Describe various abnormalities of scapulothoracic position and what muscles may be involved.

14. Compare a flexed lumbar spine posture with an extended posture in terms of the stresses on the vertebral discs, ligaments, and joint structures.

15. Compare intradiscal pressures in erect standing with erect, slumped, and relaxed sitting.

16. Describe the effects of genu varum and genu valgum on the medial and lateral tibiofemoral compartments.

17. What are the risks associated with pes planus?

18. Describe the different joint positons of the metatarsal-phalangeal, proximal phalangeal, and distal phalangeal joints for hammer toe, claw toe and hallux valgus.

19. Explain how an individual who lacks lower extremity strength and elements of postural control can maintain upright stance.

20. How does aging affect postural alignment in the spine and how might this influence postural control?

21. What is the effect of pregnancy on upright posture and identify how this posture may impact the following structures: head, vertebral column, hip, knee, and ankle?

22. Describe the role of joint ROM in sit to stand and lunging activities, and how does this influence muscle requirements?

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Chapter 14: Gait

Michael A. Hunt; Sandra J. Olney; Janice J. Eng

Introduction

In “normal” human locomotion (ambulation, gait), the reader is given the opportunity to discover how individual joints and muscles function in an integrated manner to maintain upright posture and produce motion of the body as a whole. Knowledge of the kinematics and kinetics of normal ambulation provides the reader with the necessary foundation for analyzing, identifying, and correcting abnormalities in gait.

Walking is probably the most comprehensively studied of all human movements, but the variety of technologies, coupled with the diversity of disciplinary perspectives, has produced a complex and sometimes daunting literature. The fundamental biomechanical requirements of the movements that compose gait are logical and easily understood. The purpose of this chapter is to provide comprehension of normal ambulation mechanics that will serve as the foundation for analysis of normal and abnormal locomotion, including walking, running, and stair climbing.

Gait Analysis

Early gait analysis involved cinematographic film and time-consuming frame-by-frame hand-digitizing of markers that had been placed on body landmarks to evaluate movement kinematics. Ground reaction force data from force platforms were then included to provide an estimate of joint kinetics. The accuracy, and quantity, of data were limited by the digitizing approach, though our current understanding of normal

human locomotion was directly informed by this early research. The past two decades have witnessed an explosion of technical advancements in motion analysis that offer the ability to quickly and reliably collect and process large amounts of data from a variety of movements. The next major advancement in gait analysis has already begun and involves assessment of motion outside of a clinical or laboratory setting, including in the community and even in people's homes.

A modern gait laboratory (Fig. 14–1) includes some kind of motion analysis system that tracks segment motion or precise marker locations to generate three-dimensional anatomical coordinate data that are subsequently used to model a several-segment body with joint centers and centers of mass. Also included are one or more force platforms that provide simultaneous foot-floor forces including the position (center of pressure), magnitude, and orientation of ground reaction forces. EMG systems provide simultaneous information of muscle activity using surface electrodes, or sometimes, indwelling electrodes. At a minimum, kinetic data (force platform) and EMG data are typically sampled at 600 or 1200 Hz (samples per second), while kinematic (segment movement) data are sampled at 60 or 120 Hz. An excellent and engaging report of the evolution of clinical gait analysis, including motion analysis and EMG, can be found in Sutherland's articles.[1, 2]

FIGURE 14-1

A modern gait laboratory.



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Human locomotion, or gait, may be described as a translatory progression of the body as a whole, produced by coordinated, rotatory movements of body segments.[3] The alternating movements of the lower extremities essentially support and carry along the head, arms, and trunk, which constitute about 75% of total body weight, with the head and arms contributing about 25% of total body weight and the trunk contributing the remaining 50%.[4] To make full use of this chapter you should review the relevant biomechanics and anatomy of the lower limbs and understand the basic biomechanical concepts presented in Chapter 1, the available motion of major joints of the lower limbs, and have a thorough knowledge of the major muscle groups of the lower limbs and their actions.

Major Tasks of Gait

To understand gait, let us first identify the fundamental purposes. Winter proposed the following five main tasks for walking gait:[5]

1. Absorption of mechanical energy for shock absorption and stability or to decrease the forward velocity of the body
2. Maintenance of upright posture and balance of the body
3. Maintenance of support of the head, arms, and trunk, that is, preventing collapse of the lower limb
4. Generation of mechanical energy to maintain the present forward velocity or to increase the forward velocity
5. Control of the foot trajectory to achieve safe ground clearance and a gentle heel or toe landing

Maintaining balance and stability is clearly important during ambulation, and there is an increasing body of literature on what might be called subtasks of gait that are potentially destabilizing. These include gait initiation and termination, stair climbing, turning, obstacle crossing, and negotiating a raised surface.[6–14] Gait initiation and termination and stair climbing will be introduced later in this chapter.

Phases of the Gait Cycle

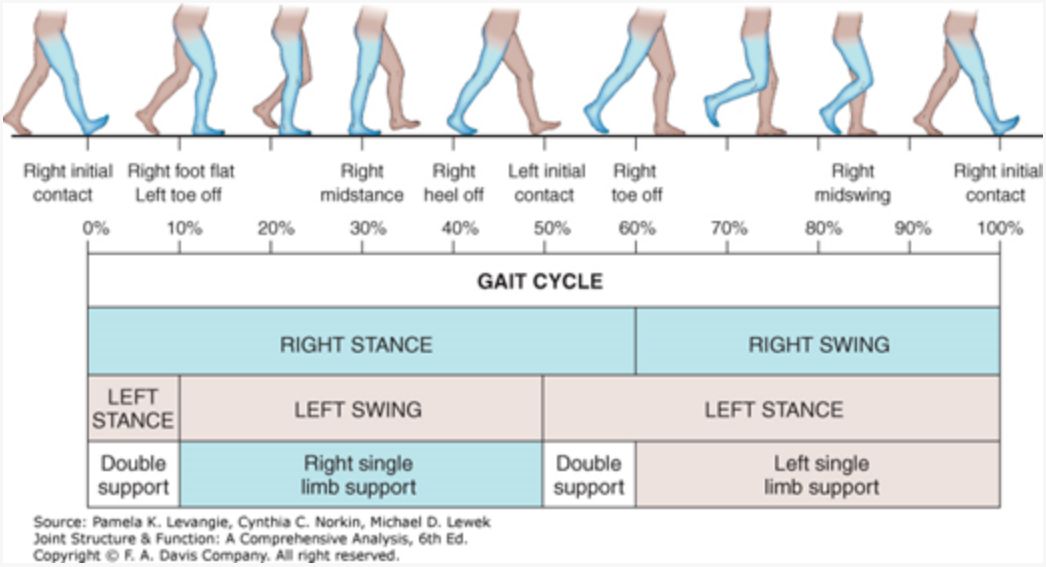
Gait has been divided into a number of time periods that make it possible to describe, understand, and analyze the events that are occurring. A **gait cycle** spans two successive events of the same limb, usually initial contact of the lower extremity with the supporting surface. The gait cycle is divided into percentiles that will be used to clarify events and phases. The normalization of the gait cycle to percentiles rather than raw time units enables the comparison of gait biomechanics independent of time,

speed, or actual movement task. Values for normal walking appear in the figures. During one gait cycle, each extremity passes through two major phases:

- A **stance phase,** when some part of the foot is in contact with the floor, which makes up about 60% of the gait cycle
- A **swing phase,** when the foot is not in contact with the floor, which makes up the remaining 40% (Fig. 14–2)

FIGURE 14-2

A gait cycle spans the period between initial contact of the reference extremity (right) and the successive contact of the same extremity. The gait cycle is shown with major events: stance and swing phases for each limb and periods of single and double support. The stance phase constitutes 60% of the gait cycle, and the swing phase constitutes 40% of the cycle at normal walking speeds. Increases or decreases in walking speeds alter the percentages of time spent in each phase.



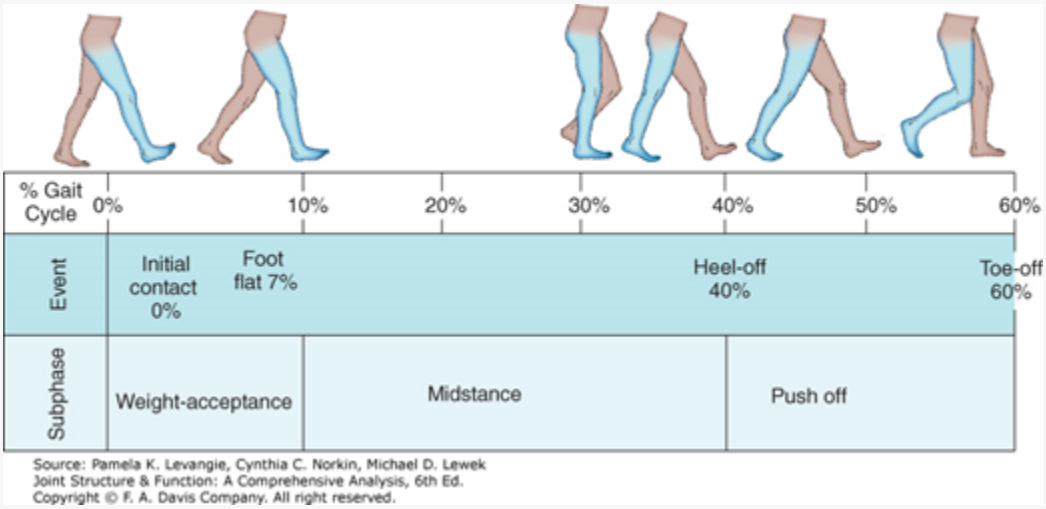
There are two periods of **double support** when both feet are in contact with the ground, occurring between the time one limb makes initial contact and the other one leaves the floor at toe-off. At a typical walking speed, each period of double support occupies about 10% of the gait cycle, for a total of approximately 20% of the gait cycle. Indeed, a given limb's gait cycle (100%) can be subdivided into a 40% swing phase when it is not in contact with the ground, and a 60% stance phase, made up of 20% double support and 40% single support (the time in which the contralateral limb is in swing).

Stance phase is further divided into subphases by various events, which mark the start and end of the subphases. Figure 14–3 identifies the events delimiting the stance phase as initial contact (sometimes referred to as heel contact or heel strike) and toe-off.

FIGURE 14-3

The stance phase of a gait cycle of the right lower limb. The events

that delimit subphases are shown and expressed as percentages of full gait cycle: initial contact, foot flat, heel-off, and toe-off. Subphases are weight acceptance phase, midstance phase, and push-off phase.



Events in Stance Phase

1. **Initial contact** refers to the instant the foot of the leading extremity strikes the ground. In normal gait, the heel is usually the point of contact, and the event is referred to as **heel contact** or **heel strike.** The word *strike* is actually a misnomer inasmuch as the horizontal velocity reduces to about 0.4 m/sec and only 0.05 m/sec vertically.[15] In abnormal gait or movements such as running or stair climbing, it is possible for the whole foot or the toes, rather than the heel, to make initial contact with the ground. Thus, initial contact is a more encompassing term.

2. **Foot flat** in normal gait occurs after initial contact at approximately 7% of the gait cycle. It is the first instant during stance when the foot is flat on the ground.

3. **Heel-off** is the point at which the heel of the reference extremity leaves the ground, usually about 40% of the gait cycle.

4. **Toe-off** is the instant at which the toe of the foot leaves the ground, usually about 60% of the gait cycle. In instances where a distinctive toe-off is not observed, the term **foot-off** may be used to describe the end of stance.

Subphases of Stance Phase

1. The **loading response** or **weight-acceptance** phase begins with initial contact and ends with foot flat and occupies only a small percentage of the gait cycle (see Fig. 14–3). This phase, which begins at initial contact and ends when the contralateral extremity lifts off the ground at the end of the double-support phase, occupies about 10% of the gait cycle. This subphase is characterized by large impact loads passing up the lower limb and requires some form of shock absorption to dissipate these loads effectively.

2. **Midstance phase** begins around the time of foot flat and contralateral toe-off at ~10% of the gait cycle and ends with heel-off at about 40% of the gait cycle. This subphase encompasses most of single limb support and maintains the forward progression of the body that requires stabilization of the upper body as it passes over the small base of support provided by one foot.

3. **Push-off phase** begins with heel-off at about 40% of the gait cycle and ends with toe-off at about 60% of the gait cycle (see Fig. 14–3). This subphase is characterized by large propulsive forces that propel the body forward through swing phase and into the next gait cycle. This time period may also be referred

to as **terminal stance** or **pre-swing.**

Swing Phase

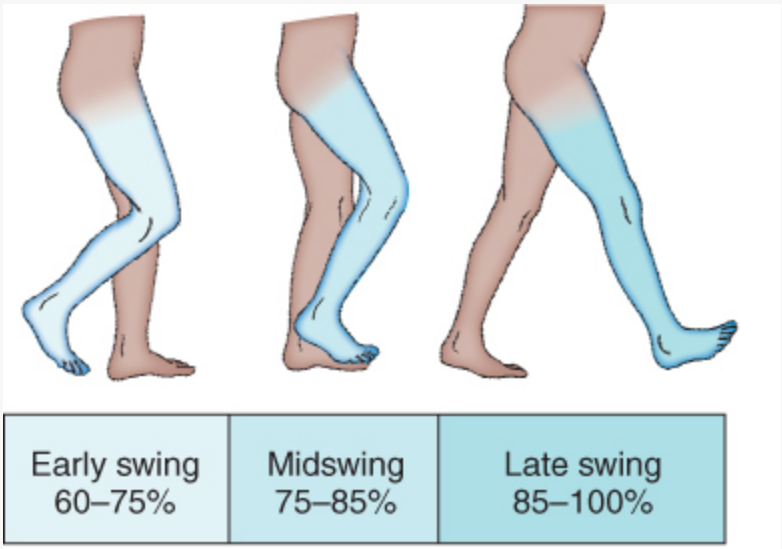
1. **Early swing phase** begins once the toe leaves the ground and continues until midswing, or the point at which the swinging extremity is directly under the body (**Fig. 14–4**). This phase is also referred to as **initial swing,** or the **acceleration phase.**

2. **Midswing** occurs approximately when the extremity passes directly beneath the body, or from the end of acceleration to the beginning of deceleration. The primary task involved in this subphase is the clearance of the toe as it passes forward beneath the body. Inadequate toe clearance increases the risk of falling.

3. **Late swing** occurs after midswing when the limb is decelerating in preparation for initial contact. It is also known as **terminal swing** or the **deceleration phase.**

FIGURE 14-4

The swing phase of a gait cycle. Early swing phase is also known as initial swing or acceleration, and late swing is also known as terminal swing or deceleration.



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Patient Application 14–1

Marlene Brown is a 63-year-old woman who sustained a stroke 15 days ago and shows hemiparesis (weakness) of her right arm and leg. She has been in a rehabilitation unit for 10 days and is making good progress walking, although she is ambulating at only 0.20 m/sec. A value of about 1 m/sec would be typical for an able-bodied person of her age. She has weakness in several muscle groups of her lower limb, notably the ankle plantarflexors, ankle dorsiflexors, knee extensors, and hip flexors, with distal muscles more affected than proximal ones. Particularly troublesome is the inability to clear her foot during the swing phase of gait. She uses a cane with a large base (a four-point cane) in her unaffected hand for stability. As you read the Gait Terminology section, ask yourself what differences from normal gait you might expect to see.

Gait Terminology

Time and Distance Terms

Time and distance are two basic parameters of motion, and measurements of these

variables provide a basic, yet important, description of gait. Together, they are commonly referred to as **spatiotemporal** characteristics of gait. **Temporal variables** include stance time, single-limb and double-support time, swing time, stride and step time, cadence, and speed. They can be reported either as raw time units (seconds) or as a percentage of the gait cycle (% cycle). The **distance (spatial) variables** include stride length, step length and width, and degree of toe-out. As distance measures, they are typically reported in meters (m), though given the effect of factors such as height, they are sometimes reported normalized to leg length (% leg length). These variables, derived in classic research from the 1970s to the 1990s, provide essential quantitative information about a person's gait and should be included in any gait description.[16–18] Importantly, these values have not changed over time, and any student or clinician should be well-versed in the expected values exhibited by healthy and pathological populations. Each variable may be affected by such factors as age, sex, height, size and shape of bony components, distribution of mass in body segments, joint mobility, muscle strength, type of clothing, and footgear. However, a discussion of all the factors affecting gait is beyond the scope of this text.

Temporal Variables

Stance time is the amount of time that elapses during the stance phase of one extremity in a gait cycle.

Single support time is the amount of time that elapses during the period when only one extremity is on the supporting surface in a gait cycle. This time is equivalent to the **swing time** of the contralateral limb.

Double support time is the amount of time spent with both feet on the ground during

one gait cycle. The percentage of time spent in double support may be increased in older persons and in those with balance disorders. The percentage of time spent in double support decreases as the speed of ambulation increases.

Step duration refers to the amount of time spent during a single step. When there is weakness or pain in an extremity, step duration may be decreased on the affected side and increased on the unaffected (stronger) or less painful side.

Cadence is the *number* of steps taken by a person per unit of time. Cadence may be measured as the number of steps per second or per minute, but the latter is more common. A shorter step length will result in an increased cadence at any given velocity.[19] As a person walks with increased cadence, the duration of the double-support period decreases. When the cadence of walking approaches 180 steps per minute, the period of double support disappears, and running commences. A step frequency or cadence of about 110 steps per minute can be considered as "typical" for adult men; a typical cadence for women is about 116 steps per minute.[4] Sometimes authors report values that refer to stride cadence, which is exactly half the step cadence, and refers to the number of strides per minute.

Walking velocity is the rate of linear forward motion of the body, which can be measured in meters or centimeters per second, meters per minute, or miles per hour. Scientific literature favors meters per second. The term **velocity** implies that direction is specified, although this is frequently not included, and the more correct term **walking speed** should be used if direction is not reported. In instrumented gait analyses, **walking velocity** is used, inasmuch as the velocities of the segments involve specification of direction:

$$\text{Walking velocity (meters/second)} = \text{distance walked (meters)/time (seconds)}$$

Clinically, walking speed/velocity is typically measured using the 10-meter walk test. In this test, patients walk a 14-meter long distance, of which the first and last 2-meter sections are used for acceleration and deceleration, respectively. The time taken to walk the middle 10 meters is recorded with a stopwatch and velocity is calculated as 10 (m)/time (seconds). For example, if it took a patient 9 seconds to walk 10 meters, their walking velocity would be

$$10\ (\text{m})/9\ (\text{seconds}) = 1.11\ \text{m/s}$$

Women tend to walk with shorter and faster steps than do men at the same velocity largely because of height and leg length differences.[19] Increases in velocity up to 1.2 m/s are brought about by increases in cadence and stride length, but above 1.2 m/s, step length levels off, and speed increases are achieved with only cadence increases.

A person's normal comfortable gait speed may be referred to as **preferred, natural, self-selected,** or **free. Slow** and **fast** speeds of gait refer to speeds slower or faster than the person's normal comfortable walking speed, designated in a variety of ways.

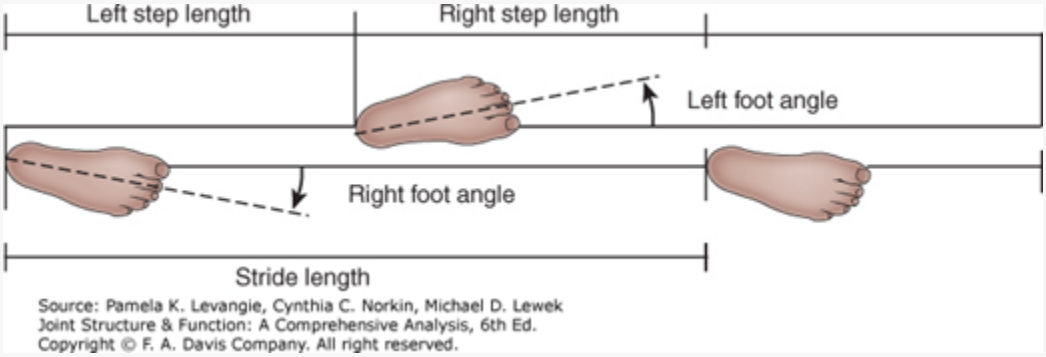
Distance Variables

Stride length is the linear distance between two successive events that are accomplished by the *same* lower extremity during gait.[20] In general, stride length is determined by measuring the linear distance from the point of one initial contact to the point of the next initial contact of the same extremity (**Fig. 14–5**). The length of one stride includes all of the events of one gait cycle. Stride length also may be measured by using other events of the same extremity, such as toe-off, but in normal gait, two successive ipsilateral initial contacts are usually used. A stride includes two steps, a

right step and a left step. However, stride length is not always twice the length of a single step, because right and left steps may be unequal. Stride length varies greatly among individuals, because it is affected by leg length, height, age, sex, and other variables. Stride length can be normalized by dividing stride length by leg length or by total body height, so people of different sizes can be compared. Stride length usually decreases in older persons and increases as the speed of gait increases.[19,21–23]

FIGURE 14-5

Stride length, step length, and step width shown with foot angle placements. The midpoint of the heel is used as a point of reference for measuring step width.



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Stride duration refers to the amount of time it takes to accomplish one stride. Stride duration and gait cycle duration are synonymous. One stride, for a normal adult, lasts approximately 1 second.[24]

Step length is the linear distance between two successive points of contact of *opposite* extremities. It is usually measured from the initial contact of one extremity to the initial contact of the opposite extremity (see Fig. 14–5). A comparison of right and left step

lengths will provide an indication of gait symmetry. The more equal the step lengths, the more symmetrical is the gait.

Step width, or width of the walking base, may be found by measuring the linear distance in the frontal plane, between the midpoint of the heel of one foot and the same point on the other foot (see Fig. 14–5). Step width has been found to increase when there is an increased demand for side-to-side stability, such as occurs in older persons and in small children. In toddlers and young children, the center of gravity is relatively higher than in adults, and a wide base of support is necessary for stability. In the normal population, the mean width of the base of support is about 3.5 inches and typically varies within a range of 1 to 5 inches.

Toe-out angle represents the angle of foot placement and may be found by measuring the angle formed by each foot's line of progression and a line intersecting the center of the heel and the second toe. The angle for men normally is about 7° from the line of progression of each foot at free speed walking (see Fig. 14–5).[21] The degree of toe-out decreases as the speed of walking increases in normal men.[21]

Kinematic Terms

Kinematics is the term used to describe movements without considering the internal or external forces that caused the movements. These measures can include positions, velocities, and accelerations of body segments or joints. Sophisticated equipment—at first, stroboscopic photography, then cinematography, and electrogoniometers, and, more recently, many types of computerized motion analysis systems—have provided comprehensive information about limb positions and their motions in normal and abnormal gait.[1, 2, 25–27]

The sophisticated equipment found in research labs, however, is rarely found in clinical settings. Instead, **observational gait analysis** is the process in which an observer makes a judgment as to whether a particular joint angle or motion varies from a norm without the use of specialized equipment. Use of standardized checklists helps the clinician to identify important aspects of gait in a systematic approach. Many different checklists are available in the literature.[28] Usually observational gait analysis is used to hypothesize causes of deviations and to direct treatment objectives. One disadvantage of the observational method of analysis is that it requires a great deal of training and practice to be able to identify the segment of gait in which a particular joint angle deviates from a norm while a person is walking. Recording the action using a video camera, smart phone, or tablet, and then viewing it using slow playback can improve this greatly. Another disadvantage of observational gait analysis methods is that they frequently have low reliability, especially between observers, although some reports have identified some variables and conditions under which reliability is satisfactory.[29]

Trajectories, or the paths in space of body parts, are of particular interest. For example, because the toe clearance above the ground during swing may be as low as 1.0 to 2.5 cm, the path of the toe during gait is important in preventing trips and falls.

Joint angles are of primary importance in analyzing gait. They may be expressed as absolute positions in space or, more commonly, as the relative angles of joints between adjacent segments, such as the hip or the knee. In most literature the anatomical position is called the “zero” position of the joint.

