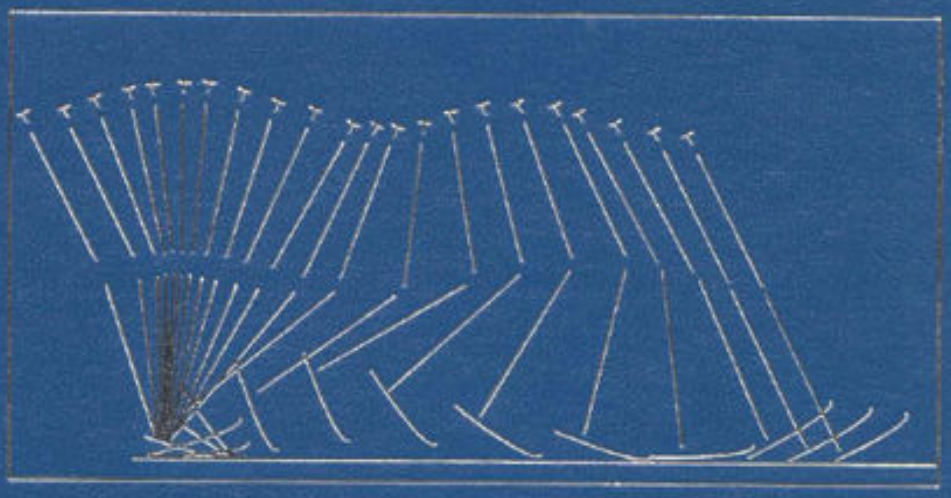
GAIT ANALYSIS

Normal and Pathological Function

SECOND EDITION



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SLACK Incorporated

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Normal and Pathological Function

SECOND EDITION

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Contents

Acknowledgments

The development of a systematic means of observational gait analysis was a collaborative effort with physical therapy supervisors and instructors from Rancho Los Amigos National Rehabilitation Center (Rancho). This group's membership has changed so frequently during the quarter century of the program's evolution that we can only broadcast an undesignated "thanks for your help" and hope each deserving person absorbs our thoughts. We particularly wish to acknowledge the help of Jacqueline Montgomery and Maureen Rodgers for their long-term involvement in the gait project.

For extensive assistance in the final preparation of this book, very special thanks go to JoAnne K. Gronley and Lydia M. Cabico. JoAnne, a physical therapist and Associate Director of Clinical Research in Rancho's Pathokinesiology Laboratory, has diligently monitored the quality of the laboratory data for the past 30 years. Lydia Cabico, of Berkley Graphics and Design and of Rancho, is a superb computer design artist who created the majority of the artwork used in the second edition of this book.

About the Authors

Jacquelin Perry, MD's interest in gait began in college (UCLA). A major in physical education (1935-1940) introduced her to anatomy and provided a strong background in kinesiology with application to sports. Part of this experience was her attendance at the Physical Therapy Clinic of the Los Angeles Children's Hospital where she began her exposure to disability. Subsequently, she became a physical therapist (Walter Reed Army Hospital, 1941), which expanded her knowledge of anatomy, kinesiology, and disability.

Her physical therapy experience in Army hospitals during World War II provided a broad clinical experience (1941-1945). In addition to working with a regular flow of trauma patients, she spent 2 years at a center that had Army programs for poliomyelitis and rheumatoid arthritis. All 3 clinical areas involved a great deal of informal observational gait analysis as one sought to improve the patient's ability to walk. During most of this time, she was also an instructor at two of the Army schools of physical therapy (Hot Springs, AR and Denver, CO). There she taught anatomy, kinesiology, and therapeutic exercise as well as the modalities. Both normal and disabled gait were strong elements of this program.

After the war ended, she used her GI bill to go to medical school (UC San Francisco, 1946-1950) for the specific purpose of becoming an orthopaedic surgeon. Dr. Perry's residency in orthopaedic surgery (UCSF, 1951-1955) occurred during the period when poliomyelitis and reconstructive surgery were strong clinical programs. Observational gait analysis and experience in correcting disabled gait became daily practice.

Her next move was to join the staff of the Rancho Los Amigos National Rehabilitation Center. In 1955, poliomyelitis was the entire focus of the rehabilitation program. Disability of lower limbs, spine, and arms were all major concerns while bracing and reconstructive surgery received equal emphasis. Working with this program further expanded her knowledge of muscle function and gait disability. In

addition, her experience in observing polio survivors exposed her to a number of different gait patterns as the type of paralysis resulting from this disease varies from patient to patient.