Kinetic Terms

Kinetics is concerned with the forces acting on the body that are the cause of the

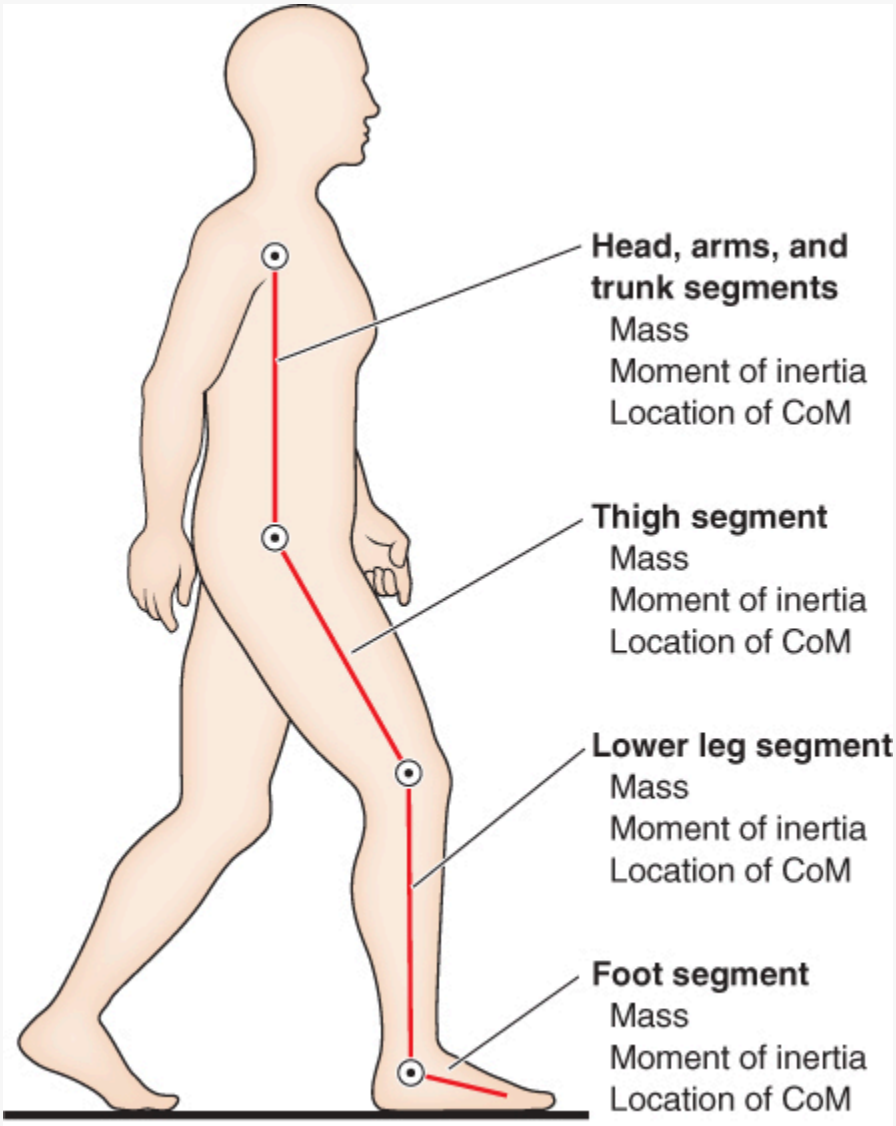
movement. A kinetic analysis is performed to understand the forces acting on the foot by the supporting surface, the forces acting on and across the joints, the forces produced by muscles, the moments of force produced by those muscles crossing the joints, the mechanical power generated or absorbed by those muscles, and energy patterns of the body during walking. Understanding the internal forces can help provide an indication of the joint load due to altered walking patterns, and could help explain or even predict joint pain or degenerative joint changes. Although there have been some reports of invasively instrumenting a joint at the time of a surgery (e.g., knee replacement surgery to measure the forces during walking), this is obviously not a practical method.[30] An alternative approach is to use knowledge of physics and mathematics, combined with measurement of segment kinematics and the external forces applied to the body, to estimate what "must have occurred internally" to produce the measured movement of the body. This is termed **inverse dynamics.**

A **link segment model** is most often used with an inverse dynamic approach to understand the internal forces during movement. This means that we look at the body as a series of segments (e.g., foot, lower leg, thigh, etc.) with links at the joints. We begin the analysis starting with the segment touching the supporting surface (foot) and use Newtonian mechanics (Fig. 14–6). To do this we must know the positions of body markers through the gait cycle, usually from the motion analysis technology. We also need to know the forces acting on the body, and for the walking person, these are the ground reaction forces derived from a force platform, the weight of the body parts (because of gravitational acceleration), and inertia of the body parts.[4,15,31] **Inertia** (units: $kg \cdot m^2$) is a measure of an object's resistance to changes in its rotation rate. It is the rotational analog of mass. Inertia is particularly important in analyzing fast-moving

parts of the body during the gait cycle, such as the swinging limb.

FIGURE 14-6

Simple four-segment link segment model of half a human body for use in gait studies. For each segment identified, the mass, moment of inertia, and location of the center of mass should be known for applications.



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Using the link segment model, we can determine what forces and moments have been acting at each joint (e.g., the ankle) to cause the segment (e.g., shank) with a particular mass and moment of inertia to move with measured linear and angular accelerations. Thus, we can estimate joint kinetics, by knowing the forces at the foot, the individual's anthropometrics, and how each segment moved. This process is progressed up the body segment by segment and solving for the more proximal joint. Larger numbers of segments and three-dimensional analyses are more complex than this, but the principles are the same.

Ground reaction forces (GRFs) are the forces being applied to the foot by the ground during stance phase. These forces are equal in size (magnitude) but opposite in direction to the forces applied to the ground by the foot. GRFs are expressed using vertical, anteroposterior, and mediolateral axes.[4,15] If we combine the force components in two or three planes, the vector sum is a single expression of the ground reaction force and is termed the **ground reaction force vector (GRFV).** When we refer to a single component, such as the vertical GRF, the abbreviation GRF will be used. If the components are combined into a single vector (force[s] with a direction), then GRFV will be used. In gait, we frequently use the sagittal GRFV. The **center of pressure (CoP) of the foot** on the supporting surface is the point where the resultant of all the floor-foot forces acts. It moves along a path during gait and produces a characteristic pattern. GRFs, GRFVs, and CoPs that are characteristic of normal walking appear later in the chapter as Figures 14–10, 14–11, 14–12, and 14–13.

A **moment of force,** usually referred to simply as **moment,** is the same as **torque,** which was introduced in Chapter 1. It is defined as the product of a force (e.g. muscle force or

GRF) and the perpendicular distance from its action line to the joint center—termed its **moment arm.** It is the angular equivalent of force and can cause a segment to rotate about an axis (i.e., joint).



Expanded Concepts 14–1

Understanding How the Kinetics of Gait Are Studied

A two-dimensional analysis requires the solution of three equations. The following three equations are applied, and are standard for each segment:

$$\Sigma F_x = ma_x \text{ (1) where x indicates the horizontal direction}$$

$$\Sigma F_y = ma_y \text{ (2) where y indicates the vertical direction}$$

$$\Sigma M_0 = I\alpha \text{ (3)}$$

$\Sigma F_x = ma_x$ (1) where x indicates the horizontal direction

$\Sigma F_y = ma_y$ (2) where y indicates the vertical direction

where

Σ means “the sum of all of the”

F_x = forces in the designated x direction, in this case horizontal, in Newtons (N)

m = mass of the segment, derived from anthropometric tables, in kilograms (kg)

a_x = acceleration of the segment’s center of mass in the x direction, derived from position and time data, in meters per second squared (m/sec^2)

F_y = forces in the designated y direction, in this case vertical, in N

a_y = acceleration of the segment’s center of mass in the y direction, derived from position and time data, in m/sec^2

M_0 = moment about selected point 0, the center of mass, in newton-meters (N·m), largely attributable to muscle activity, with ligaments, tendons, joint capsules, and bony components involved to a lesser extent

I = moment of inertia, a measure of an object’s resistance to changes in its rotation rate. It is the rotational analog of mass. It is derived from anthropometric tables and expressed in $kg \cdot m^2$

α = angular acceleration of segment derived from segment position and time data,

expressed in radians per second squared (rad/sec^2). A radian is about 57°.

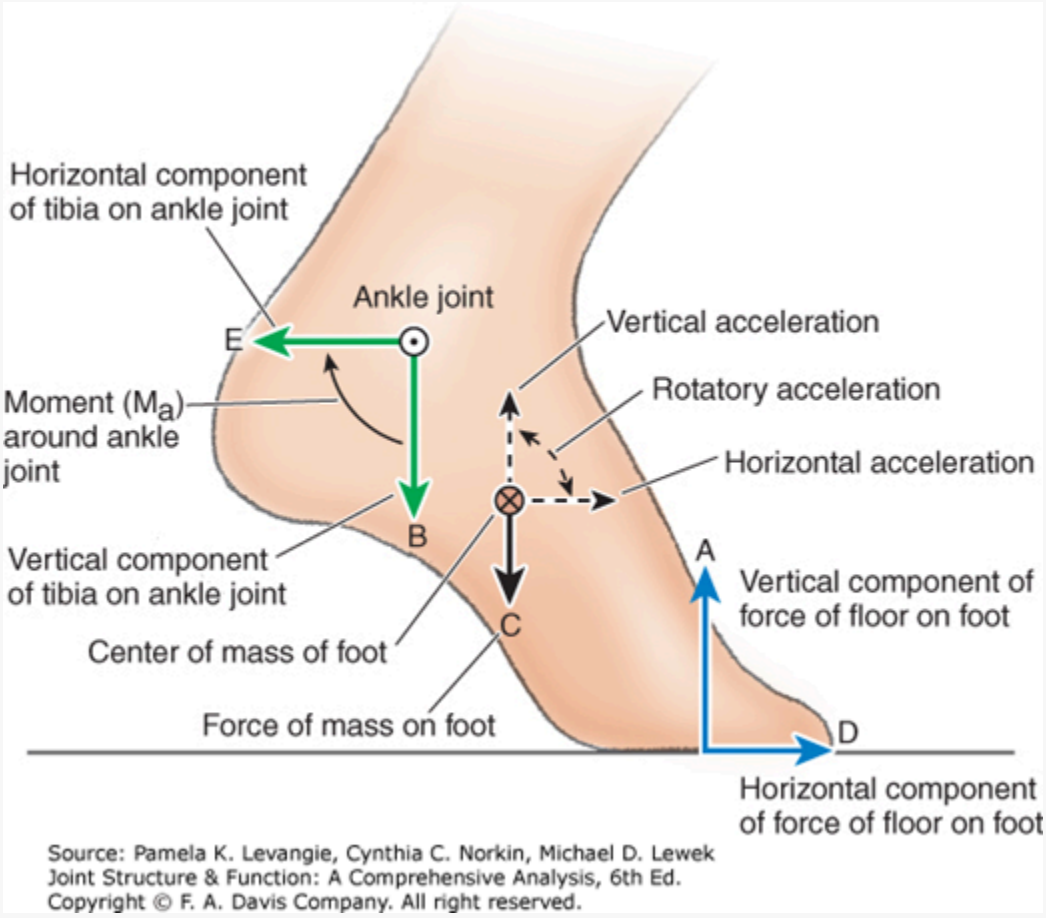
If we refer to Figure 14–7 with reference to the foot segment, equation (1) says that the sum of all horizontal forces must equal the product of the mass of the foot and its acceleration in the horizontal direction. Equation (2) says that the sum of all vertical forces must equal the product of the mass of the foot and its acceleration in the vertical direction. Equation (3) says the sum of the moments about any designated center (we are choosing the center of mass of the foot) must equal the product of the moment of inertia (which can be visualized as the resistance to rotation) and the angular (rotatory) acceleration of the segment. Figure 14–7 shows that there are three things we do not know: the horizontal force at the ankle (E), the vertical force at the ankle (B), and the moment around the ankle (M_a). Foot-to-floor forces are shown as A and D, and are derived from a force plate.

Because we have three equations, we can solve for three unknowns but we cannot calculate the muscle moments on opposite sides of the joint if there is co-contraction. Indeed, a limitation of using inverse dynamics is that calculated biomechanical outcomes (in particular, joint reaction forces) may be largely inaccurate in instances where there is co-contraction present. Solving these three equations provides the horizontal force of the tibia on the foot (at the ankle), the vertical force of the tibia on the foot (at the ankle), and the moment about the ankle. *In other words, we are finding out what forces and moments had to have been acting at the ankle that the foot with that particular mass and moment of inertia moves with those* particular *linear and angular accelerations.* We can now continue to calculate the moments and forces as we progress up the body, segment by segment, and solve for the lower leg, then the thigh, then the pelvis/trunk. Larger numbers of segments and three-dimensional analyses are more complex than this, but the principles are the same.

FIGURE 14-7

Diagram of the foot segment in stance shown with proximal joint (ankle) and all forces and moments acting on the segment. No moment is present at the free end of the segment. Known forces and

their location consist of the forces of the floor on the foot (A and D) and the force caused by the foot's mass (C); moments that can be calculated about the center of mass are moments caused by A and D, and the foot's moment of inertia. Unknown forces are joint reaction forces on the ankle (B and E), and the unknown moment is the net moment Ma at the ankle joint being caused by muscles, in this case, largely the ankle plantarflexors.



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Internal moments are moments generated by active or passive forces from internal structures (e.g., muscles, joint capsules, and ligaments) to counteract the external forces acting on the body. External forces such as the ground reaction forces produce

external moments about the joints. For example, when the weight is on the heel at initial contact (see Fig. 14–11), an external plantarflexion moment is caused by the GRFV that will act to plantarflex the ankle. This external plantarflexion moment must be opposed by an internal ankle dorsiflexor moment produced by anterior tibial muscles to maintain equilibrium. We will use this internal moment convention in this chapter. For ease of reading, the word *moment* will refer to an internal moment unless otherwise indicated.

Authors use different **moment conventions** to display internal moments in figures, but they indicate the direction of the moment by the words *flexor/extensor, abductor/adductor,* or *internal/external rotator.* The moment profiles presented in this chapter display the following internal moments as positive: ankle plantarflexor, knee extensor, and hip extensor.

Energy is the capacity to do **work,** and both work and energy are expressed in the same units, joules (J). Work is performed by the application of force, which produces accelerations and decelerations of the body and its segments. Muscles use metabolic energy to perform mechanical work by converting metabolic energy into mechanical energy. A main objective of locomotion is to move the body through space with the least expenditure of energy.

The overall metabolic cost incurred during locomotion may be measured by assessing the body's rate of oxygen uptake or consumption (VO_2) per unit of distance traveled. To measure oxygen consumption, the person breathes into a mask or tube attached to a system called a metabolic cart, which analyzes a number of parameters, including breathing rate and the amount of air inhaled and exhaled. If a long distance is traveled

but only a small amount of oxygen is consumed, the metabolic cost of that particular gait is low. Approximately 32% of maximum oxygen consumption is needed by 20- to 30-year-olds to walk at a comfortable speed under steady-state conditions; this rises to 48% for 75-year-olds or when mechanical work is higher because of a chronic medical condition.[32,33] **Metabolic equivalents (METs)** are also used to express energy cost of the activity as multiples of the resting metabolic rate. Oxygen consumption for a person walking at 4 to 5 km/hour averages 100 mL/kg body weight per minute, typically 2.5 to 4 METS. The greatest efficiency is attained when the least amount of energy is necessary to travel a unit of distance. When asked to walk at a comfortable speed, people choose the speed at which they are most energetically efficient, and if the speed of walking deviates above or below this, the energy cost per unit of distance walked increases.[32]

Power generation is accomplished when muscles shorten (concentric contraction). They do positive work and add to the total energy of the body. Power, expressed in watts, is the work or energy value (in joules) divided by the time over which it is generated. The power of muscle groups performing gait is calculated through an inverse dynamic approach. The power generated or absorbed across a joint is the product of the net moment and the net angular velocity across the joint.[34] If both are in the same direction (flexors flexing, extensors extending, for example), positive work is being accomplished by energy generation.

Power absorption is accomplished when muscles perform a lengthening (eccentric) contraction. They do negative work and reduce the energy of the body. If joint motion and moment are in opposite directions, negative work is being performed through energy absorption.

Electromyography

Electromyography (EMG) is a technique for evaluating and recording the activation signal of muscles. EMG is performed using an instrument called an **electromyograph,** to produce a record called an **electromyogram,** in which the electrical activity generated by a muscle, or group of muscles, is recorded. Electrodes may be placed on the skin surface or inserted into the muscle. There is a great deal of information about EMG, the varieties of techniques that can be used, and the patterns obtained during gait.[34–37] EMG is often used in conjunction with force plates, goniometry, or motion analysis systems to link the muscle activity with other events during the gait cycle. The EMG record provides information about the time when particular muscles are acting and the relative level, or profile, of their activity. Importantly, it does not explain why the muscles are acting or how much force the muscles are generating. The reader is encouraged to follow the developing literature on muscle function that is derived from elaborate mathematical simulations involving modeling precise muscle geometry and anthropometrics.[38,39] Although this work has progressed exponentially over the past 20 years, there is still much that is unknown about the true behavior of muscles and joints in vivo.[40]



Patient Application 14–2

Ms. Brown walks with a speed of 0.20 m/sec and a cadence of 25 steps per minute. It is evident even on visual inspection that double-support time is considerably longer than normal on both steps. Stance phase is more than 60% of the gait cycle on her affected side, but she spends an even greater proportion of time in stance on the unaffected side. Her left step length (unaffected side) is shorter than her right (affected side), but her stride lengths are equal. Why? If you understand that a person walking in a straight line must have equal stride lengths but may have unequal step lengths, you understand the concepts of steps and strides.

Characteristics of Normal Gait

Time and Distance Characteristics

Means and standard deviations of walking speed, stride length, and step cadence appear in Table 14–1. The values were derived from the classic work of Finley and Cody, who surreptitiously measured the gait of 1,100 pedestrians, from Kadaba and colleagues, and from Oberg and colleagues, who obtained gait laboratory measurements.[16–18] Knowledge of the range of values during normal gait is imperative if one wishes to identify abnormal gait. Importantly, minimum threshold values have been identified for certain key parameters that enable independent, community ambulation. For example, a walking speed of 0.8 m/s is commonly suggested as the minimum speed that enables someone to ambulate functionally in the community and perform tasks such as crossing a street safely.[41]

Table 14-1

Normative Values for Time and Distance Variables



Foundational Concepts 14–1

Normative Values for Time and Distance Gait Variables

It is helpful in clinical practice to keep approximate time and distance measures in mind. From Table 14–1 we can see that the length of a stride is usually between a meter and a quarter (1.25 m) and a meter and a half (1.50 m); the speed of walking is approximately this stride length (one stride) per second (1.2–1.5 m/s); and step

cadence is approximately 120 per minute, or two steps per stride and one stride per second.

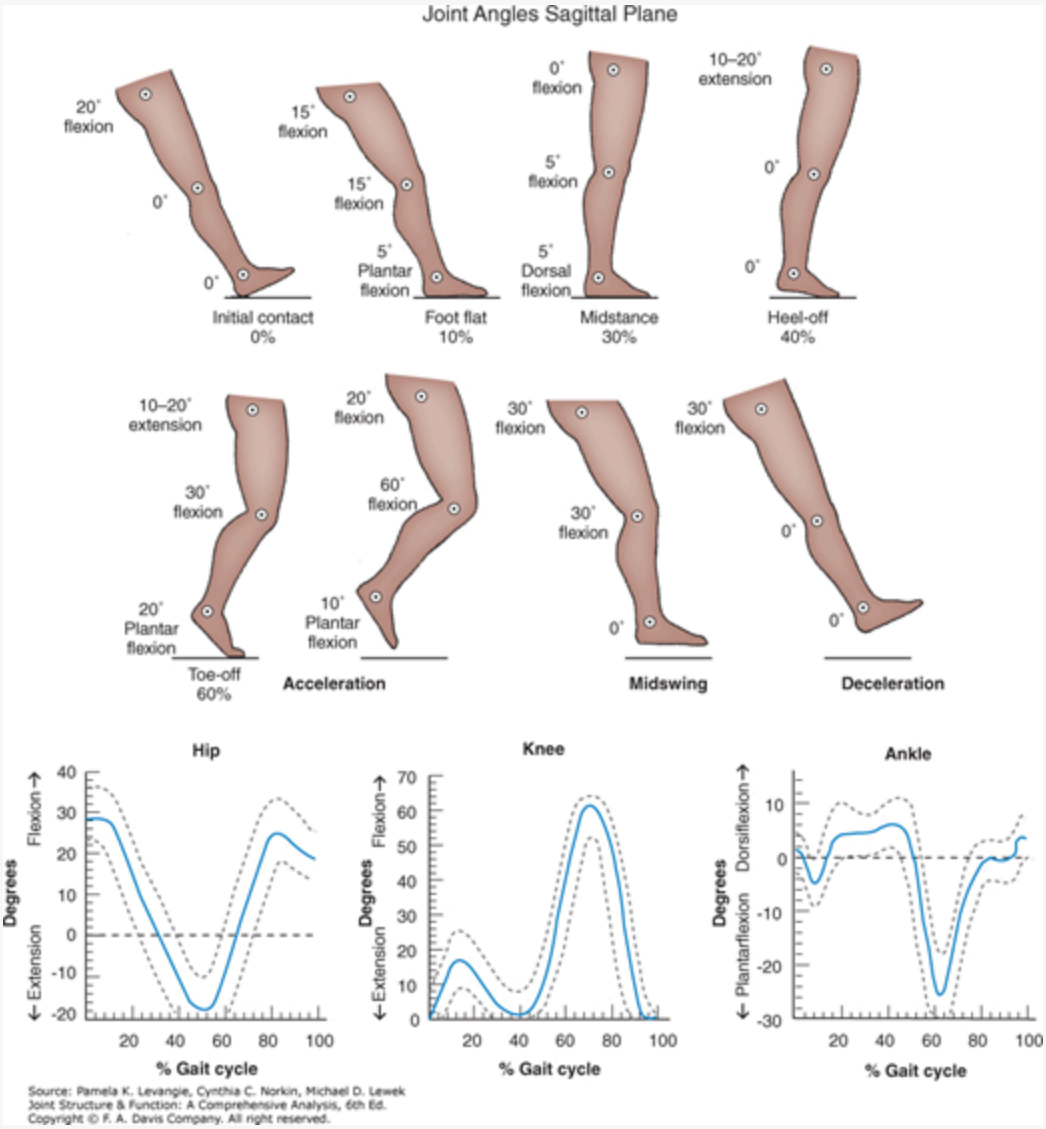
Sagittal Plane Joint Angles

Sagittal and frontal plane kinematics and kinetics have been reasonably well described, but transverse plane data are inconsistent and dependent on variations in joint positions and the specific methodologies (e.g., link-based model definition) used and will not be included here.

The approximate sagittal plane range of motion (ROM) needed in normal gait and the time of occurrence of the maximum flexion and extension positions for each major joint may be determined by examining the joint angle profiles in Figure 14–8. The standard deviation bars (dashed line) around the mean profiles (solid lines) give an indication of how much person-to-person variation exists, demonstrating that 67% of subjects' values fell within the range shown. Results reported in normal gait studies vary with age, gender, height, and walking speed of subjects and with the method of analysis. Data presented here were derived from three-dimensional analyses.[34] For simplicity, the mean value shown in the figures will be referred to in the text, taken to the nearest 5°, and, to remind the reader that these are not fixed values, the "approximately" sign (~) will be used. The anatomical position is defined by a hip, knee, and ankle of 0°, otherwise known as neutral. For the purposes of this text, flexion for the hip and knee and dorsiflexion for the ankle are given positive values, whereas extension and plantarflexion are given negative values.

FIGURE 14-8

Joint angles in degrees at the hip, knee, and ankle in the sagittal plane. The dotted lines in the angle diagrams represent the standard deviation values, and the solid lines represent the mean values. *(Joint angle diagrams redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263-265. St. Louis, MO: Mosby-Year Book, 1994, with permission from Elsevier.)*



In Figure 14–8, it can be seen that the hip achieves maximum flexion (~20° to 30° of flexion) around initial contact at 0% of the gait cycle and reaches its most extended position (~10° to 20° of extension) at about 50% of the gait cycle, between heel-off and toe-off. The knee is straight (0°) at initial contact and nearly straight again just before heel-off at 40% of the gait cycle. During the swing phase, the knee reaches its maximum flexion of ~60° at ~70% of the gait cycle. Note also that a small knee flexion phase occurs at 10% of the gait cycle and peaks at ~15° of knee flexion. The ankle reaches

maximum dorsiflexion of ~7° of dorsiflexion at approximately heel-off at about 40% of the gait cycle and reaches maximum plantarflexion (25° of plantarflexion) at toe-off (60%).



Foundational Concepts 14–2

Hip, Knee, and Ankle Range of Motion Needed for Normal Walking

For normal walking, we need a hip ROM from approximately 20° of extension to 20° of flexion, a knee range from straight (0°) to 60° flexion, and an ankle range from 25° of plantarflexion to 7° dorsiflexion. If these joint ranges are not available, a gait pattern would be expected to show considerable deviation from the norm.

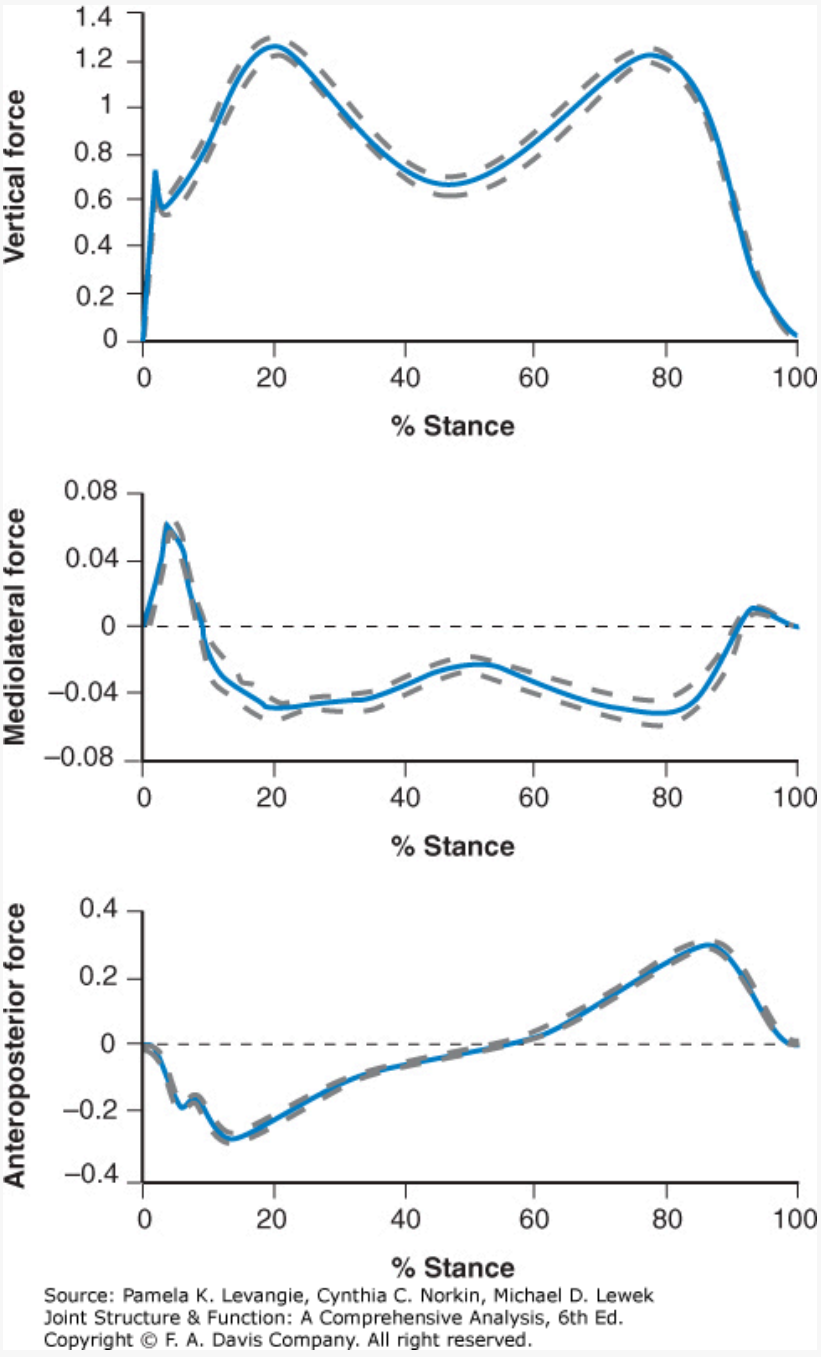
Frontal Plane Joint Angles

During the first 20% of stance, the pelvis on the contralateral (swing) side drops about 5°, which results in adduction of the ipsilateral (stance) hip (**Fig. 14–9**). The hip then abducts smoothly to about 5° of abduction, peaking about toe-off, then returns to neutral at initial contact. The knee remains more or less neutral, except for a brief abduction peaking at about 7° in midswing, and then returns to neutral.[27] From the figure, it can be seen that the ankle complex everts from about 5° of inversion to 5° of eversion in early stance and inverts about 15° during push-off.

FIGURE 14-9

Joint angles in degrees at the hip, knee, and ankle in the frontal plane. *(Redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263-265. St. Louis, MO: Mosby-Year Book,*

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Patient Application 14–3

An examination of video-captured joint angles reveals that Ms. Brown has no knee flexion phase in early stance, and she tends to fully extend her knee in midstance. She

has minimal ability to dorsiflex her ankle and has difficulty clearing the floor with her affected limb as a result of poor dorsiflexion and because she does not bend her knee more than a few degrees during swing phase. Instead, she tends to lift her pelvis ("hike") to clear her foot during swing on the affected side. Her affected hip does not extend beyond neutral. How do Ms. Brown's joint angle profiles vary from normal in stance and swing phases?

Ground Reaction Force and Center of Pressure

The ground reaction forces during stance phase have a typical pattern from initial contact to toe-off (Fig. 14–10).[42] In the vertical direction, the magnitudes are low at first but increase to values that are greater than body weight both in early stance and again in terminal stance, with lower values at midstance. The first hump is related to the weight acceptance when the body's downward velocity is being slowed (upward acceleration of the body's mass). The second hump is because of push-off and shows that the body's center of mass is being accelerated upward to increase its upward velocity. In the anteroposterior direction, the GRF is directed posteriorly against the foot that is making initial contact and prevents the foot from slipping forward. It acts as a "braking force" that decelerates the body and reaches a maximum magnitude of about 20% of body weight just after weight acceptance. At midstance, the force is oriented vertically, but as the body enters the second half of stance phase, the vector is directed anteriorly against the foot, accelerating the person forward. This is commonly referred to as the propulsion component. The mediolateral forces are small in magnitude and are variable across individuals. In general, there tends to be a small medially directed force acting on the foot. If someone were to walk with a very wide base of support, however, the medially directed force would become larger. Figure 14–11 shows the GRFVs in the sagittal plane relative to the joint positions in early stance phase. You can

see that the GRFV at initial contact passes behind the ankle (tending to plantarflex it), but in front of the knee and hip (tending to extend the knee and flex the hip). In early stance, the GRFV passes behind the knee (tending to flex it), then moves ahead of it until terminal stance. About midstance (Fig. 14–12), the GRFV passes behind the hip joint and remains there until swing phase begins. The implications of the GRFV position relative to the joints will be discussed in detail later.

FIGURE 14-10

Ground reaction forces as a proportion of body weight over the stance phase. Positive values represent upward, lateral, and anterior forces from the ground acting on the foot. *(Adapted and redrawn from Hunt AE, Smith RM, Torode M, et al: Intersegment foot motion and ground reaction forces over the stance phase of walking. Clin Biomech [Bristol, Avon] 16:592, 2001.)*

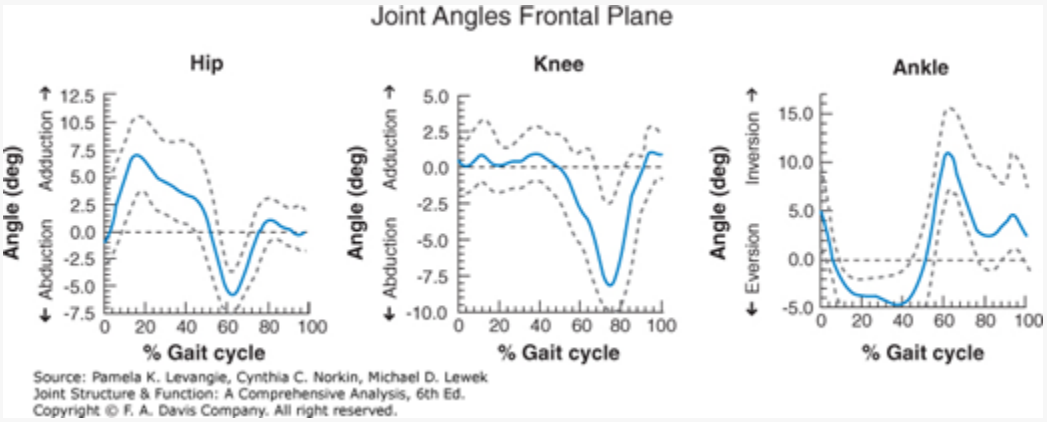
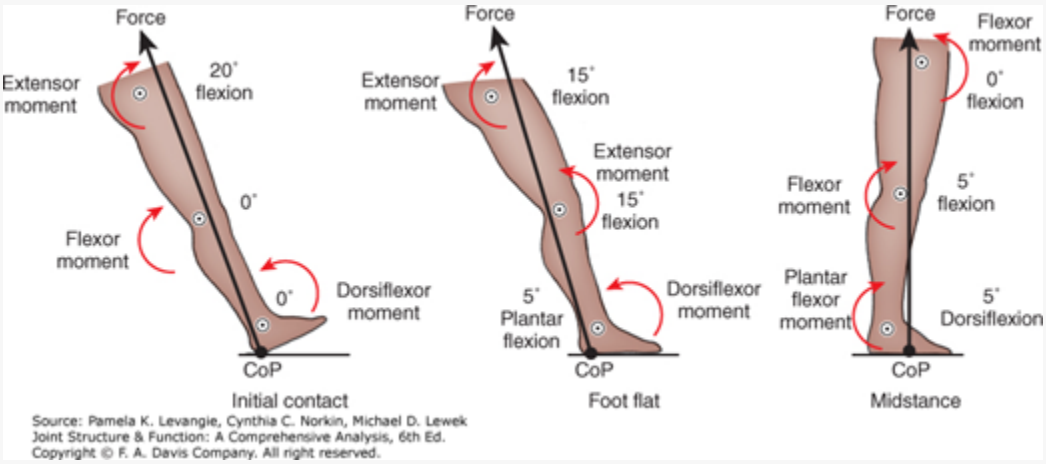


FIGURE 14-11

Diagram of joint positions with center of pressure (CoP), ground

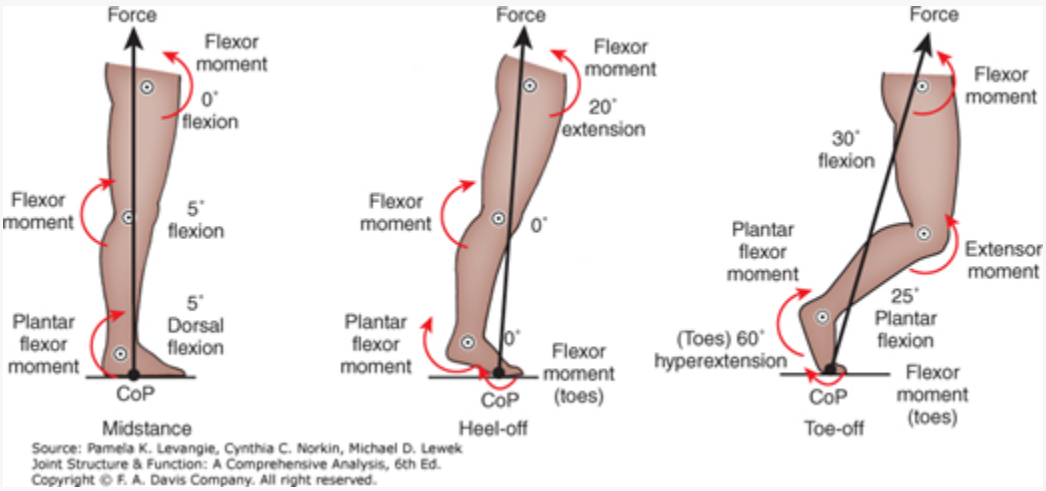
reaction force vector (GRFV), and internal net moments for gait cycle events of initial contact, foot flat, and midstance.



Source: Pamela K. Levangie, Cynthia C. Norkin, Michael D. Lewek
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FIGURE 14-12

Diagram of joint positions with center of pressure (CoP), ground reaction force vector (GRFV), and internal net moments for gait cycle events of midstance, heel-off, and toe-off.



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The single point on the foot at which the resultant surface pressure may be considered

to be acting is called the **center of pressure** (CoP). This point is the starting point for the GRFV. The CoP of the foot on the supporting surface produces a characteristic pattern (Fig. 14–13). The pattern for normal individuals during barefoot walking differs from the patterns in which various types of footwear are used.[43] In barefoot walking, the CoP starts at the posterolateral edge of the heel at the beginning of the stance phase and moves in a nearly linear manner through the midfoot area, remaining lateral to the midline, and then moves medially across the ball of the foot with a large concentration along the base of the first metatarsal. The CoP then moves toward the second and first toes during terminal stance.

FIGURE 14-13

A center of pressure (CoP) pathway is shown by the position of **A.** the blue dot at initial contact, **B.** at foot flat, **C.** just before heel-off, and **D.** just before toe-off.



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Expanded Concepts 14–2

Why Don't Hip, Knee, and Ankle Moment Figures Look Familiar to Me?

Sometimes when we are reading journal articles that include figures and explanations of joint moments at the hip, knee, or ankle during walking, the shapes seem totally unfamiliar and the explanations do not make sense. In fact they often feel "reversed," which, in fact, is exactly what has happened. There are two ways of reporting moments. Some authors use "external" moments, which refer to the moments produced by the calculated external forces (e.g., gravity, GRFV) acting around the joint. Here we have chosen to use "internal" moments, which refer to the *moments produced by the muscles and other internal structures around the joint.* To achieve equilibrium, internal moments are equal and opposite of the calculated external moments, and must occur to produce the movements and accelerations of body segments that we observe with motion capture systems. The net angular acceleration (and ultimately, the rotation about a joint) will be the result of these net moments. For example, if the GRFV passes behind the knee "trying to" flex it in early stance, there will be an external knee flexion moment, which must be opposed by an internal knee extension moment caused by the quadriceps muscle. If the external knee flexion moment is larger than the internal knee extension moment, a *net flexion* moment will result, thereby producing flexion at the knee joint. Similarly, an internal abductor moment opposes an external adductor moment—equal in magnitude, but opposite in direction. Therefore, internal moments must match the magnitude of the external moments, but in the opposite direction, to result in the positions and motions that we observe.

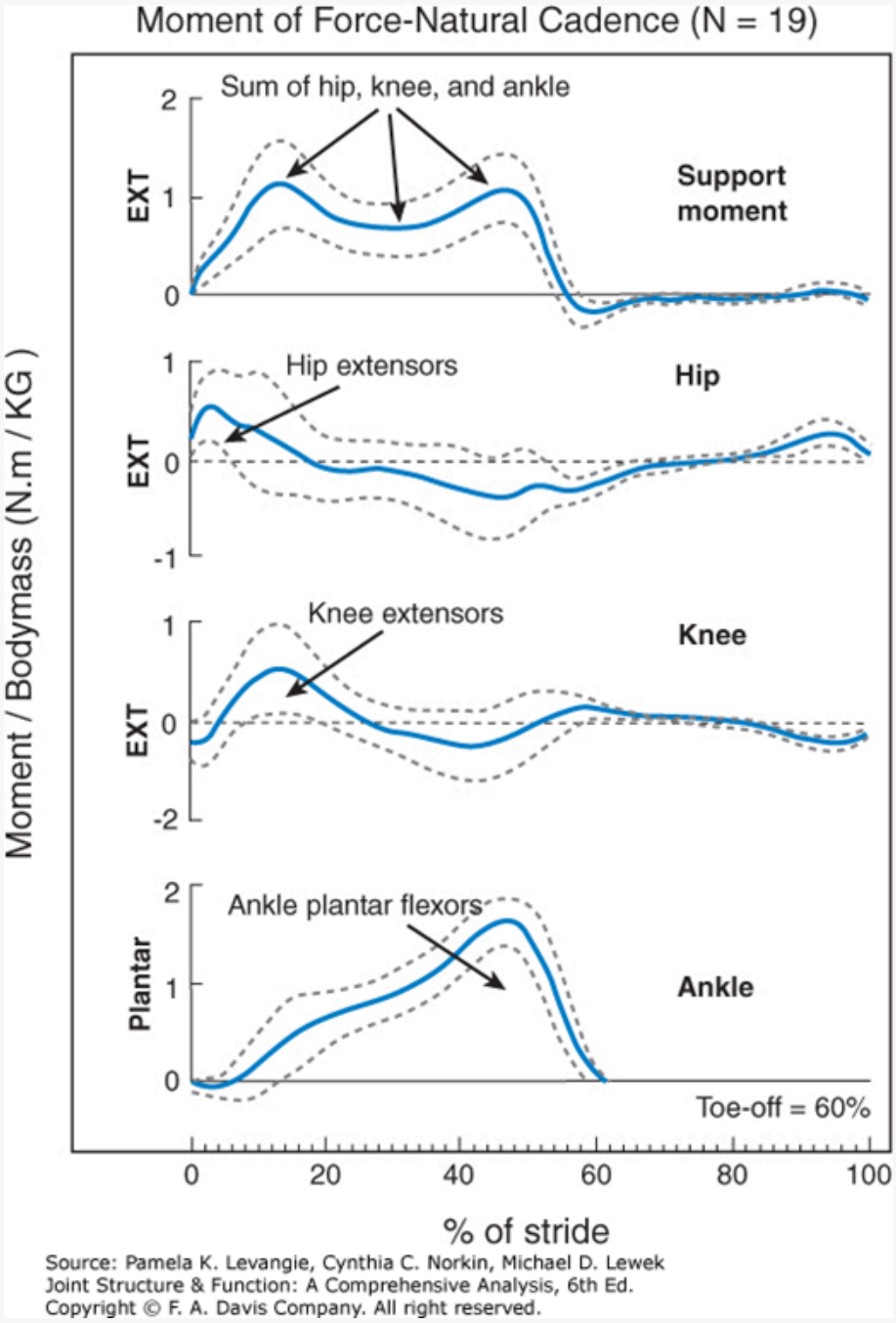
Sagittal Plane Moments

Before concerning ourselves with the individual sagittal plane moments about the hip, knee, and ankle, let us consider the concept of the support moment, which will help us to understand the moment patterns through the gait cycle. The sum of the hip, knee, and ankle moments keeps the leg from collapsing during the stance phase. That is, for most of the stance phase, the sum of all moments acting at the hip, knee, and ankle is a positive or extensor moment (Fig. 14–14). Winter called this total limb synergy a **support moment.**[5] He found the extensor support moment to be consistent for all walking speeds for both normal individuals and persons with disabilities. In Figure

14–14, notice that hip extension provides all of the positive moment in early stance, but it is soon joined by a knee extensor moment. (Remember that the moment identifies only the “supportingness” of the muscle group; it does not mean that muscle shortening or lengthening is occurring. To find out if the muscle is shortening or lengthening, one needs to consult the joint angle profiles: Even if there is no change in joint angle, a moment may be present. For example, in normal gait the knee extensors are first lengthening and then shortening.) As stance phase proceeds, there is increasing support provided by the ankle plantarflexors until they become *the only support in most of terminal stance.* The support moment changes from a net extensor to a net flexor moment in terminal stance (55% to 60% of the gait cycle), which is involved in the initiation of swing. In late swing, a small net extensor moment appears again, to assist in the final positioning and preparation of the limb for initial contact.[5,43]

FIGURE 14-14

Typical pattern of sagittal plane moments at the hip, knee, and ankle, shown with their algebraic sum, the support moment. *(Redrawn from Winter DA: The biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario: Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

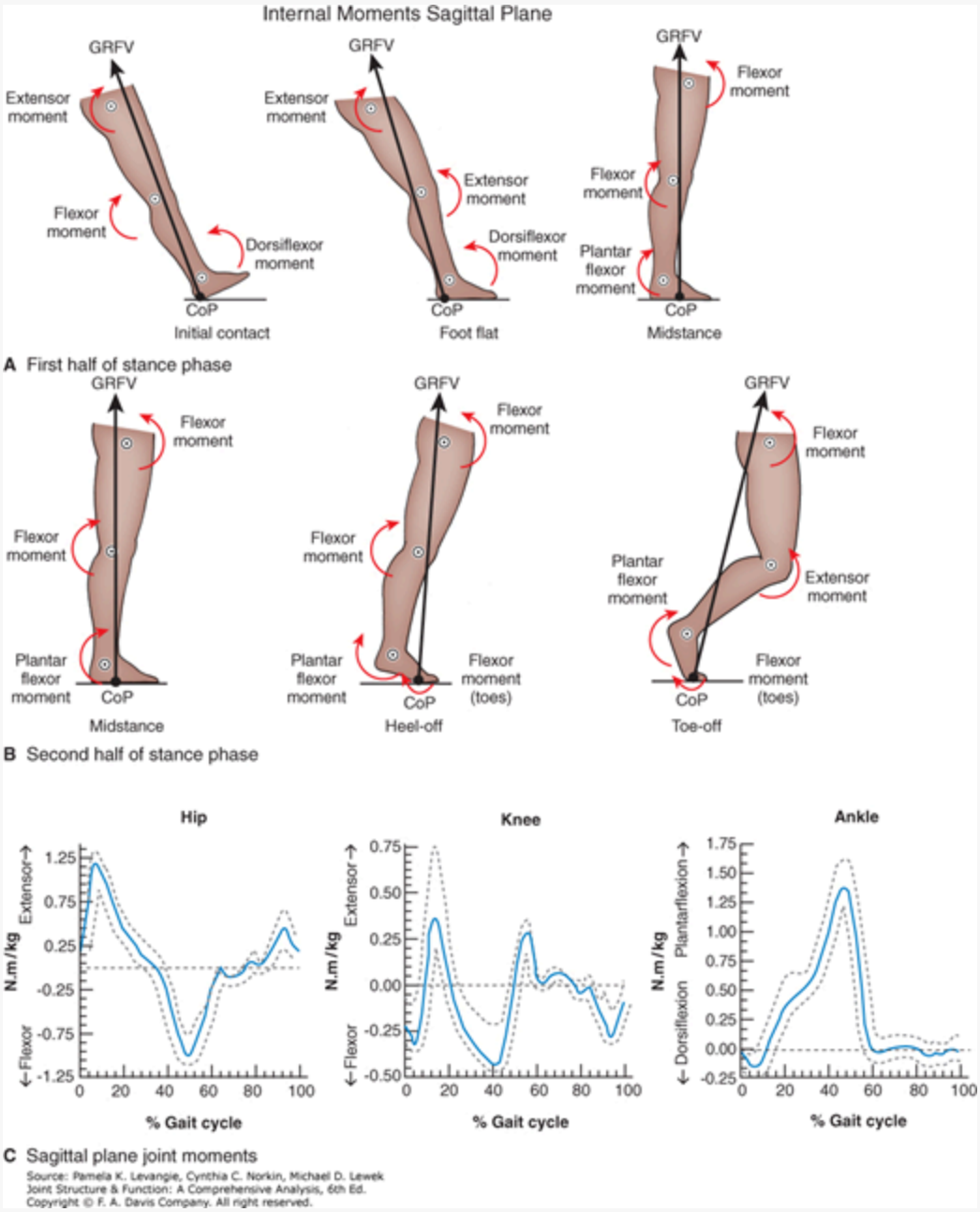


A clinically important feature of the support moment is that as long as the moments add up to be extensors, the body can vary how it accomplishes its support. For example, if the ankle plantarflexors are weak, the hip extensors or the knee extensors can compensate. If contraction of the knee extensors causes pain, the hip extensor or

ankle plantarflexors can contract harder. The support moment helps us to understand the joint moment profiles for each joint. The sagittal plane moment profiles for the hip, knee, and ankle are shown with GRFVs in Figure 14–15, and with joint angle profiles and muscle work in Figure 14–16.

FIGURE 14-15

Patterns of internal moments in the sagittal plane at the hip, knee, and ankle with center of pressure (CoP) and ground reaction force vectors (GRFVs). The dotted lines represent the standard deviations, and the solid lines represent the mean values. *(Diagrams of internal moments redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263-265. St. Louis, MO: Mosby-Year Book, 1994, with permission from Elsevier.).*

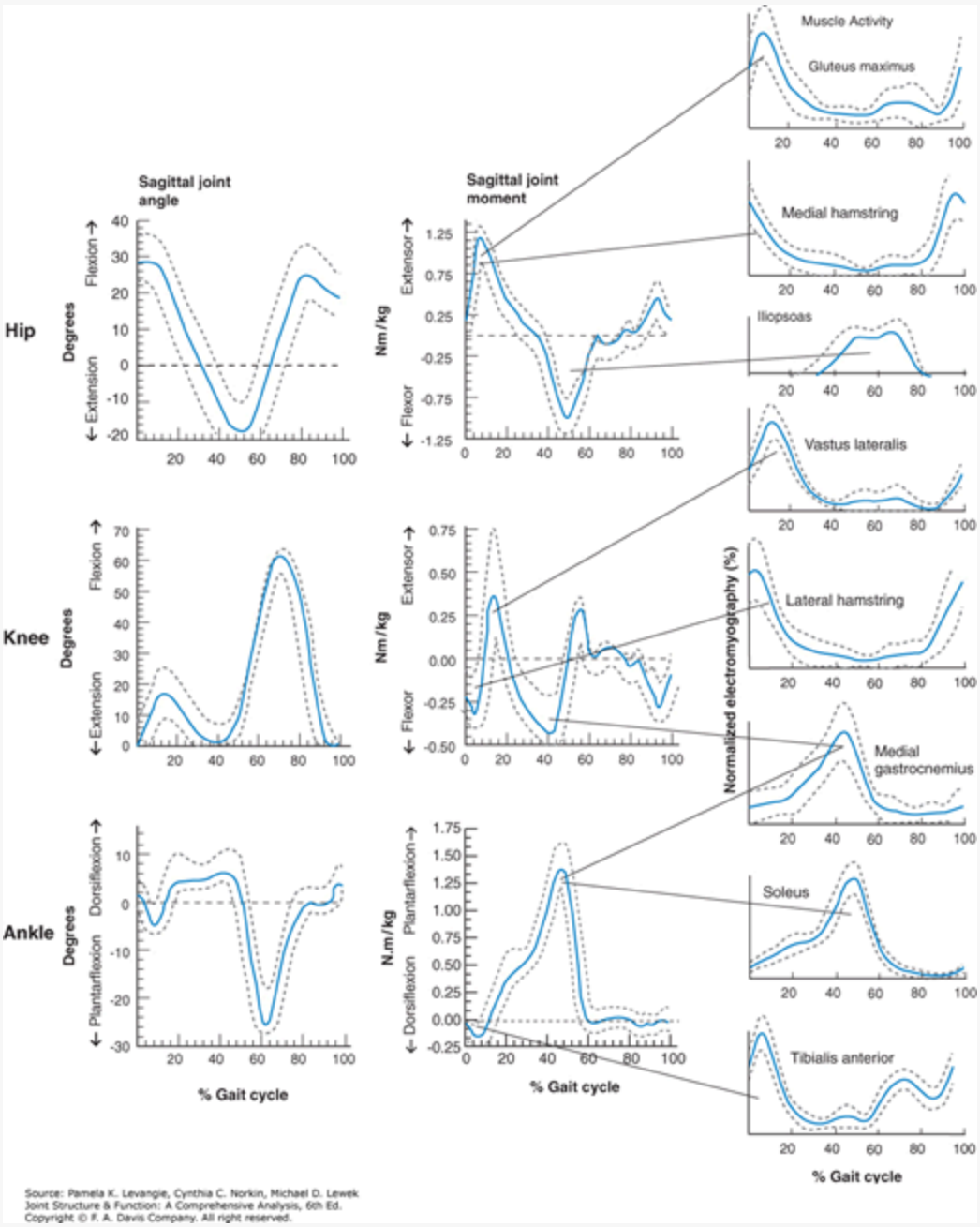


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FIGURE 14-16

Joint angles and net internal joint moments in the sagittal plane, and EMG profiles of representatives of major contributors to joint

moments of hip, knee, and ankle during adult gait. *(Angle and moment profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263-265. St. Louis, MO: Mosby-Year Book, 1994, with permission from Elsevier. Muscle activity redrawn from Winter DA: The biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario:, Waterloo Biomechanics, 1991, with permission from David A. Winter. Iliopsoas muscle activity redrawn from Bechtol CO: Normal human gait. In American Academy of Orthopaedic Surgeons: Atlas of Orthotics, p 141. St Louis, MO: CV Mosby, 1974, with permission from Elsevier.)*



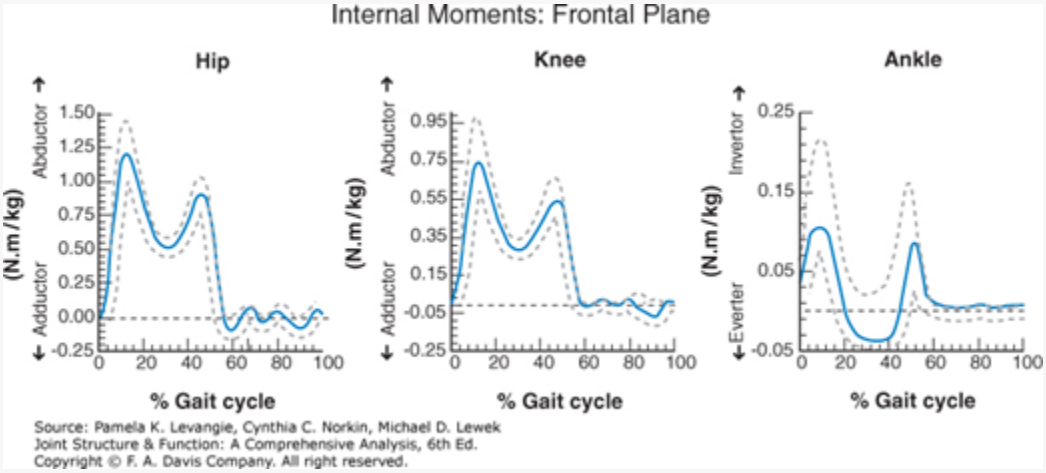
Frontal Plane Moments

Frontal plane moments are less frequently reported than sagittal plane moments, though the biomechanical concept is the same. Frontal plane moment profiles are shown in Figure 14–17 and with joint angle profiles and muscle activity in Figure 14–18.

Large abduction moments of similar shapes occur at the hip and knee, and a smaller one occurs at the ankle. These are provided largely by ligament forces across the knee and ankle joints, and are necessary as the center of mass of the body is considerably medial to the point of support on the foot throughout most of stance. Of particular clinical importance is the knee abduction moment—typically denoted as the external knee adduction moment in the literature—as it has been shown to be an important biomechanical outcome in the study and treatment of knee osteoarthritis.[44,45] It has been suggested to be a valid proxy of tibiofemoral joint load distribution, with higher values equating to a greater risk of medial compartment knee OA progression.[45]

FIGURE 14-17

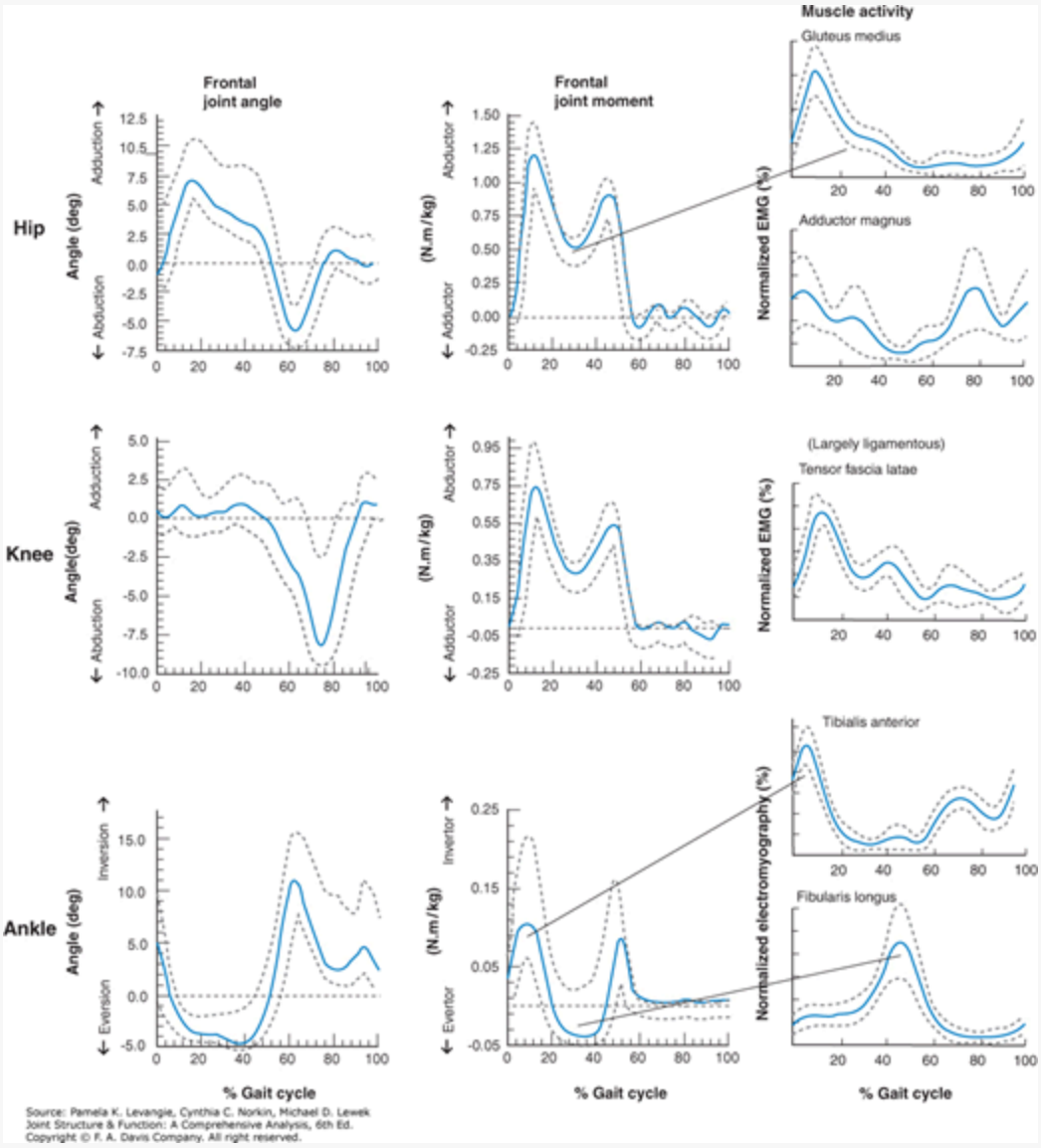
Patterns of internal moments in the frontal plane at the hip, knee, and ankle. The dotted lines represent the standard deviation, and the solid lines indicate the mean values. *(Redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263–265. St. Louis, MO: Mosby-Year Book, 1994, with permission from Elsevier.)*



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FIGURE 14-18

Joint angles and net internal joint moments in the frontal plane, and EMG profiles of representatives of major contributors to joint moments of hip, knee, and ankle during adult gait. *(Angle and moment profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263–265. St. Louis, MO: Mosby-Year Book, 1994, with permission from Elsevier. Muscle activity redrawn from Winter DA: The biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario: Waterloo Biomechanics, 1991, with permission from David A. Winter.)*



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Patient Application 14–4

Ms. Brown has generalized muscle weakness in her lower limb, and specifically has low levels of activation of her ankle plantarflexors, knee extensors, and, to a lesser extent, hip extensors. This will make it difficult for her to develop an adequate support moment. To gain knee stability she tends to thrust her knee backward. We know that she has smaller deficits at the hip than at the ankle, and so we would encourage more active hip extension in early stance to assist with the support moment, as well as trying to stimulate the knee extensors. During this early post-stroke period, we would expect some natural increase in force-generating capability

of the muscles (strength) and we would be prepared to take advantage of it by strengthening the muscles during functional movements, as well as perhaps via electrical stimulation activated within the walking cycle (functional electrical stimulation). If over time she is unable to provide enough support, an ankle orthosis would provide a passive extensor moment, but there may be added energy costs associated with the addition of the orthosis.

 Expanded Concepts 14–3

Kinematics and Kinetics of the Foot and Ankle

Specific descriptions of the biomechanics of the foot during gait have been hindered by the complexity of the composite movements created by the ankle, subtalar, and transverse tarsal joints. These kinematics are referred to as *pronation* (a composite of dorsiflexion, eversion, and abduction) and *supination* (a composite of plantarflexion, inversion, and adduction). Usually, the foot movement is described as consisting of pronation early in stance, followed by progressive supination. However, three-dimensional analyses of the lower leg and foot, modeling the foot as a rearfoot segment and a forefoot segment (rather than as a single segment, which is the most common simplified approach), have provided more insight into its behavior during stance.[46,47] Although kinematics and kinetics are highly variable and depend on subject, foot position, terrain, and method of analysis, some general features are thought to occur. The rearfoot segment everts with respect to the lower leg early in stance and inverts during push-off. In the transverse plane, the rearfoot segment externally rotates, (abducts) under the tibia in early stance, followed by internal rotation (adduction) under the tibia through push-off. The forefoot segment adds considerably to dorsiflexion during early stance.

Sagittal Plane Powers

Sagittal plane power profiles across the hip, knee, and ankle are compared with joint angle and major muscle activity profiles in **Figure 14–19**. A summary of the phases of power generation and absorption during the full gait cycle appear in Table 14–2. Table 14–3 shows powers with joint motion, GRFs, moments, and muscle activity from initial contact to midstance, and Table 14–4 for midstance to toe-off. Despite the complexity of

the appearance of this information, the important facts are straightforward. Of greatest importance is the fact that most of the positive work of walking is provided by the ankle plantarflexors at push-off, the hip extensors in early stance, and the hip flexors in terminal stance and early swing ("pull-off"). Second, the knee extensors normally provide very little power, and act largely as absorbers, especially during loading in early stance. To make it possible to compare subjects, power values have been normalized by dividing the power in watts by the subjects' mass in kilograms. Important points have been given notations that relate to the joint, the time point, and the plane of motion; for example, H1-S refers to the first (1) power peak of the hip joint (H) in the sagittal plane (S), while K2-F would refer to the second local peak of the knee joint in the frontal plane. These peaks can also be thought of as "bursts" of energy generation or absorption during specific times of the gait cycle.

FIGURE 14-19

Joint angles and joint powers in the sagittal plane, and EMG profiles of representatives of major contributors to joint powers of hip, knee, and ankle during adult gait. *(Angle profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263-265. St. Louis, MO: Mosby-Year Book, 1994, with permission from Elsevier. Power profiles redrawn from Eng JJ, Winter DA: Kinetic analysis of the lower limbs during walking: What information can be gained from a three dimensional model? J Biomech 28:753, 1995, with permission from Elsevier. Muscle activity redrawn from Winter DA: The*

biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario: Waterloo Biomechanics, 1991, with permission from David A. Winter. Iliopsoas muscle activity redrawn from Bechtol CO: Normal human gait. In American Academy of Orthopaedic Surgeons: Atlas of orthotics, p 141. St Louis, MO: CV Mosby, 1974, with permission from Elsevier.)

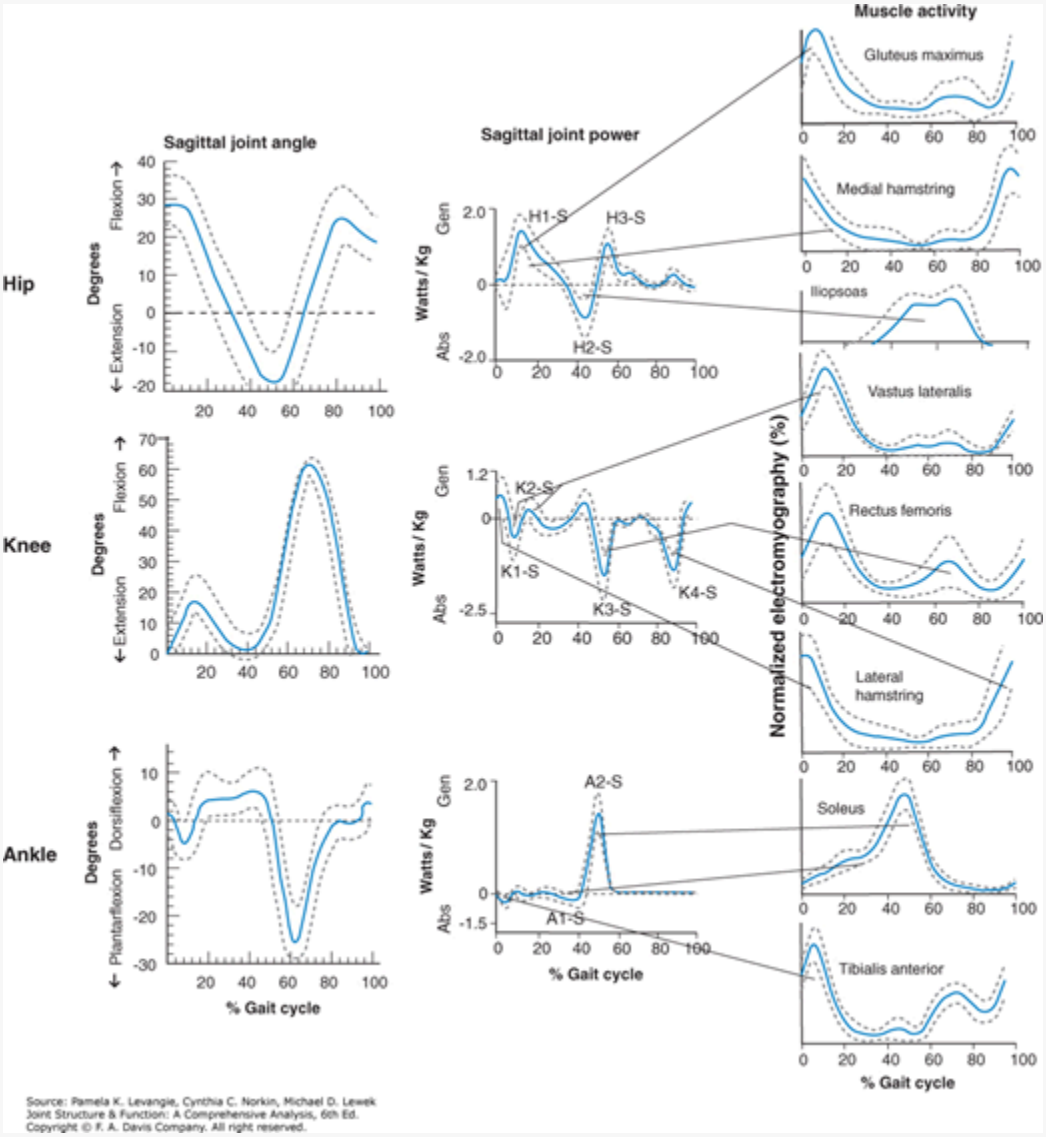


Table 14-2

Summary of Major Phases of Power Generation and Absorption During Full Gait Cycle

Table 14-3

Summary of Gait Characteristics From Initial Contact to Midstance

Table 14-4

Summary of Gait Characteristics From Midstance to Toe-Off

In the sagittal plane (see Fig. 14–19), a burst of positive work (energy generation) occurs as the hip extensors contract concentrically during early stance (H1-S), while the knee extensors perform negative work (energy absorption) by acting eccentrically (K1-S) to control knee flexion during the same period.[48] Following a tiny period of negative work by the dorsiflexors at initial contact, negative work is performed by the plantarflexors (A1-S) as the leg rotates over the foot during the period of stance from foot flat to about

40% of the gait cycle. However, a small amount of positive work is done by the knee extensors at the beginning of this period (K2-S), extending the knee after foot flat.[48] Positive work of the plantarflexors at push-off (A2-S: terminal stance, ~40% to 60% of the gait cycle) and hip flexors at pull-off (H3-S: terminal stance and in early swing, ~50% to 75% of the gait cycle) increases the energy level of the body. During this 40% to 60% of the gait cycle when energy is being generated from A2-S and H3-S, simultaneous absorption is occurring by knee extensors (K3-S). In late swing, negative work is performed by the knee flexors (K4-S) as they work eccentrically to decelerate the leg in preparation for initial contact. Recent advances in technology have attempted to capitalize on this relationship to turn the negative work with respect to the body into positive work for external sources. For example, Donelan and colleagues secured a knee brace to the lower limbs of study participants to capture power during the deceleration phase of the knee joint in terminal swing (K4-S), similar to "regenerative braking" in hybrid cars.[49] It is possible that the power that is captured during this part of the gait cycle can be harnessed and used to operate portable devices such as mobile phones or powered prosthetic limbs.



Patient Application 14–5

Recall that Ms. Brown hiked up her affected side (lifted her affected hip and pelvis) to clear her foot during swing phase. This has been shown to have a considerable energy cost as the full weight of the upper body is raised and lowered, and there are no opportunities for savings by kinetic-potential exchange.[50] Because the inability to adequately dorsiflex her foot appeared to be part of the problem, a light ankle-foot orthosis was prescribed, which held her ankle in a neutral position during swing phase. The orthosis also assisted with her inability to provide sufficient support during stance. The somewhat stiff orthosis material accomplished this by resisting dorsiflexion of her ankle during stance, as her body progressed forward over her foot.

She gained the ability to bend her knee in later stance, thus avoiding excessive hip hiking. Watch for possible drawbacks when you read the section on power and work. If the orthosis does not permit any ankle plantarflexion during push-off, she will lose the energy that would otherwise be provided by plantarflexor generation.

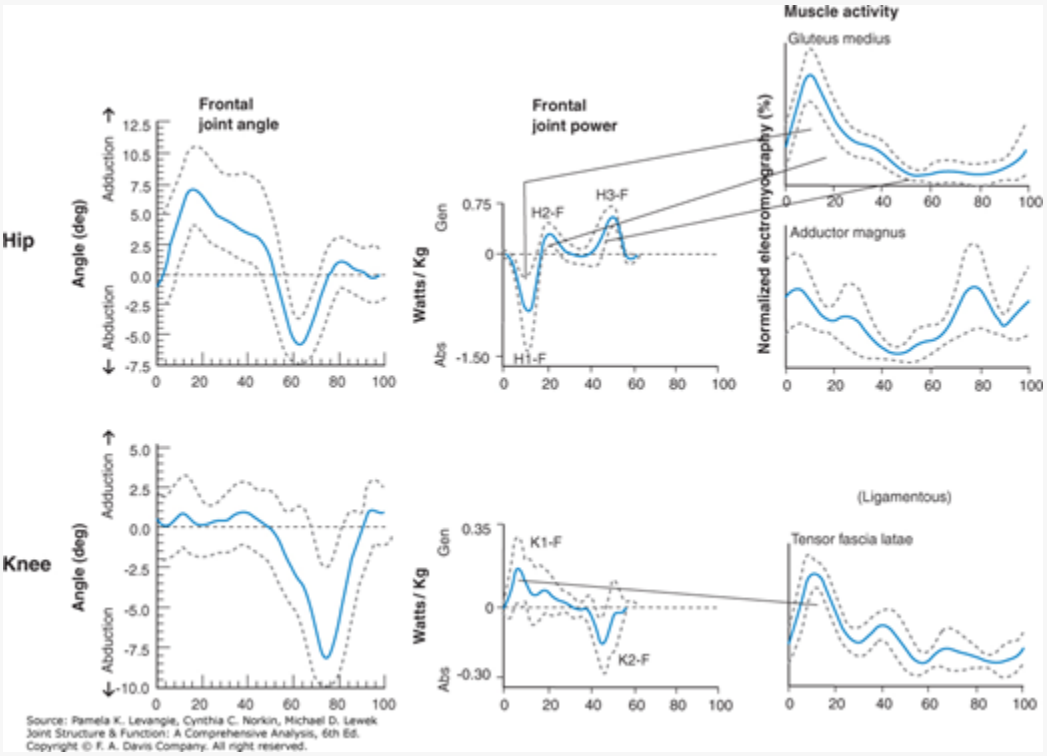
Frontal Plane Powers

In the frontal plane (Fig. 14–20), an initial period of absorption by the hip abductors (H1-F) is followed by two small bursts of positive work in the remainder of stance (H2-F, H3-F). These bursts provide fine control of the mediolateral position of the center of mass of the body. At the knee, there is a very small generation pattern during the first half of stance (K1-F), followed by a small absorption (K2-F) caused by abductor structures. Ankle power patterns are not shown as they are small and somewhat inconsistent.

FIGURE 14-20

Joint angles and joint powers of hip and knee in the frontal plane, and EMG profiles of representatives of major contributors to joint powers of hip and knee during adult gait. *(Angle profiles redrawn from Winter DA, Eng JJ, Isshac MG: A review of kinetic parameters in human walking. In Craik RL, Otis CA [eds]: Gait analysis: Theory and application, pp 263–265. St. Louis, MO: Mosby-Year Book, 1994, with permission from Elsevier. Power profiles redrawn from Eng JJ, Winter DA: Kinetic analysis of the lower limbs during walking: What information can be gained from a three dimensional model? J Biomech 28:753, 1995, with permission. Muscle activity redrawn from*

Winter DA: The biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario: Waterloo Biomechanics, 1991, with permission from David A. Winter.)



Muscle Activity

The muscle work of gait can be simplified by anticipating the muscle groups that must be functioning for specific purposes. Muscle work is logical, and the reader already knows that two major features are going to determine its patterns:

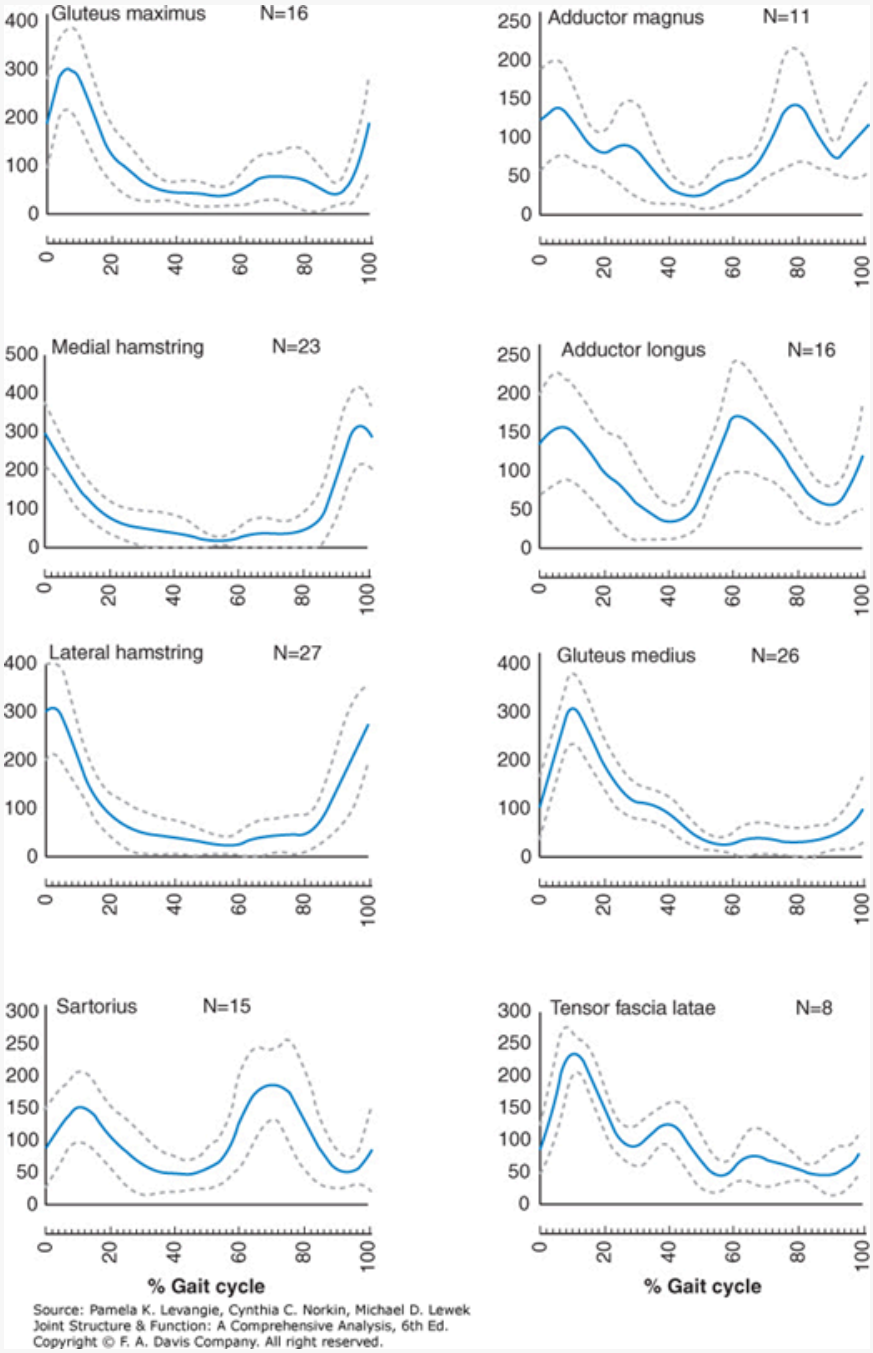
- The need to provide a support moment through stance
- The need to generate energy to move.

EMG studies of gait are used to augment the understanding provided by a link segment

analysis, to validate theoretical models that attempt to explain why muscles are needed for certain functions, and in theoretical models developed to explain the muscle activity found by EMG.[37] The EMG reported in the section that follows is derived from the classical work of Winter and is shown in **Figures 14–21** and **14–22**.[51] EMG with respective joint angles and moments appears in Figures 14–16 and 14–18, and with respective joint angles and powers in Figures 14–19 and 14–20.

FIGURE 14-21

Electromyographic activity profiles of major muscle groups of the hip during one stride of gait. Signals were derived from bipolar surface electrodes as data normalized to means for each subject. Note absence of the profile for the hip flexor, the iliopsoas muscle, which is inaccessible to surface electrodes. *(Redrawn from Winter DA: The biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario: Waterloo Biomechanics, 1991, with permission from David A. Winter.)*

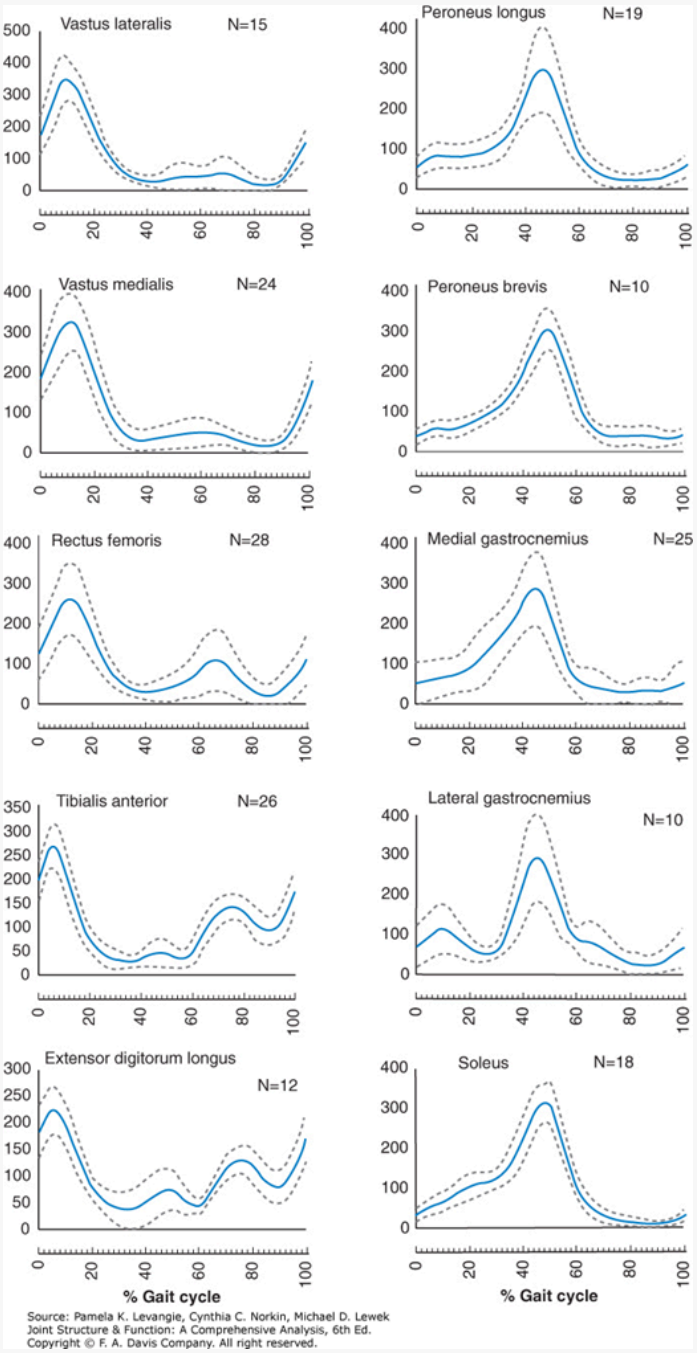


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FIGURE 14-22

Electromyographic activity profiles of major muscle groups of the knee and ankle during one stride of gait. Signals were derived from

bipolar surface electrodes as data normalized to means for each subject. *(Redrawn from Winter DA: The biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario: Waterloo Biomechanics, 1991, with permission from David A. Winter.)*



Let us follow the logic of what muscle work around the hip, knee, and ankle is expected if we consider what we know about the support moment (see Figs. 14–21 and 14–22). We already have discussed the fact that the **hip extensor muscles** produce an extensor moment early in stance. **Knee extensor muscles** produce an extensor moment during

loading, and then **flexor moments** are produced at both the hip) and knee before they bend in terminal stance in preparation for swing phase. While these two muscle groups are flexing the segmented system, the **ankle plantarflexors** take the lead and provide support and forward propulsion. We should not be surprised, then, to find activity in the hip extensors **(hamstrings)** early in stance and knee extensors **(quadriceps)** almost immediately after, followed by a smoothly increasing contraction of the **ankle plantarflexors (soleus, medial** and **lateral gastrocnemius muscles** and other minor contributors) that continues until mid-push-off and then declines and ceases at about 60% of the cycle. With the exception of the ankle plantarflexor activity during terminal stance (which is primarily related to the forceful push-off required to propel the body forward) most of the muscle work described above relates to supporting the body during stance, or eccentrically controlling joint rotation caused by external forces (for example, quadriceps activation to control the rate of knee flexion during loading; see Expanded Concept 14–4 for further discussion).



Expanded Concepts 14–4

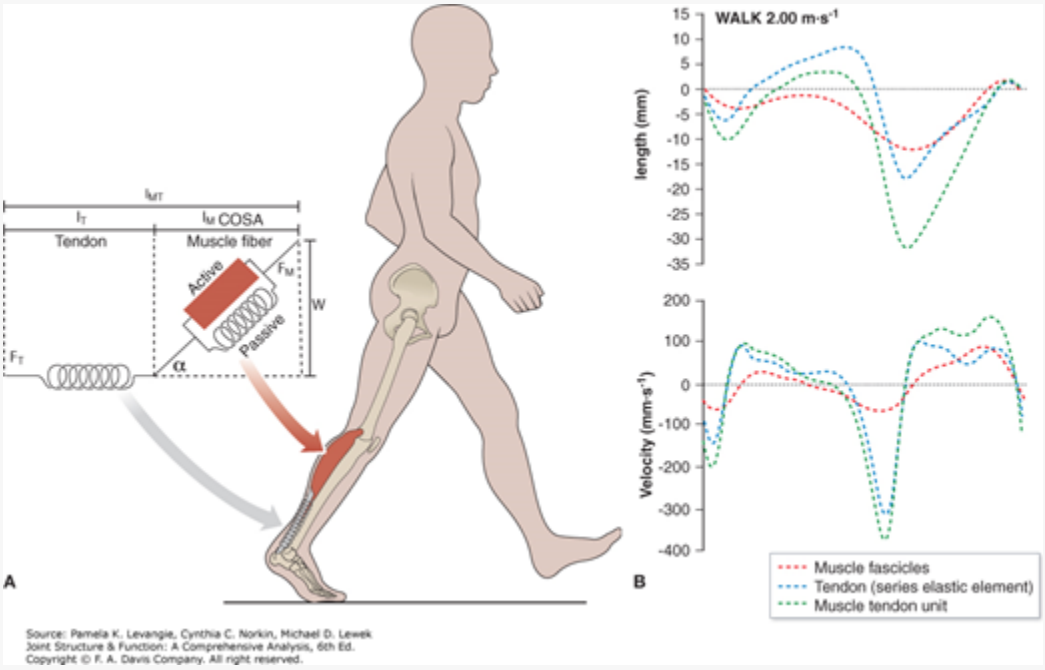
Function of the Plantarflexor Musculotendinous Unit During Gait

Although tendons are often thought of as rigid struts that pass the muscle's force to the bones, the reality is that there is a fair amount of elasticity in tendons. The Achilles tendon is a great example of this. As previously described, the plantarflexor musculotendinous unit (both muscle and tendon) lengthens throughout much of stance, followed by a rapid shortening of the entire unit. Ultrasound studies performed during walking, however, have revealed that the muscle fibers of the gastrocnemius and soleus remain largely isometric during this period. Rather than having the muscle fibers shorten and lengthen, our bodies use the Achilles tendon like a spring. During stance the tendon is therefore stretched (elongated) like a spring, which allows it to return the stored energy during push-off as the entire musculotendinous unit shortens (Figure 14–23). This is metabolically more efficient

than having the muscle fibers shorten and lengthen to produce joint power

FIGURE 14-23

A. Muscle tendon unit shown as Hill-type muscle model. **B.** The elastic nature of the Achilles tendon is evident as the muscle fascicles function isometrically to load the tendon.



Now let us look for the muscle work that is responsible for the main bursts of positive work, relating the muscle work shown in Figures 14–21 and 14–22 to power profiles shown in Figures 14–19 and 14–20. In the sagittal plane, we note that the **hip extensors (gluteus maximus, medial hamstring,** and **lateral hamstring muscles)** are active in early stance (H1-S); indeed, they were serving the function of providing support during that period. The small energy-generating K2-S that peaks in early stance is reflected in activation of the **quadriceps (vastus medialis, vastus lateralis, vastus intermedius,** and **rectus femoris muscles).** Recall that the largest contribution to the work of walking

comes from the **ankle plantarflexors** (largely **soleus, medial,** and **lateral gastrocnemius muscles).** These muscles work eccentrically (lengthening) from early stance until about 40% of the gait cycle, when they are eccentrically controlling the forward movement of the tibia over the talus as the upper body passes over the fixed foot. At about 40% of the gait cycle, they produce a burst of concentric activity (A2-S), ending at toe-off. A similar sequence of first eccentric and then concentric activity occurs in the **hip flexors: iliopsoas** and **rectus femoris muscle.** First, they lengthen, producing an energy-absorbing contraction as the hip extends (H2-S); then the muscle force overcomes the external moment from the opposing ground reaction force and begins to act concentrically, causing an energy-generating "pull-off" phase (H3-S). The **iliopsoas** muscle is the major hip flexor, but it is inaccessible to surface electrodes. However, the **rectus femoris** muscle, being the only one of the quadriceps muscles that crosses the hip, also shows hip flexor function, and its activity in terminal stance correlates with that of iliopsoas in terminal stance. We can see **rectus femoris** activity peaking around 70% of the cycle, reflecting this hip-flexor function.

Now let us look at the major energy-absorbing phases and the muscle groups that are responsible. K1-S, occurring before 20% of the cycle, is the eccentric phase of the **knee extensors (vastus lateralis, medialis, intermedius,** and **rectus femoris),** which precedes its concentric K2-S energy-generating phase. We have discussed H2-S, the eccentric action of the **hip flexors (iliopsoas** and **rectus femoris),** during midstance and terminal stance. Note that the **knee extensor** and the **hip extensor** muscles begin their contraction at the end of swing phase, although at this time the dominant moments are being provided by the knee flexors. K3-S is a small energy-absorbing knee extensor moment occurring when the knee is flexing rapidly (~50% to 70% of cycle), and this is

reflected in low levels of contraction of the **vastus muscles,** particularly the **rectus femoris,** which, because it crosses both hip and knee, is active at that time as a **hip flexor** (H3-S). K4-S, energy absorption of the **knee flexors** at the end of swing, is reflected in EMG records as activation of the **medial** and **lateral hamstrings.** Note that the **gastrocnemius** muscles, which cross the knee as well as the ankle, also begin activity in late swing phase. At the ankle, A1-S absorption through much of early stance and midstance is attributable to **ankle plantarflexor** activity, which we have already discussed.

Now let us see whether we have missed any major EMG features shown in Figures 14–21 and 14–22 by deducing muscle activity from what we know about support moment and power profiles. First, in the sagittal plane, we have not considered the **ankle dorsiflexors (tibialis anterior, extensor digitorum longus,** and **extensor hallucis longus muscles),** which are eccentrically active in early stance before foot flat and act to control the lowering of the foot to the floor. They are active again during swing phase to hold the foot at a neutral angle to prevent the toes from catching on the ground, and show varying but small levels of activity at other times of the cycle, probably positioning the foot.

We have not so far noted the activity in the frontal plane of the stance phase hip abductor **gluteus medius,** along with **gluteus minimus** and **tensor fasciae latae** muscles (not shown). These muscles control the lateral drop of the pelvis toward the side of the swinging leg. Activity of these muscles diminishes during midstance and ceases when the opposite limb has contacted the ground.[21] Weakness of the hip abductor muscles will result in an observable drop of the pelvis to the contralateral side (a positive

Trendelenburg sign), unless compensated for by other mechanisms such as trunk lean over the stance limb, which acts to reduce the work required by the weakened hip abductors.



Foundational Concepts 14–3

Relationships Among External Forces, Internal Moments, Muscle Activation, and Resultant Kinematics

Now that we can identify what muscles are active and when, it is important to know *why* they are active when they are. Recall that the resultant movement of a particular joint (referred to as kinematics) is dictated by the net linear and angular forces that it experiences. Although muscle activation is an obvious source of these forces, so too are external forces such as gravity and the GRF.

Consider the brief period after initial contact. Looking at Figure 14–16, one can see that the ankle joint plantarflexes approximately 5°. Normally, one would expect that plantarflexion would result from activation of the triceps surae group (gastrocnemius and soleus muscles). However, close inspection of the sagittal joint moment graph and the EMG patterns clearly shows predominant dorsiflexor activity at this time. How is this explained? First, at this point the GRFV is situated *posterior* to the ankle joint center and directed *upward,* creating an external plantarflexion moment tending to plantarflex the ankle. Second, the center of mass of the foot segment is situated *anterior* to the ankle joint and directed *down.* Together, these two forces generate the external moments required to plantarflex the ankle (Fig. 14–24). It would not be energetically efficient to generate power from concentric triceps surae activation; rather, energy absorption (identified as A1-S in the power curve) and eccentric tibialis anterior muscle activity to control the rate of plantarflexion is exhibited.

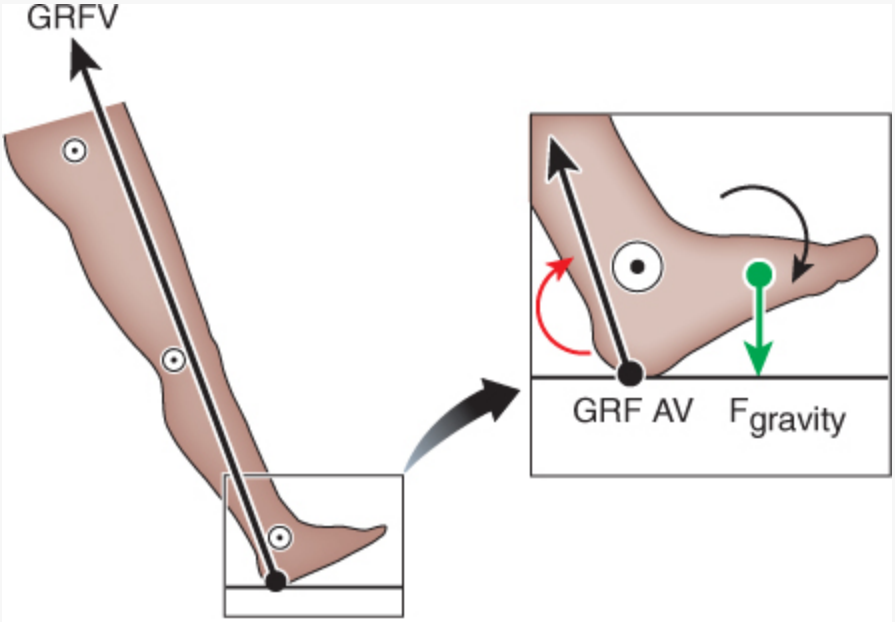
The same concept is prevalent during knee flexion in early stance (predominantly "caused" by the GRFV passing posterior to the knee, resulting in a tendency to create knee flexion and requiring eccentric activity of the quadriceps), and at the ankle joint throughout midstance as the anterior positioned GRFV passively creates the dorsiflexion that is required. Indeed, recall that the gastrocnemius acts eccentrically up until about 40% of the gait cycle to control the movement of the tibia over the talus, and only acts concentrically (and to a much larger extent) when the ankle is

required to begin plantarflexion. The large increase in gastrocnemius activity at this point is because of a need to (1) generate power to plantarflex the ankle, and (2) overcome the external ankle dorsiflexion moment created because of the anterior position of the GRFV.

It is imperative to note that the relationships discussed here are to be used for understanding the relationship between external forces and the internal muscle requirements. Calculation of the internal and external moments by simply multiplying the magnitude of the GRFV by its moment arm to a joint should never replace a full inverse dynamics approach.

FIGURE 14-24

Relationship between the GRFV and gravitational forces and resultant sagittal plane rotation at the ankle joint after initial contact. By virtue of the GRFV passing posterior to the ankle joint center and directed upward, an external ankle plantarflexion moment is generated. Similarly, gravity acts downward at the center of mass of the foot, also generating an external plantarflexion moment. Because the ankle actually undergoes plantarflexion at this time, two key aspects can be taken from this situation: (1) the magnitude of the external (in this case plantarflexion) moments are larger than any internal moments, and (2) internal muscle activity is likely generated by the dorsiflexors to control the plantarflexion “caused” by the external moments. Indeed, this is detailed nicely in the ankle power and EMG curves in Fig 14–19.



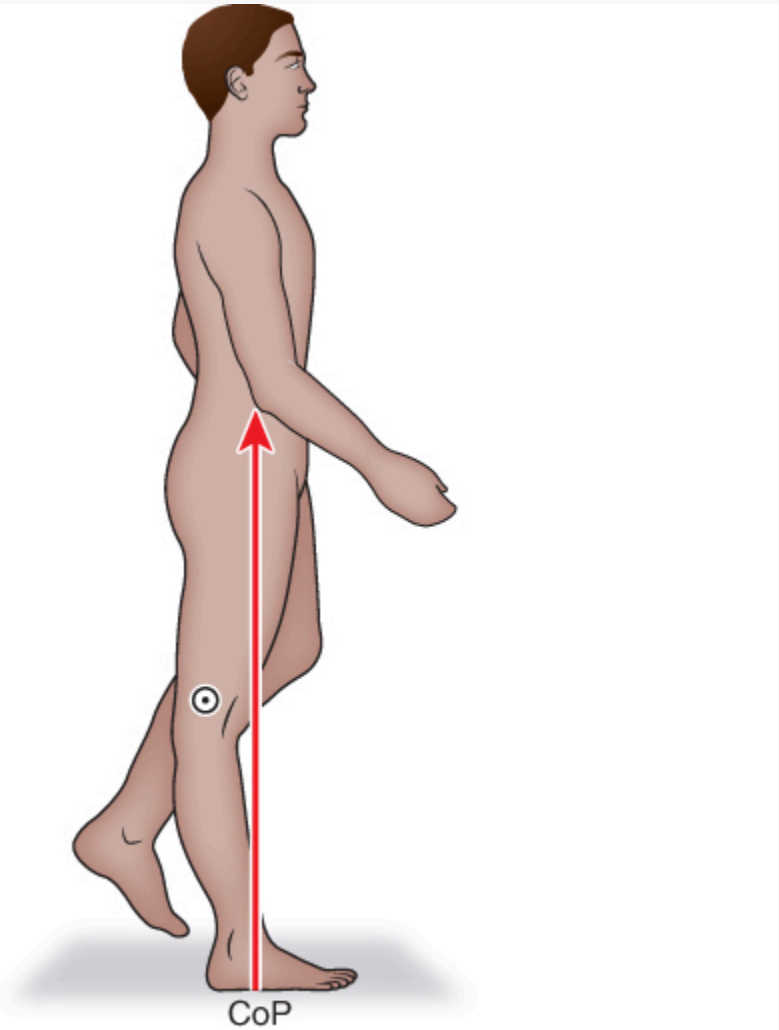
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Patient Application 14–6

At heel-off (~40% of gait cycle), the GRFV tending to extend the hip and knee (see Fig. 14–12) is consistent with the opposition provided by the hip flexor moment (iliopsoas and rectus femoris muscles) and the knee flexor moment (gastrocnemius muscles) (see Fig. 14–16). Consider the effects of a larger than normal tendency to extend the knee, which occurred, for example, when Ms. Brown thrust her knee back into full extension in an effort to gain knee stability (**Fig. 14–25**). Because the knee flexors that are active at that time (gastrocnemius muscles) did not overcome this excessive moment, Ms. Brown had difficulty flexing the knee, which prevented flexion of both the hip and the ankle and reduced the opportunity to generate work from the ankle at A2-S and the hip at H3-S. Avoiding this “knee locking” in stance by rigorous encouragement of knee flexion at the end of stance and by temporary use of an ankle-foot orthosis was important in Ms. Brown’s gait re-education.

FIGURE 14-25

GRFV passing excessively anterior to the knee joint center, making it difficult for the person to flex the knee.



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Expanded Concepts 14–5

What Gait Information Is Important?

Given the availability of information about walking, it is often not clear what information is helpful for any given situation. First, it is important to determine why you want the information. Usually the reasons include one or more of the following:

(1) to gain an understanding of normal or pathological gait, (2) to assist movement diagnosis and identify specific causes of pathological gait, (3) to inform treatment selection, and (4) to evaluate the effectiveness of treatment. Second, you may ask what gait measures are important for the situation. For example, if you want to know whether energy costs are decreased with provision of an ankle orthosis, a measure of self-selected speed of walking may be sufficient. If you want to know which muscle groups could be exploited to gain increased walking speed in treating a person with a neurological condition, you would want to see a power analysis or EMG assessment. If you wished to know whether a new ankle-foot orthosis really did return energy during push-off, you would also want a power analysis. If you wanted to determine whether surgery to realign the tibia and fibula was successful in decreasing a varus or valgus moment on the knee, you would want a moment analysis in the frontal plane. There is a tendency for power analyses to be more useful in neurological conditions and moment analyses in musculoskeletal conditions, particularly those involving pain.

We have so far not noticed **adductor longus** and **brevis muscles,** which act in both the frontal and sagittal planes, and show two fairly equal peaks of activity at ~10% and 65% to 80% of the gait cycle. The first peak is concurrent with the **hip abductors** and may be providing stabilization of the hip joint; the second occurs early in swing, providing hip flexion to assist **iliopsoas** and **rectus femoris muscles.** Further information can be found in the literature.[35,36,52] Trunk muscle activity is discussed in a later section.

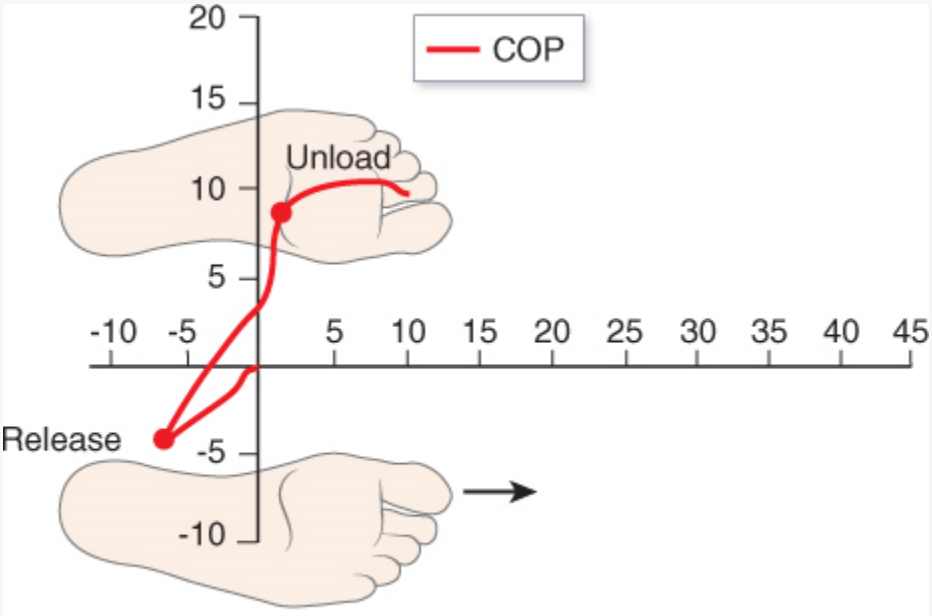
Gait Initiation and Termination

The previous sections have discussed gait as a continuing activity. However, both starting to walk and stopping walking require a certain sequence of motor events. These events are interesting because they are quite different from continuing gait, they are potentially destabilizing to a person's balance, and yet they are essential to the function of walking.

Gait initiation is defined as a stereotyped activity that includes the series or sequence of events that occurs from the initiation of movement to the beginning of the gait cycle. Gait initiation begins in the erect standing posture with an inhibition of the gastrocnemius and soleus muscles closely followed by activation of the tibialis anterior muscles.[53] Bilateral concentric contractions of the tibialis anterior muscles (pulling the tibias forward over stable feet) results in a sagittal moment that inclines the body anteriorly from the ankles. Initially, the CoP shifts posteriorly, and briefly, toward the first swing foot from its resting location between the feet and anterior to the ankle joints (Fig. 14–26). As the heel of the swing foot lifts off the floor the CoP moves rapidly to the stance foot heel; then, as the swing foot toe lifts off the floor, it progresses anteriorly to the forefoot.[6]

FIGURE 14-26

Path of the CoP during gait initiation.



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Abduction of the swing hip, with activation of the gluteus medius, occurs almost simultaneously with contractions of the tibialis anterior. As weight is transferred to the stance limb, activation of gluteus medius on the stance side begins. According to Elble and colleagues, the support limb's hip, and knee flex a few degrees (3° to 10°), and the CoP moves anteriorly and medially toward the support limb.[54] A healthy individual may initiate gait with either the right or left lower extremity, and no changes will be seen in the pattern of events. In contrast, a patient will tend to favor a particular starting limb dependent upon factors such as muscle strength, ROM, and pain. The temporal and spatial patterns of gait initiation are generally preserved in the elderly, though there is a trend for displacements to be smaller, velocities lower, and co-contraction of lower leg muscles to increase.[6, 53]

Termination of gait has received much less attention than has initiation (for a review, see Sparrow and Tirosh, 2005).[7] Anticipation of stopping appears to influence EMG activity even before initial contact.[55] The body attempts to maintain the stance limb anterior to the whole body center of mass, which is initiated first in the stance limb by reducing push-off and activating hip extensors and knee extensors as energy absorbers. The reduced push-off in the stance limb is accomplished by inhibition of soleus and strong activation of the tibialis anterior.[55] In the swing limb, the vastus lateralis is usually activated first.[7] Braking forces are then produced by the swing limb at initial contact with a large soleus muscle burst to control ankle plantarflexion, and reduced activity in the tibialis anterior to inhibit ankle dorsiflexion. To maintain knee extension and stabilize the trunk, the vastus lateralis activity is continued and the erector spinae are activated.

Patient Application 14–7

Patients with hemiparesis, like Ms. Brown, demonstrate some differences in gait initiation depending on whether the step is started with the affected or unaffected limb. When the person stands on the unaffected limb and steps forward with the affected leg, the timing and pattern of events are practically the same as they would be in a nonaffected person, but when the person with paresis attempts gait initiation with the nonaffected leg, the weight must first be shifted onto the affected side. The result is an erratic pattern of events, and stability can be seriously threatened.[56]

Trunk and Upper Extremities

Trunk

The trunk remains essentially in the erect position during normal free-speed walking on level ground, varying only 1.5°.[57] Krebs and coworkers found that a forward flexion peak of low amplitude occurred near each initial contact and an extension peak of low amplitude occurred during single limb support.[58] Winter explained that at initial contact, the forward acceleration of the head, arms, and trunk is quite large and acts at a distance from the hip joint, thus producing an unbalancing moment that acts strongly to cause flexion of the trunk during weight acceptance.[51] However, an almost equal and opposite moment is provided by the hip extensors (which we have seen before in their role in support and in energy generation at H1-S). In the frontal plane, a similar situation exists. The center of mass of the head, arms, and trunk, always being medial to the center of pressure and thus the knee and hip joints, exerts a considerable moment that is balanced primarily by an internal hip abductor moment from the supporting limb (see Fig. 14–18) and is assisted by the medial linear acceleration of the hip.

Current biomechanical models typically do not distinguish between the pelvis and the

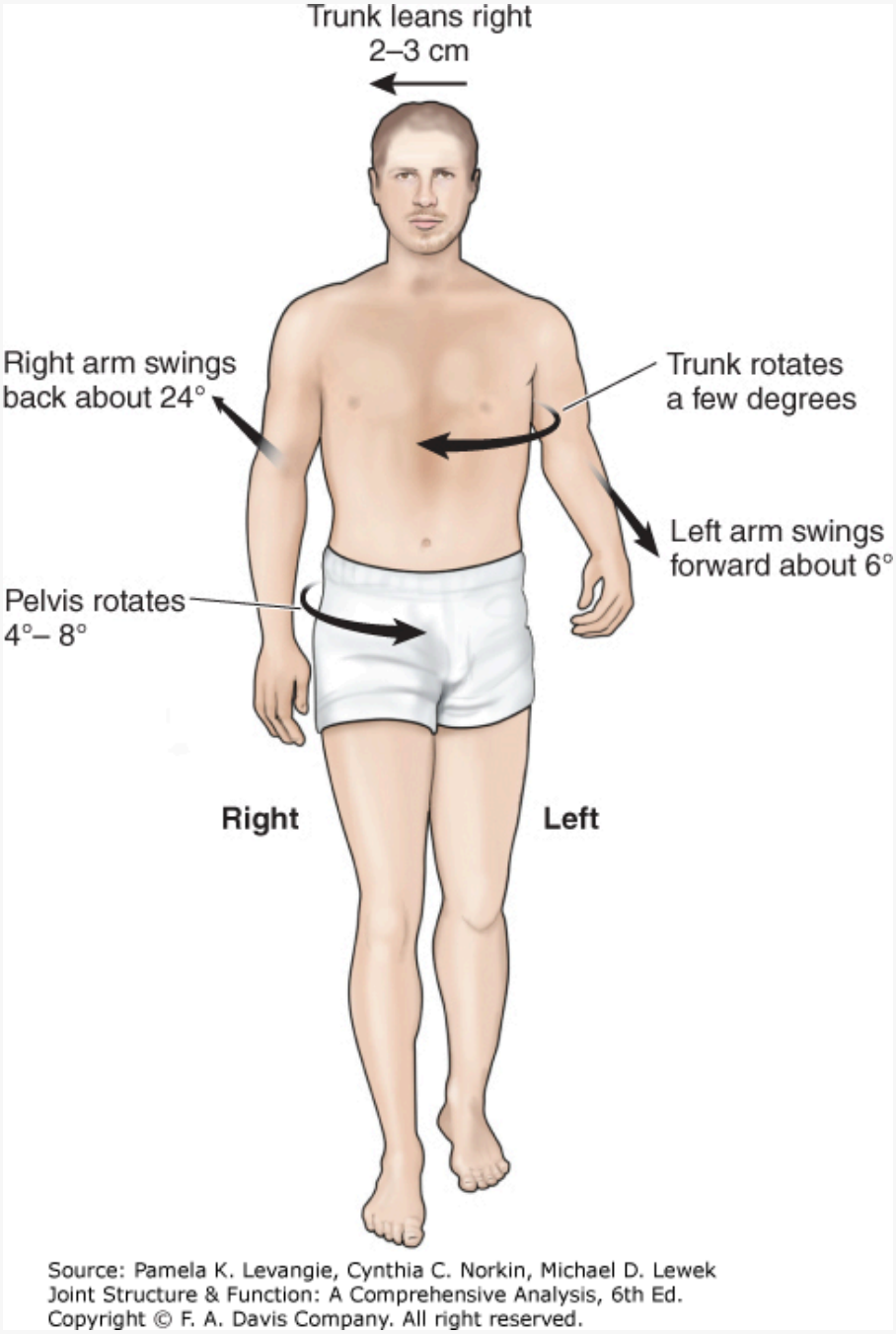
trunk. In the sagittal plane, the pelvis translates in a sinusoidal curve up and down 4 or 5 cm with each step, the low point coinciding with initial contact of each foot and the high point coinciding with midstance when the knee is most extended. In the frontal plane, the pelvis translates from side to side about the same amount toward the stance limb and rotates downward about 5° toward the swinging limb on each side during early to midstance. In the transverse plane, looking at the pelvis from above, the contralateral pelvis rotates 4° to 8° toward the stance limb during early stance, goes through a neutral position about midstance, then continues rotating the same amount, but in the opposite direction, during swing as the other limb is in stance.

When the pelvis rotates, the trunk rotates in the opposite direction with regard to the pelvis to keep the trunk directed forward. This reciprocal rotation of the trunk and pelvis is important to maintain stability and reduce energy consumption (Fig. 14–27). As the pelvis rotates forward with the swinging lower extremity, the trunk on the opposite side rotates forward as well. Actually, the thorax undergoes a biphasic rotation pattern with a reversal directly after liftoff of the stance leg. The trunk is rotated backward during double support and then slowly rotates forward during single support. This trunk motion helps prevent excess body motion and helps counterbalance rotation of the pelvis. Krebs and coworkers found that at a free speed of gait, transverse rotation reached a maximum of 9° at 10% of the cycle after each initial contact.[58] Mediolateral translations of the trunk occur as side-to-side motions (leans) in relation to the pelvis. For example, the trunk is leaning laterally or moving to the right from right initial contact to left toe-off, at which point the trunk begins a lean to the left until right toe-off. The average total ROM that occurs during the mediolateral trunk leans is about 5.4 cm from neutral.[59] Hirasaki and coworkers used a treadmill and a video-based motion

analysis system to study trunk and head movements at different walking speeds.[60] These authors found that the relationship between walking speed and head and trunk movements was the most linear in the range of walking speeds from 1.2 to 1.8 m/sec. At velocities above and below this range, head and trunk movements were less well coordinated.

FIGURE 14-27

Pelvis, trunk, and arm motion. Note that the trunk and arms rotate in a direction opposite to that of the pelvis.



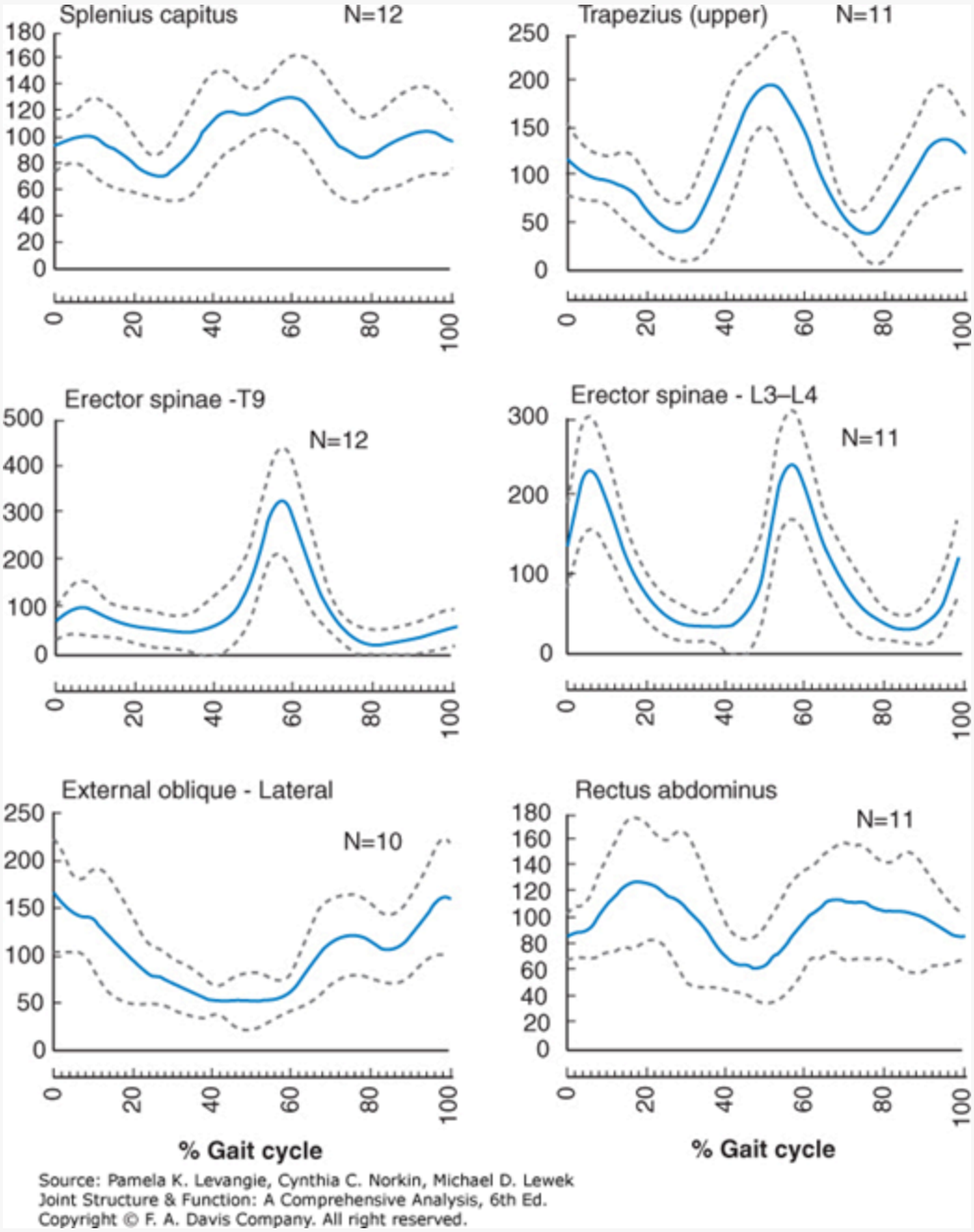
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EMG profiles and probable functional roles are shown for some trunk muscles in Figure 14–28. Accurate assessments of muscle activation from trunk musculature are difficult to obtain given the depth of many of the muscles relative to the skin, and the challenge in isolating specific single muscles in this area using surface EMG. Recent EMG studies

on the trunk muscles during gait have shown that there are subgroups of people who show similar patterns of muscle activity when cluster analysis is used on the EMG data.[61] This applies to the **internal oblique, external oblique, rectus abdominis,** and **lumbar erector spinae** muscles. Although most people showed low levels of activity throughout the gait cycle, the **internal oblique** and **erector spinae muscles** had more distinct bursts, usually occurring close to initial contact. Some researchers have shown two periods of activity for the **erector spinae muscle,** one at initial contact and the second at toe-off.[62] It is thought that its function is to oppose the unbalancing moment that acts strongly to cause flexion of the trunk during weight acceptance.

FIGURE 14-28

Electromyographic activity profiles of major muscle groups of the trunk during one stride of gait. Signals were derived from bipolar surface electrodes as data normalized to means for each subject. *(Redrawn from Winter DA: The biomechanics and motor control of human gait: Normal, elderly and pathological [ed. 2]. Waterloo, Ontario: Waterloo Biomechanics, 1991, with permission from David A. Winter).*



Upper Extremities

Detailed kinetics of the upper extremity during normal gait are not well-reported, although extensive mapping of EMG was classically performed several decades ago.[62, 63] Although the lower extremities are moving alternately forward and backward, the

arms are swinging rhythmically and are primarily controlled by the shoulder musculature, with structural support from the muscles of the scapulothoracic joint. However, the arm swinging direction is opposite to that of the legs and pelvis but similar to that of the trunk (see Fig. 14–27). The right arm swings forward with the forward swing of the left lower extremity whereas the left arm swings backward. This swinging of the arms provides a counterbalancing action to the forward swinging of the leg and helps to decelerate rotation of the body, which is imparted to it by the rotating pelvis. The total ROM at the shoulder is not very large. At normal free velocities, the ROM is only approximately 30° (24° of extension and 6° of flexion). It is clear, however, that restricting arm motion increases energy costs of walking at prescribed speeds.[64]

Recent work supports neuronal coordination of arm and leg movement during locomotion. Dietz and colleagues and Wannier and colleagues reported the behaviors of the arm to leg corresponding to a system of two coupled oscillators.[65,66] Results were compatible with the assumption that the proximal arm muscles are associated with the swinging of the arms during gait as a residual function of quadrupedal locomotion.

Treadmill, Stair, and Running Gaits

Treadmill Gait

Treadmills used for gait measurement and training have a number of advantages including a small space requirement, the availability of weight support for patient safety, and increasing sophistication of instrumentation that may include simultaneous metabolic analysis as well as embedded force platforms. Comparisons of walking on a treadmill versus overground walking have shown that treadmill walking results in

higher cadence and shorter stance times at comparable speeds, although others do not report these differences.[67–70] Differences in joint ROMs have also been reported, but these have usually been less than 2.5° and are not considered important.[71,72] Kinetic comparisons have revealed similar joint moments for treadmill and overground walking, although push-off forces and GRF maxima have generally been lower with treadmill walking, especially in the anteroposterior direction.[70,72] It is still not certain whether metabolic costs are different. Whereas Pearce and colleagues reported lower oxygen uptake in association with treadmill walking compared with overground walking at the same speed in young and older men, more recent work by Parvataneni and colleagues found significantly higher metabolic costs on the treadmill in healthy older adults when speeds on the treadmill were matched with overground speeds.[73,74] These findings are consistent with reports of higher heart rates by Greig and colleagues.[69] More research is needed to assess energetic differences between treadmill and overground walking across age groups, and to determine their cause as well as the clinical significance.

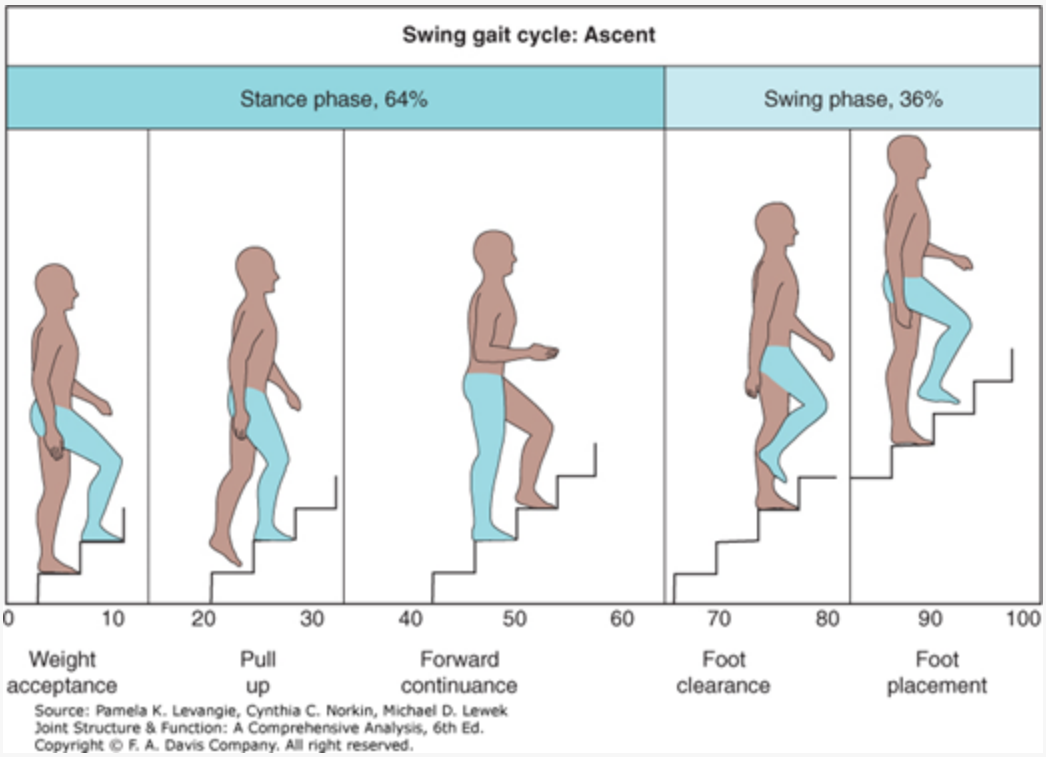
Stair Gait

Ascending and descending stairs is a basic body movement required for performing many normal activities of daily living. Although many similarities exist between level ground locomotion and stair locomotion, the difference between the two modes of locomotion may be significant for a patient population. The fact that a patient has adequate muscle strength and joint ROM for level ground walking does not ensure that the patient will be able to walk up and down stairs. Stair walking represents additional demands and may reveal differences that are not apparent in level ground walking, especially in pathological populations.[75]

Locomotion on stairs is similar to level ground walking in that stair gait involves both swing and stance phases in which forward progression of the body is brought about by alternating movements of the lower extremities. Further, the relative time spent in stance (64%) and swing (36%) is similar to walking.[76] Also, in both stair and level ground gait, the lower extremities must balance and carry along the head, arms, and trunk. McFayden and Winter (using step dimensions of 22 cm for the stair riser and 28 cm for the tread) performed a sagittal plane analysis of stair gait.[76] These investigators collected kinetic and kinematic data for one subject during eight trials. The stair gait cycle for stair ascent presented in Figure 14–29 is based on data from McFayden and Winter's study.[76] Although ankle moments were approximately the same as level ground walking, the knee extensor moment in both stair ascent and descent was approximately three times larger than that of level ground walking. This may cause particular mobility problems in patients with knee pain or weakness, which is common in such conditions as osteoarthritis and patellofemoral pain syndrome. Although the knee has primarily an absorptive function in level walking, it has a large generative role in stair ascent given the need to extend the knee to raise the body up each stair. In general, joint powers are largely generative in stair ascent and absorptive in descent for all joints.

FIGURE 14-29

Stair gait cycle.



Similar to level ground walking, the stance phase of the stair gait cycle can be divided into the three subphases and the swing phase into two subphases.[76] The subdivisions of the stance phase are weight acceptance, pull-up, and forward continuance. The subdivisions of the swing phase are foot clearance and placement. As can be seen in Figure 14–29, weight acceptance comprises approximately the first 14% of the gait cycle and is somewhat comparable to the initial contact through to the loading phase of walking gait. However, in contrast to walking gait, the point of initial contact of the foot on the stairs is usually located on the anterior portion of the foot and travels posteriorly to the middle of the foot as the weight of the body is accepted. The pull-up portion, which extends from approximately 14% to 32% of the gait cycle, is a period of single-limb support. The initial portion of pull-up is a time of instability, inasmuch as all of the body weight is shifted onto the stance extremity when it is flexed at the hip, knee, and

ankle. This has significant implications for the ability to maintain a support moment. During this period, the task is to pull the weight of the body up to the next stair level. The knee extensors are responsible for most of the energy generation required to accomplish pull-up. The forward continuance period is from approximately 32% to 64% of the gait cycle and corresponds roughly to the midstance through toe-off subdivisions of walking gait. In this subphase, the greatest amount of energy is generated by the ankle plantarflexors. Data regarding joint ROM and muscle activity for ascending stairs are presented in Tables 14–5, 14–6, and 14–7.[76] Similar findings have been reported by Protopapadaki, Costigan, Salsich, and their colleagues.[77–79] Variations between studies have been attributed to differences in stair dimensions, trunk inclination, foot contact positions, and differences in subject characteristics (such as height) and methodologies.

Table 14-5

Sagittal Plane Analysis of Stair Ascent (Fig. 14–29): Stance Phase–Weight Acceptance (0%–14% of Stance Phase) Through Pull-Up (14%–32% of Stance Phase)

Table 14-6

Sagittal Plane Analysis of Stair Ascent (Fig. 14–29): Stance Phase–Pull-Up (End of Pull-Up) Through Forward Continuance (32%–64% of the Stance Phase of Gait Cycle)

Table 14-7

Sagittal Plane Analysis of Stair Ascent (Fig. 14–29): Swing Phase (64%–100% of Gait Cycle)—Foot Clearance Through Foot Placement



Expanded Concepts 14–6

Differences Between Level Ground Gait and Stair Gait

Table 14–5 shows that considerably more hip and knee flexion are required in the initial portion of stair gait than are required in normal level ground walking. Therefore, a patient would require a greater available ROM for stair climbing (the same stair dimensions and slope) than for normal level ground walking. More important, the requirement to raise the body against gravity results in significantly greater muscle force to perform stair ascent. As a result, individuals lacking in sufficient lower body muscle strength to achieve this find stair climbing to be much more challenging. Naturally, muscle activity and joint ROMs will change if stairs of other dimensions are used.

Ascending stairs involves a large amount of positive work that is accomplished mainly through concentric action of the limb extensors. Descending stairs is achieved mostly through eccentric activity of the same muscles and therefore involves energy absorption. The support moments during stair ascent, stair descent, and level ground walking exhibit similar patterns; however, the magnitude of the moments is greater in

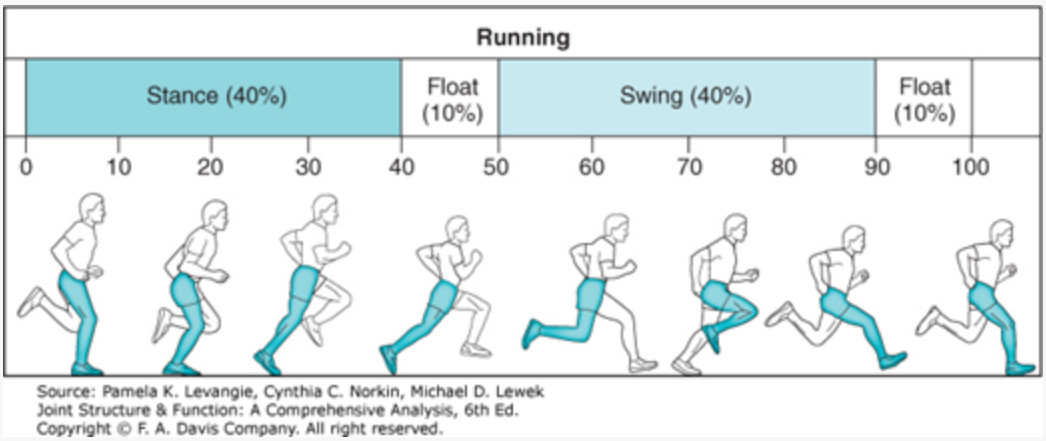
stair gait, and, consequently, more muscle strength is required.

Running Gait

Running is another locomotor activity that is similar to walking, but certain differences need to be examined. As in the case of stair gait, a patient who is able to walk on level ground may not have the ability to run. Running requires greater balance, muscle strength, and ROM than does normal walking. Greater balance is required because running is characterized not only by a considerably reduced base of support but also by an absence of the double-support periods observed in normal walking and the presence of float periods in which both feet are out of contact with the supporting surface (Fig. 14–30). The walking gait cycle in Figure 14–2 can be used to compare the gait cycle in walking with running gait. The primary difference is the relatively less time spent during stance—approximately 40% during running, although the amount decreases with increasing running speed. The percentage of the gait cycle spent in float periods will increase as the speed of running increases. Muscles must generate greater energy both to raise the head, arms, and trunk higher than in normal walking and to balance and support them during the gait cycle. Muscles and joint structures must also be able to absorb more energy to accept and control the weight of the head, arms, and trunk.

FIGURE 14-30

Running gait cycle.



For example, in normal level ground walking, the magnitudes of the vertical GRFs at initial contact are approximately 70% to 80% of body weight and rarely exceed 120% of body weight at any point of the gait cycle.[80] However, during running, the GRFs at initial contact have been shown to reach 200% of body weight and increase to 250% of body weight during the running cycle. Furthermore, the knee is flexed at about 20° when the foot strikes the ground. This degree of flexion helps to attenuate impact forces but also increases the forces acting at the patellofemoral joint. In addition, the base of support in running is considerably less than in walking. In walking, the step width dictates the base of support, and is about 2 to 4 inches, whereas in running, both feet fall in the same line of progression, and so the entire center of mass of the body must be placed over a single support foot.

Joint Motion and Muscle Activity During Running

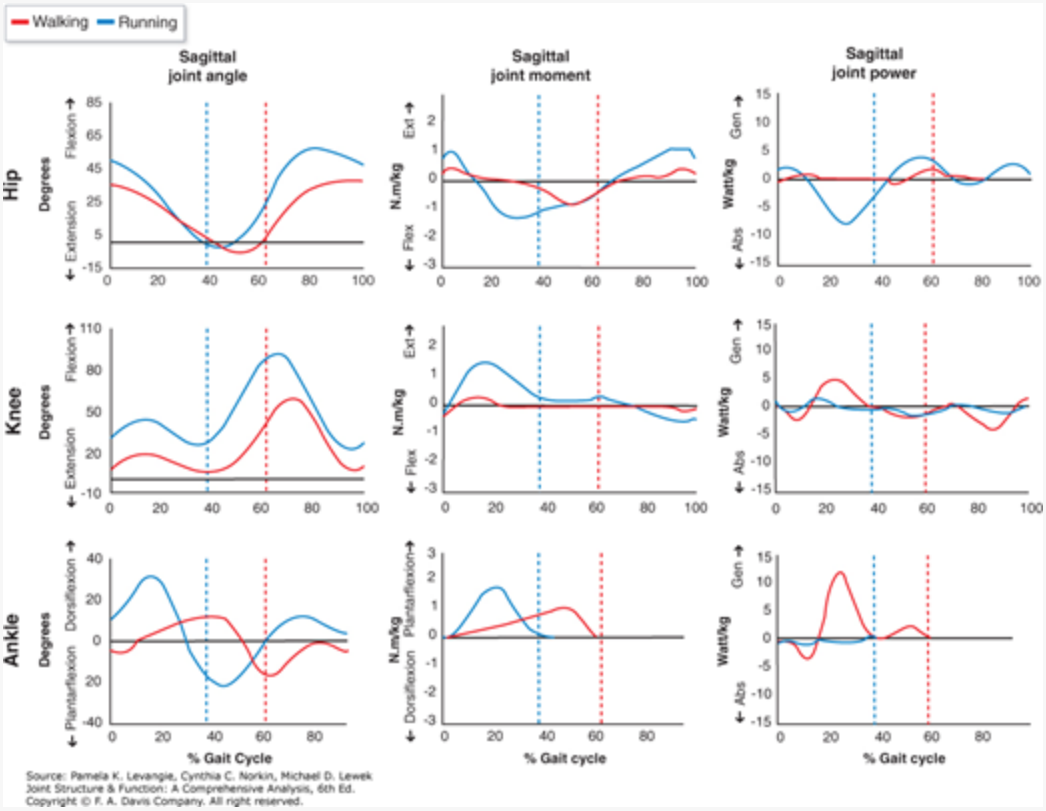
JOINT MOTION

The ROM is highly variable between people and is influenced by factors such as running speed and footstrike pattern. In general, joint ROM will increase as running speed increases for all joints. As a result, specific values for each joint are difficult to

determine across a group of people. Values listed below provide an overview of running gait parameters, and are estimates only. A general comparison of joint angles, moments, and powers is shown in Figure 14–31 and provides a general overview of the differences between running and walking.[81] One main difference is that toe-off occurs much earlier during running—approximately 40% of the gait cycle, as compared with 60% for walking. Another major difference is the disappearance of double support periods, and the emergence of a "float period," in which no feet are in contact with the ground.

FIGURE 14-31

Sagittal plane joint angles, moments, and powers for running (blue line) and walking (red line). Joint angles are plotted with flexion and dorsiflexion positive. Moments are internal moments normalized to body mass, plotted with extensor and plantarflexor positive. Powers are normalized to body mass, plotted with generation positive and absorption negative. The vertical blue line near 40% of gait cycle represents toe-off for running; the vertical red line near 60% of gait cycle represents toe-off for walking. *(Joint angles, moments, and powers for running redrawn from Novacheck TF: The biomechanics of running. Gait Posture 7:77, 1998, with permission from Elsevier.)*



At the beginning of the stance phase of running (non-sprinting), the hip is in about 45° of flexion at initial contact and extends during the remainder of the stance phase until it reaches about 20° of extension just after toe-off.[81] The hip then flexes to reach about 55° to 60° of flexion in late swing. Just before the end of the swing phase, the hip extends slightly to about 45° in preparation for subsequent initial contact. The knee is flexed to about 20° to 30° at initial contact and continues to flex to 30° to 40° during the loading response. Thereafter, the knee begins to extend, reaching approximately 20° of flexion just before toe-off. During the swing phase and initial float period, the knee flexes to reach a maximum of approximately 90° to 100° in the middle of the swing phase. In late swing, the knee extends to 20° to 30° in preparation for initial contact. In Figure 14–31, note these differences in joint angles from level ground walking. At each joint, the

maxima for running exceed those of walking. In the case of the hip, only flexion range is increased. The knee range in running is not very different from that in walking, but it takes place in approximately 25° more flexion.

The ankle joint motion can be affected considerably by the chosen footstrike pattern. The two primary footstrike patterns are a forefoot strike (forefoot hits the ground first) and a rearfoot strike (rearfoot/heel hits the ground first). Differences in the ankle kinematics between these two types of footstrike patterns will be most apparent as weight is accepted onto the limb from initial contact into midstance. A runner who lands with a forefoot strike will hit the ground at initial contact with approximately 10° to 15° of plantarflexion, and proceed to dorsiflex the ankle until foot flat is reached (or approached) around midstance. In contrast, a runner who initially strikes the ground with the rearfoot will exhibit approximately 15° of dorsiflexion, and proceed to plantarflex until midstance when foot flat is attained. Plantarflexion reaches a maximum of 25° in the initial period of the swing phase. Throughout the rest of the swing phase, the ankle dorsiflexes to prepare for initial contact (either plantarflexed or dorsiflexed depending on footstrike pattern). The footstrike pattern can also influence knee kinematics with a forefoot strike typically associated with more flexed knees and rearfoot striking associated with more extended knees during early stance.[82]

The whole extremity begins to medially rotate during the swing phase. At initial contact, the extremity continues to medially rotate and the foot pronates. Lateral rotation of the stance extremity and supination of the foot begins as the swing leg passes the stance limb in midstance. The ROM in the lower extremities needed for running, in comparison with the ROM required for normal walking, is presented in Table 14–8. The largest

differences in the total ROM requirements between the two activities appear to be at the knee and hip joints. At the knee joint, up to an additional 30° of flexion is required for sprinting versus walking. At the hip joint, sprinting requires about twice the amount of motion that was needed for normal walking.

Table 14-8

Typical Peak Kinematic Values at the Hip, Knee, and Ankle: Comparison Between Running and Walking[81]

MUSCLE ACTIVITY

As may be expected because of the increased speed and external forces associated with running compared with walking, there are increased demands on the lower limb musculature. In general, muscle activation patterns are similar, but larger in magnitude. Importantly, however, factors such as footstrike pattern greatly influence the biomechanical characteristics of running.[82] The **gluteus maximus** and **gluteus medius** muscles are active both at the beginning of the stance phase and at the end of the swing phase. The **tensor fasciae latae** muscle is also active at the beginning of stance and at the end of swing as well as between early and midswing. The **adductor magnus** muscle shows activity for about 25% of the running cycle from terminal stance through the early part of the swing phase. Activity in the **iliopsoas** muscle occurs for about the same percentage of the gait cycle as the **adductor longus** muscle, but iliopsoas activity also occurs during the swing phase from about 35% to 60% of the running cycle.

The **quadriceps** muscles become active prior to initial contact in preparation for loading, and then act eccentrically during the first 10% to 20% of the stance phase to control knee flexion when the knee is flexing rapidly. The quadriceps ceases activity after the first part of stance, and no activity occurs until the last 10% to 20% of the swing phase, when concentric activity begins to extend the knee (to 40° of flexion) in preparation for initial contact. The **hamstrings** are active for 20% to 30% prior to initial contact, and throughout the first 20% to 30% of stance. During the period of early stance, the knee is flexing and the hip is extending, and the hamstrings act to extend the hip and control the movement of the knee. In late swing, the hamstrings contract eccentrically to control knee extension and to re-extend the hip.

As described above, the kinematics of the ankle joint will differ depending on the footstrike pattern at initial contact. As a result, the muscle activation requirements will differ accordingly. For a forefoot striker who hits the ground in plantarflexion, the **gastrocnemius** muscle will immediately activate eccentrically to control the rate of heel descent (i.e., dorsiflexion). In contrast, the dorsiflexion that is exhibited at initial contact in a rearfoot striker will be accompanied by eccentric **tibialis anterior** activation to control the lowering of the forefoot as the ankle plantarflexes. This is similar to what is observed in walking, though the magnitude of the activation will be larger in running. Also similar to walking, concentric tibialis anterior activity is present through much of swing to ensure toe clearance.

The differences in joint kinematics and muscle activation between rearfoot and forefoot strike patterns are important to understand because of their implications for injury. There is a growing body of literature that is comparing injury rates, and importantly,

types, based on footstrike pattern. Indeed, while “running injuries” have typically been studied and common running injuries have been identified, such as patellofemoral pain syndrome, tibial stress syndrome, and plantar fasciitis, recent research has shifted the focus to examine differences in loading rate and the resultant injury based on footstrike pattern.[83] For example, higher impact loading in a rearfoot striker may increase the risk of injuries such as tibial stress fractures, whereas the repetitive eccentric loading of the Achilles tendon in a forefoot striker may increase the risk of tendinopathy of the plantarflexors.[84,85] In general, a rearfoot striker will tend to load the knee extensors more than a forefoot striker, whereas a forefoot striker will increase the load across the ankle plantarflexors. This shift in joint work between the ankle and the knee has implications for injury and rehabilitation. Further, the effect of different types of footwear has received much interest with the recent resurgence of minimalist shoes/barefoot running.[86] However, this is a new area of research and longer-term follow-up studies are needed to best determine a cause and effect relationship.

Moments and Powers During Running

In Figure 14–31, note the differences in internal moments and powers for running from those of walking (see Figs. 14–16 and 14–19). In all joints, the moments are greatly increased, particularly the knee extensor moment in early stance. Similarly, all the power generators are greatly increased in running. The chief generators are, again, A2, H3, and H1, but an additional source, K2, is now important. Note that the knee extensors in early stance (K2) are important in running. The absorption phases typical of walking are increased at all joints in running.

Effects of Age and Sex On Gait

Age

The gait of young children has received less attention than that of adults. The relatively few studies of children's gait that have been conducted have shown that the age at which independent ambulation begins is extremely variable among individuals and that this variability continues throughout the developmental stages of walking. Cioni and coworkers found that for 25 full-term infants, the age at which independent walking was attained (ability to move 10 successive steps without support) varied between 12.6 and 16.6 months.[87] In the first stage of independent walking, none of the toddlers had heel strike, reciprocal arm swinging, or trunk rotation. However, 4 months after attainment of independent walking, nearly half of the children had heel strike, and nearly two-thirds had reciprocal arm swinging and trunk rotation.

The toddler has a higher relative center of mass than does the adult and walks with a wider base of support, a decreased single-leg support time, a shorter step length, a slower velocity, and a higher cadence in comparison with normal adult gait. A study of 3- and 5-year-old children showed that some relationships between these variables were similar to the relationships found in adult gaits.[88] For example, as a group, the 3- and 5-year-old children showed significant increases in stride length adjusted for leg length, step length, and cadence from a slow to a free speed and from a free to a fast speed of gait. In a study that included children from 6 to 13 years of age, Foley and associates reported that the ROMs for flexion and extension of the joints of the lower extremities were almost identical to the values obtained for adults.[89] However, linear displacements, velocities, and accelerations were found to be consistently larger for these children than they were for adults.[89]

Sutherland and colleagues, who studied 186 children from 1 to 7 years of age, suggested that the following five gait parameters could be used as indicators of gait maturity: duration of single-limb support, walking velocity, cadence, step length, and the ratio of pelvic span to ankle spread (the ankle spread is the inter-ankle distance measured during periods of double support, and is indicative of base of support).[90] Increases in all of these parameters except for cadence are indicative of increasing gait maturity. In Sutherland and colleagues' study, the duration of single-limb stance increased from about 32% of the gait cycle in 1-year-olds to 38% in 7-year-olds (normal mean adult value is ~40%).[90] Walking velocity also increased steadily, whereas cadence decreased with age.[90] Beck and colleagues found that time and distance measures and GRF measurements depended on the speed of gait and the age of the child.[91] Increases in height were the major factors in determining changes in time and distance measures with age. Average stride length was 76% of the child's height at a walking speed of 1.04 m/sec, regardless of a child's age. It is generally agreed that children's gait patterns have matured at least by the age of 7 years, with certain variables considerably earlier.[91, 92] Studies involving young children are difficult to perform and often complicated by the fact that the child's musculoskeletal and nervous systems are in various stages of development.

In contrast to the dearth of gait studies of young children, the effects of aging on gait continue to be the subject of many studies.[18,43,93] Some of this interest in older adult gait has been prompted by the large number of falls and subsequent hip fractures experienced by older persons, as well as the increase in incidence of chronic diseases affecting gait function, such as osteoarthritis. Fifty percent of older persons who were able to walk before a hip fracture are not able to either walk or live independently after

the fracture.[94] Furthermore, it is estimated that an older person experiences at least two falls per year, with the incidence higher in women and the rate increasing with age.[94,95] Increased sway and physiological decline are thought to be implicated and many falls occur during walking and while turning the head.[96] Therefore, many studies are directed toward determining what constitutes normal older adult gait and whether falls are caused by deficits in motor functioning or control, or by other deficits that may accompany normal aging.[97]

The use of different age groups and levels of activity (sedentary versus active groups) among investigators has made it difficult to draw definitive conclusions about the effects of normal aging. With respect to temporal and kinematic variables, older adult groups, in comparison with younger groups, usually demonstrate a decrease in natural walking speed, shorter stride and step lengths, longer duration of double-support periods, and smaller ratios of swing to support phases, all of which are thought to increase stability.[18, 98, 99] Himann and associates found that between 19 and 62 years of age, there was a 2.5% to 4.5% decline in the normal speed of walking per decade for men and women, respectively.[98] After age 62, there was an accelerated decline in normal walking speed, that is, a 16% and 12% decline in walking speed for men and women, respectively.[98] Winter and associates, comparing fit and healthy elderly subjects with young adults, found that the natural cadence in older adults was no different from that in young adults but that the stride length of the older adults was significantly shorter, and both stance time and the double support times were longer in the older adults than in young adults.[51] A significantly higher horizontal heel velocity at initial contact was also reported, which would increase the potential for slip-induced falls, which occurred even though gait speed in the older adults was lower than that of

the young subjects. Kerrigan and associates found that older persons, in comparison with younger persons, had reduced plantarflexion ROM, peak hip extension ROM, and increased anterior pelvic tilt, and suggested that subtle hip flexion contractures and plantarflexor weakness might be causes of the joint changes in older adults.[99]

Differences in kinetic measures of gait have also been reported. Winter and colleagues reported less vigorous push-off by ankle plantarflexors in power analyses, as did Kerrigan and associates.[99] Mueller and coworkers found that plantarflexor peak torque and ankle dorsiflexion were interrelated.[100] These authors suggested that walking speed and step length might be improved by increasing ankle plantarflexor peak torque and dorsiflexion ROM.[100]

Strengthening and conditioning programs aimed at changing kinetics typically show similar results to those of Lord and associates, who conducted an exercise program for women 60 years of age and older.[101] After the program, the authors found significant increases in cadence and stride length, as well as reductions in stance time, swing time, and stance duration. However, detraining may be substantial. Connelly and Vandervoort showed a strength decline of 68.3% one year after a quadriceps training program ended for a group of elderly persons having a mean age of 82.8 years.[102] The speed of self-selected gait in this group also declined by 19.5%.

That a less efficient gait is typical of aging is certain, although the importance of possible causes awaits further research. Winter reported both lower push-off work by the ankle plantarflexors and higher levels of absorption by the knee extensors (K3) at the same time in the gait cycle.[51] Although several causes for the lower push-off are possible, the piston-like action of a strong push-off may be perceived as more

destabilizing, and avoided by older subjects. The reduced amount of work done by the plantarflexors to generate the work of walking may itself explain much of the inefficiency of the gait of older people, as shown by Kuo.[103] Using a simple theoretical model, Kuo persuasively showed that using larger amounts of ankle plantarflexor work (A2) is much more efficient than using the other two muscle groups, the hip extensors (H1) and hip flexors (H3), to generate the positive work of walking.[103] Less plantarflexor work and more hip work is typical of the older walker, and hence would be expected to be much less efficient. Note, however, that the increased absorption by the knee extensors also reported by Winter would also produce a less efficient gait.[51] There is also mounting evidence for increased co-contraction of antagonist muscles in the gait of older subjects, which would reduce efficiency.[104] In addition, age-related changes appear to affect balance, and the resulting compensations may also explain some of the increased energetic cost of walking in older adults.[105]

Although the implications of gait assessment and performance in young people may not be obvious, walking ability has important implications for health in the older adult population. Measures of walking are not only indicators of independence and general health, but also have predictive value for future health.[106] Self-paced gait speed is the most common outcome measure for gait and reflects the ability to transport the body from one place to another in a timely manner. Perry and colleagues have suggested that an average self-paced walking speed of 0.4 m/sec is the minimum criterion for limited community ambulation, and 0.8 m/sec for unlimited community ambulation.[41] Daily step counts are also a new and popular method of assessing walking function. It has been suggested that less than 5,000 steps a day reflects a sedentary lifestyle and more than 12,000 a highly active lifestyle.[107] Mean values for healthy older adults are 5,000 to

6,000 per day.[108,109] Walking function has predictive value. The ability to walk 400 meters and the time taken to do so have been found to be important predictors for mortality, cardiovascular disease, and mobility disability in a large sample of community-dwelling older adults.[110]

Walking is known to be important for bone health. For example, poorer walking endurance, as measured by a 6-minute walk test, or lower ground reaction forces during walking in people with stroke, has been shown to correlate with lower paretic hip bone density, a condition that contributes to hip fracture risk.[106] Fast walking also appears to have positive benefits. The osteogenic index, which is a reflection of peak vertical GRF (or body weight), the number of loading cycles, and the number of walking sessions, is higher in fast walking than in walking at self-selected speeds, and may promote greater bone health.[111,112]

Sex

The research regarding sex differences in gait is fraught with the same difficulties as found in gait research with regard to age. Variations among methods, technologies, and subjects used in various studies make it difficult to come to many conclusions regarding the effects of sex. When differences in height, weight, and leg length between the sexes are considered, sex differences are not very great. Oberg and associates found significant differences between men and women for knee flexion/extension at slow, normal, and fast speeds at midstance and swing.[113] They found a significant increase in joint angles as gait speed increased. For example, the knee angle at midstance increased from 15° to 24° in men, and from 12° to 20° in women. However, Oberg and coworkers looked at only the knee and hip. In another study, the authors looked at

velocity, step length, and step frequency.[18] Gait speed was found to be slower in women than in men (118 to 134 cm/sec for men and 110 to 129 cm/sec for women), and step length was shorter in women than in men. Kerrigan and associates found that women had significantly greater hip flexion and less knee extension during gait initiation, a greater internal knee extension moment in terminal stance, and greater peak mechanical joint power absorption at the knee at that time (K3).[114] Kinetic data were normalized for both height and weight. These authors also found that women had a longer stride length in proportion to their height and that they walked with a greater cadence than did their male counterparts.[114]

Abnormal Gait

Both quantitative and qualitative evaluations of gait are useful assessors of human function. The most important quantitative variable is gait speed, which has been shown to be related to all levels of disablement.[115] An individual's gait pattern may reflect not only physical or psychological status but also any defects or injuries in the joints or muscles of the lower extremities. In assessing an abnormal gait, it is helpful to separate the cause or causes of deviations into structural impairment(s) or functional impairment(s). Functional impairments can be further categorized by general cause: whether directly because of abnormalities of the muscular or nervous system or their control or by pain. Although an extensive review is beyond the scope of this book, commonly encountered examples will provide a framework for examining abnormal gait.

Structural Impairment

These are structural malformations that are congenital, caused by injury or by structural changes occurring secondary to injury.

A common structural abnormality is leg length discrepancy. Kaufman and coworkers undertook a study to determine the magnitude of limb length inequality that would result in gait abnormalities.[116] Many minor limb inequalities are found in the general population but many of these do not necessitate any particular treatment or intervention because they do not have any significant effects on gait. The authors concluded that a limb length discrepancy of 2.0 cm resulted in an asymmetrical gait and had the potential for causing changes in articular cartilage. Song and colleagues evaluated neurologically normal children who had limb length discrepancies of 0.8% to 15.8% of the length of the long extremity (0.6 to 11.1 cm).[117] The compensatory strategies observed were equinus position of the ankle and foot of the short limb (toe walking), vaulting over the long limb (similar to a limp), increased flexion of the long limb, and circumduction of the long limb. Children who used toe walking had a greater vertical translation of the body's center of mass during gait than did normal controls.

Structural problems may be implicated in running injuries. Increases in the Q-angle, tibial torsion, and pronation of the foot may contribute to patellofemoral syndromes. In running, stresses are greater than in walking, and so there is an accompanying increase in the likelihood of injury. In a survey of the records of 1,650 running patients between the years 1978 and 1980, 1,819 injuries were identified.[118] The knee was the site most commonly injured in running, and patellofemoral pain was the most common complaint.

At the foot, pes cavus and pes planus can cause alterations in pressure distributions at the foot-floor interface and may cause abnormal stresses at the hip or knee. In pes cavus, the weight is borne primarily on the hindfoot and metatarsal regions, and the midfoot provides only minimal support. In running, the metatarsals bear a disproportionate share of the weight. In pes planus, the weight is borne primarily by the midfoot rather than being distributed among the hindfoot, lateral midfoot, metatarsals, and toes, as it is in the normal walking foot. The propulsive phase of gait can be severely compromised.

Functional Impairment

This group includes all causes in which the timing or amplitude of muscle activity is abnormal, whether because of abnormalities of the muscular or nervous system or their control, pain, or compensations/adaptations to the abnormalities or pain.

Abnormalities in the central nervous system are responsible for conditions such as Parkinson's disease, stroke, and cerebral palsy. These all produce characteristic gaits that are easily recognized by a trained observer. The Parkinsonian gait is characterized by an increased cadence, shortened stride, lack of heel strike and toe-off, and diminished arm swinging. The muscle rigidity that characterizes this disease interferes with normal reciprocal patterns of movement.[119] A stroke produces gait abnormalities that arise from reductions of strength and power of muscles of one lower limb that are usually worse distally. These deficits may be compounded by deficits in flexibility and motor control. Children with cerebral palsy sustain brain lesions that are similar to lesions in adults who have had strokes; however, the children sometimes have both lower limbs affected, and the immature system produces shortened muscles that do

not keep up with bone growth. Conversely, children with cerebral palsy often show compensations that people with stroke do not use.

When the plantarflexors, which are the major source of mechanical energy generation in gait, are unable to generate sufficient power, muscles at other joints may compensate and provide even more energy than is typical of normal gait.[4] For example, Winter found that individuals with below-the-knee amputations and below-the-knee prostheses used the gluteus maximus, semitendinosus, and knee extensor muscles as energy generators to compensate for loss of the plantarflexors.[120] Olney and associates found that in children with cerebral palsy who had unilateral plantarflexor weakness, the involved plantarflexors produced only 33% of the energy generation, in comparison with the 66% produced in normal gait.[121]

It is common for people with patellar pain to inhibit quadriceps contraction and produce a gait that appears kinematically normal. The quadriceps function is easily compensated for if a person has normal hip extensors and plantarflexors. The gluteus maximus and soleus muscles pull the femur and tibia, respectively, posteriorly, which results in knee extension. Additional compensation, if the hip extensor and ankle plantarflexor activity is inadequate, may be accomplished by forward trunk bending after initial contact. The forward shifting of the weight moves the GRFV anterior to the knee (at initial contact and during the loading response period). It also may force the knee into hyperextension and eliminate the need for any quadriceps activity. This tendency is often seen in the early weeks after stroke, if not corrected with gait training.

 Expanded Concepts 14–7

Paresis of Dorsiflexors

The normal functions of the dorsiflexors in gait are (1) to maintain the ankle in neutral so that the heel strikes the floor at initial contact; (2) to control the external plantarflexion moment at heel strike; (3) to dorsiflex the foot in initial swing; and (4) to maintain the ankle in dorsiflexion during midswing and late swing. If these functions are absent, the following would be expected to occur: (1) The entire foot or the toes would strike the floor at initial contact; and (2) the amount of flexion at the hip and knee would have to increase to clear the foot in swing phase. An orthosis that supports the foot, called an *ankle-foot orthosis (AFO),* is frequently used to avoid these problems. An orthosis that incorporates an electrical stimulation at the appropriate time, called *functional electrical stimulation (FES),* or *functional neuromuscular stimulation (FNS),* has also been shown to be effective.[122] Additionally, when examined in isolation, a recent study found that FES was comparable to a passive AFO in functional outcomes such as gait speed or endurance.[123]

Pain

This group includes all causes of variations that are attributable primarily to pain. All overuse injuries and most joint pathologies fall into this category. It is common for people with patellar pain or knee osteoarthritis to inhibit quadriceps contraction and produce a gait that appears kinematically normal. The quadriceps function is easily compensated for if a person has normal hip extensors and plantarflexors, a situation that is described above with reference to stroke.

Many gait variations can be seen in osteoarthritis.[124] For example, hip joint pain causes an individual to reduce the level of contraction of the hip abductor, the gluteus medius muscle, during single limb stance. This results in a typical pattern called Trendelenburg gait. Normally, the gluteus medius stabilizes the hip and pelvis by controlling the drop of the pelvis during single-limb support. If gluteus medius activity on the side of the stance leg is reduced, the pelvis, accompanied by the trunk, will tend to fall excessively

toward the swing side, which may result in a loss of balance. To prevent the trunk and pelvis from falling toward the unsupported side, the individual may laterally bend the trunk over the stance leg at initial contact, during weight acceptance, and through single stance. This trunk motion enables the person to bring the center of mass closer to the hip joint, thus reducing the need for such a large hip abductor contraction and the pain it would have caused (see Chapter 10).[125]

Knee joint pain is the principal clinical problem in 20% of people over 55 years of age with osteoarthritis of the knee, and one quarter of these are severely disabled, with extensive gait limitations.[126] As knee varus (*bow-leggedness*) is commonly associated with medial compartment knee osteoarthritis, the internal knee abductor moment (often reported in the literature as the external adductor moment, or simply *adductor moment*) is also high, and is associated with abnormally large compressive forces across the medial compartment of the knee. Efforts to limit knee forces in the frontal plane include gait modification (see explanation below), weight loss, use of a cane, "unloader" braces, and use of shoe orthotics that change the orientation of the GRFV.

Pain also appears to be a factor leading to an increase in oxygen consumption. As pain increases, oxygen consumption has been found to increase.[127]



Expanded Concepts 14–8

Contralateral Compensations

Movement compensations don't always occur on the limb that exhibits the initial problem (e.g., pain, weakness, inflexibility). Instead, it is possible to see movement compensations on the contralateral limb. For example, one might see excessive plantarflexion at the ankle during the terminal stance phase in an unimpaired limb. The primary cause could be in the other limb: for example, an inability to clear the

toes in swing phase as a result of inadequate knee flexion. The excessive plantarflexion by the stance limb would be an adaptation.

 Expanded Concepts 14–9

Accommodating a Knee Flexion Contracture During Swing

A somewhat different example of adaptation could result from a knee flexion contracture. When the affected extremity is weightbearing, the unaffected extremity will have difficulty swinging through in a normal manner, as the body's center of mass has been lowered. A method of "equalizing" leg lengths is necessary for the swing leg to swing through without hitting the floor. Extra plantarflexion during push-off of the affected side, described previously, would be one means. In this case, there is no structural or functional abnormality in the adaptive movement. Alternatively, the person could increase the amount of flexion at the hip, knee, and ankle of the swinging unaffected side. Again, the limb showing the adaptation has no structural or functional impairment. Other methods that produce relative shortening of the swinging leg are hip hiking, or circumduction of the leg. Each of these compensations makes it possible to walk, although they increase the energy requirements above normal levels.

Treatment of Gait Disorders

Of course, the accurate assessment and analysis of gait is imperative when understanding normal and abnormal function. However, just as important is the knowledge of how to treat gait disorders in patients that present to a clinic. Fortunately, a number of strategies exist that aim to provide some form of external support to account for neuromuscular or structural impairments, or to modify a patient's gait pattern to optimize function. The sections below summarize some common strategies of the treatment of gait, but are not an exhaustive list.

Assistive Devices

Walking without the use of assistive devices (crutches, canes, and walkers) is preferred by most people. However, such devices often are necessary either after a lower-extremity fracture, when the healing bone is unable to bear full body weight, or as a more permanent adjunct for balance, to compensate for muscle deficiencies, or for joint pain. Recall from Chapter 10 that a very small force at the hand can produce a large balancing moment about the standing leg because of the long perpendicular distance from the point of application of the force on the hand to the hip joint center. You may wish to review the sections in Chapter 10 on the use of a cane and the accompanying figures at this time. Canes have typically been used on the side contralateral to an affected lower extremity to reduce forces acting at the affected hip, the reason being that a lower abductor muscle force on the affected side would be necessary to balance the weight of the upper body during single-limb stance if an upwardly directed force was provided by the hand on a cane at some distance from the hip joint center. Krebs and coworkers tested the effect of cane use on reduction of pressure through the use of an instrumented femoral head prosthesis that quantified contact pressures at the acetabular cartilage.[128] The prosthetic head contained 13 very sensitive pressure-sensing transducers. The magnitude of acetabular contact pressure was reduced by 28% on one transducer and by 40% on another transducer in cane-assisted gait in comparison with unaided gait. The reduction in pressure at the hip coincided with reductions in EMG amplitude in comparison with the same pace in unaided gait trials. The authors concluded that the use of a cane on the contralateral side allows the person to increase the base of support and to decrease muscle and ground reaction forces acting at the affected hip. The hip muscle abductor force was

reduced, and gluteus maximus activity was reduced approximately 45%. The use of a cane on the contralateral side has also been shown to reduce the forces passing through the knee in people with osteoarthritis.[129] Importantly, a dose response was found in that the more weight that was borne through the cane, the less force was exerted through the stance-limb knee.

Recall from our Patient Application that Ms. Brown used a four-point cane on her unaffected side, because her arm was also affected. For her, the most important objective was to gain balance and stability, not to reduce joint forces. Another advantage of using a cane in people with a stroke is that it provides an additional point of support with the ground, which improves stability.

Orthoses

The function of orthoses (for example, knee braces, ankle-foot orthoses, or shoe-worn orthotics) in gait is to alter the mechanics of walking. They are used to support normal alignment, to prevent unwanted motion, to help prevent deformity, to reduce unwanted forces or moments, and, more recently, to augment joint power.[130–133] Although a full discussion is beyond the scope of this book, the student can deduce the effects of various types of devices with knowledge of the mechanics of gait. In all cases, the wish is to reduce or prevent unwanted movement or undesirable forces while permitting as normal mechanics as possible. For example, one may wish to limit ankle plantarflexion in a child with cerebral palsy if there is excessive plantarflexion caused by muscle spasticity. However, in so doing, one would prefer to encourage the active energy-generating activity of the ankle plantarflexors (A2) during push-off. Although some orthoses are rigid and do not permit any ankle motion, many designs are hinged

to prevent excessive plantarflexion by use of a posterior stop, but permit the ankle to move into dorsiflexion in terminal stance. Therefore, a child with cerebral palsy is able to generate some power through the plantarflexion that follows. There is some evidence of decreases in energy costs with use of an orthosis composed of elastic straps that assist the hip, knee, and ankle movements in persons with stroke.[134] Modern carbon-fiber-spring-ankle-foot orthoses and a carbon-fiber knee-ankle-foot orthosis optimize potential energy stored in the orthosis and can improve gait as well as performance in sporting events.

With respect to amputees, there have been substantial advances in the technology of the design and manufacture of artificial limbs. Though beyond the scope of this book, prosthetic limbs have changed from simple hinge-like representations that were heavy and relatively immobile, to tri-axial, multi-joint, electronically controlled devices made of lightweight carbon fiber and titanium. New approaches are even starting to integrate with the skeletal system using osseointegration and neuromuscular system through neural control of prostheses. In fact, amputees wearing prosthetics exhibit relatively normal gait patterns that are indiscernible to the naked eye.



Patient Application 14–8

Recall that Ms. Brown was prescribed an ankle-foot orthosis to assist with providing an adequate support moment during stance phase and to prevent foot drop and the resulting energy-costly hip hiking. However, if the orthosis was rigid, it would make it impossible for her to achieve any A2-S push-off during terminal stance. Two options were considered: (1) a hinged orthosis that permitted unlimited dorsiflexion but had a stop beyond 10° of plantarflexion, thus allowing limited push-off; and (2) a flexible ankle-foot orthosis that narrowed posterior to the ankle, thus permitting a limited range of both dorsiflexion and plantarflexion. Because it was hoped that use of the device would be temporary, the latter was chosen. Gait reeducation included

progressive training without the orthosis as strength was gained, and at discharge, Ms. Brown used her orthosis only for outdoor use. As her stability improved, she was able to progress from a four-point cane to a straight cane. When she was discharged from outpatient treatment, she was walking at 0.55 m/sec.

Gait Retraining

Although assistive devices and orthoses have been shown to be effective in improving gait biomechanics in a number of pathological populations, their intent is to accommodate gait deficiencies (for example, by providing additional structural support or realigning lower limb anatomy) rather than change the inherent gait characteristics of the individual. Instead, methods of retraining one's gait aim to directly change one or more deficient aspects of gait to optimize performance. Remarkably, many people with pain or functional impairment already exhibit gait compensations that result in improvements in function, without the assistance of a healthcare professional. Most of these compensations that are made are performed unconsciously, and if the disturbance is slight, such as excessive pronation of the foot, the individual may not be aware that the gait pattern is in any way unusual. However, most compensations will result in an increase in energy expenditure and may result in excessive stress on other structures of the body.[135]

The most common goal in gait retraining is to attempt to normalize gait. For example, a reduction in step length is common in a number of pathologies including stroke, Parkinson's disease, and many musculoskeletal injuries such as anterior cruciate ligament rupture. This has an impact on gait speed as well as overall gait symmetry. Accordingly, attempts to increase step length via verbal or visual (for example, instructing the patient to step over lines on the floor) cues represents an important

component of overall treatment for these patients. In some instances, external assistance is needed to enable the muscles or joints to achieve these changes. For example, with some neurological conditions like stroke, clinicians use EMG biofeedback systems to inform patients of the activation of one or more muscles through auditory or visual feedback during walking. There is evidence that EMG biofeedback training can improve walking in stroke patients.[136]

In patients with bilateral lower extremity paresis or paralysis, such as that caused by a spinal cord injury, walking usually involves the use of long leg braces and crutches. In this form of gait, the trunk and upper extremity muscles must perform all of the work of walking, and the energy cost of walking is much greater than normal. As a result, functional electrical stimulation has been suggested to activate the paralyzed lower extremity muscles so that these muscles can generate energy for walking and produce "near normal" gait. However, the energy cost of functional electrical stimulation–induced walking is still well above that of normal gait.[137] A new option is a powered robotic exoskeleton device which is a wearable technology that straps to the legs and has electrically actuated motors that control joint motion to enable overground walking in people with weakness or paralysis who would not normally be ambulatory.[138] Powered exoskeletons have been used as a rehabilitation training tool, but home versions have now been developed to use as an assistive device for people with thoracic spinal cord injuries. It is clear that great advances have been made in the technological applications to gait retraining over the past decade, and methods to decrease costs and increase accessibility are currently being explored.

When abnormal forces are present that increase the risk of injury it is often necessary to

change one or more gait parameters to attempt to reduce these forces. In these instances, it is possible that an individual is retrained to make a rather abnormal kinematic or kinetic parameter visually normal. For example, it is suggested that high vertical loading rates play an important role in the development of running injuries, and attempts have been made to instruct the person to “run lighter” or to modify aspects of their running gait such as changing footstrike pattern, increasing or decreasing stride length, or increasing knee flexion at initial contact.[139–141] Numerous studies (for example, Crowell and Davis) using real-time biofeedback of running performance have been conducted and have shown that discrete measures of running gait can be effectively modified with training.[142]

Gait modification for knee osteoarthritis is a growing area in the literature. Similar to retraining runners, the goal of gait modification for knee osteoarthritis is to change the loading of the knee joint – specifically the internal knee abductor moment – by producing otherwise abnormal changes in kinematic parameters. Changes to frontal plane measures such as ipsilateral trunk lean and rotation of the feet (toeing-in or toeing-out) have received much interest due to their ability to reduce the abductor moment.[143,144] Increases in trunk lean or toe-out in excess of 100% over normal values have been reported and dose-responses have been found; that is, greater changes produce greater reductions in knee load.[125] Recent clinical trials investigating long-term effects of gait modification in this population have also shown promising results on clinical outcomes, such as pain and function.[44, 145] State-of-the art real-time biofeedback of performance is commonly used in the laboratory setting to provide instantaneous visual cues of the modified parameter of interest, whereas simpler and less expensive methods such as a standard wall mirror may be sufficient in the clinical

setting.[146,147]

Summary

- As a result of the efforts of many investigators, our present body of knowledge regarding human locomotion is extensive. However, gait is a complex subject, and further research is necessary to standardize methods of measuring and defining kinematic and kinetic variables, to develop inexpensive and reliable methods of analyzing gait in the clinical setting, and to augment the limited amount of knowledge available regarding kinematic and kinetic variables in the gaits of children and elderly persons.

- Standardization of equipment and methods used to quantify gait variables, as well as standardization of the terms used to describe these variables, would help eliminate some of the present confusion in the literature and make it possible to compare the findings of various researchers with some degree of accuracy. Some standardization has been provided by the manufacturers of motion analysis systems; as a result, investigators using similar systems tend to use the same conventions.

- Recent advances in mobile technology have permitted the easy assessment (albeit not as accurate as lab-based systems) of gait outside the laboratory setting and in real-world situations. Wearable sensor technology has also improved greatly in recent years and systems incorporating gyroscopes and accelerometers interfaced with online algorithm processing enables accurate assessment of gait with real-time biofeedback capabilities. Additionally, freely available software permits the

quick estimation of simple parameters such as stride length, speed, and two-dimensional kinematics. These measures provide a simple means for objective assessment of a patient's status and for detecting overall change. Increases in step length and decreases in step duration may be used to document a patient's progression toward a more normal gait pattern; however, a normal gait pattern may not be appropriate for many patients. Although many movement practitioners feel that it is best to aim for normal gait during early stages of rehabilitation, it is also important to identify the means by which a person can use the flexibility of the human body to compensate for deficiencies.[5, 50] Automated gait analysis computer programs can provide the clinician with information about all of the kinematic and kinetic gait parameters related to a particular subject or patient. However, the researcher or clinician must have sufficient knowledge of the kinematics and kinetics of normal gait to interpret and use the information for the benefit of the patient.

- The objectives of gait analysis are to identify deviations from the norm and their causes. Once the cause has been determined, it is possible to take corrective action aimed at improving performance, eliminating or diminishing abnormal stresses, and decreasing energy expenditure. Sometimes the corrective action may be as simple as using a lift in the shoe to equalize leg lengths or developing an exercise program to increase strength or flexibility at the hip, knee, or ankle. In other instances, corrective action may require the use of assistive devices such as braces, canes, or crutches. However, an understanding of the complexities of abnormal gait and the ability to detect abnormal gait patterns and to determine the causes of these deviations must be based on an understanding of normal structure and

function. The study of human gait, like the study of human posture, illustrates the interdependence of structure and function and the large variety of postures and gaits available to the human species.

Study Questions

1. What percentage of the gait cycle is occupied by the stance phase in normal walking? How does an increase in walking speed affect the percentage of time spent in stance?

2. What percentage of the gait cycle is spent in double support? How is double support affected by increases and decreases in the walking speed?

3. Describe the subdivisions of the stance and swing phases of the walking gait cycle.

4. During which period of the gait cycle does maximum knee flexion occur?

5. What are the approximate values of maximum flexion and extension required for normal gait at the knee, hip, and ankle?

6. How does the total range of motion required for normal gait at the knee, hip, and ankle compare with the ROM required for running and stair gaits?

7. What is the difference between an internal moment and an external moment?

8. What is the concept of the support moment, and what major muscle groups are responsible?

9. What moments are acting at the ankle, knee, and hip at initial contact? Answer the same question with regard to different gait events: foot flat, midstance, heel-off, and toe-off.

10. What is largely responsible for the internal abductor moment at the knee in stance phase?

11. What are the roles of the hamstrings in normal gait? Do they contribute to support or to power?

12. What are the major muscle groups that contribute to the positive work of walking, and when in the gait cycle do their contributions occur?

13. Why does walking faster than normal and walking slower than normal usually result in increased energy costs?

14. Why do long double-support times usually result in increased energy costs?

15. What is the role of the quadriceps muscle during walking gait?

16. What is the function of the plantarflexors during walking gait?

17. What are the functions of the dorsiflexors in normal walking gait?

18. How is the swinging motion of the upper extremities related to movements of the trunk, pelvis, and lower extremities during walking gait?

19. Compare muscle action in walking gait with muscle action in running gait.

20. Explain what would happen in walking and running if a person's plantarflexors were weak. What compensations might you expect?

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