Following introduction of the Salk vaccine, polio was conquered so Dr. Perry and her colleagues redirected their attention to other types of chronic impairments. This change was the beginning of their intensive rehabilitation program for spinal cord injury, hemiplegia, arthritis, and children's disorders (primarily muscular dystrophy, myelodysplasia and cerebral palsy). Later, amputee and problem back services were added. At first, the program was for general rehabilitation. Then as the patient groups became large, they formed separate clinical categories with a ward for each (1961). While continuing the polio spine surgery program, Dr. Perry also developed a stroke unit.

Responsibility for persons disabled by a stroke forced her to expand her analysis process, as the functional pathology of individuals with hemiplegia is much more complex than that of polio. Because the standard clinical examination findings correlated poorly with the gait dysfunctions, a system of observational gait analysis was initiated. Developed in conjunction with a group of knowledgeable and dedicated physical therapists, the Rancho Los Amigos Observational Gait Analysis System became highly organized. For the first time, there was a means of cataloging the multiple dysfunctions that occur with the various types of pathology. For the past 25 plus years, they have taught this program nationwide. The organizational background of this book is based on this program.

A second development was the gait laboratory (1968). Its initial purpose was to document the improvement from reconstructive surgery in patients who could not be returned to normal function based on traditional rehabilitation therapy. This system was designed to help ascertain whether surgery actually was the better alternative for these patients. Out of this beginning was developed a functional diagnostic system to be used for planning the reconstructive surgery of patients with spasticity. The emphasis of the program was, and still is, kinesiological electromyography because the primary disability of patients with spasticity is inappropriate muscle action

(errors in timing and intensity). Footswitches were developed to define the patient's stride characteristics, and an electrogoniometer to record joint motion during gait also was developed. Clinical service and research have had equal emphasis from the beginning. Another novel emphasis has been on energy cost analysis of walking. An outdoor court was designed where habitual gait could be studied (Dr. Robert Waters spearheaded this). Today, the Pathokinesiology Laboratory at Rancho Los Amigos National Rehabilitation Center is fully equipped with automated motion analysis (CODA™), force plates and walking aids instrumented with force transducers. All types of disability have been studied over the years and continue to be seen as the clinical need increases (cerebral palsy, hemiplegia, spinal cord injury, post polio, arthritis, joint replacement, amputees, myelodysplasia, and muscular dystrophy).

During her career, Dr. Perry has received numerous awards for her pioneering work in many areas of gait and orthopaedics. She received the Kappa Delta Award (Orthopedic Research Society, 1976) for landmark work with dynamic electromyography to define muscle function in cerebral palsy and the Isabelle and Leonard H. Goldson Award in Technology (United Cerebral Palsy Research and Education Foundation, 1981). She was a Shands Lecturer (American Orthopaedic Association, 1988) and received the Shands Award (Orthopaedic Research Society, 1999). Dr. Perry received Lifetime Achievement Awards from the Gait and Clinical Movement Analysis Society (2000) and The Scoliosis Research Society (2008). In December of 2008, the University of Southern California dedicated the Jacquelin Perry Musculoskeletal Biomechanics Laboratory in her honor. Dr. Perry continues her lifelong dedication to the research and clinical application of gait. This publication encompasses the extensive work of Dr. Perry and her successful years as a therapist and a surgeon renowned for her expertise in human gait.

Judith M. Burnfield, PhD, PT is a most welcome co-author. She has excellent analytical skills and has an outstanding command of words. In addition, her scientific preparation and professional experience give her a unique perspective of impaired gait.

Dr. Burnfield's interest in gait began during a clinical internship in 1986 on the Arthritis Service at Rancho Los Amigos National Rehabilitation Center (Rancho). The analytic process encouraged during weekly gait sessions (many led by Dr. Perry) tapped into her investigative nature. Following graduation from the Physical Therapy Program at the State University of New York at Buffalo (1986), Dr. Burnfield accepted a position at Rancho Los Amigos National Rehabilitation Center. Her initial work on the Ortho-Diabetes Service further sparked her passion for understanding gait. Differentiating the influence of human impairments from prosthetic alignment factors was essential for improving patient's walking ability and helped her develop a greater appreciation for the role alignment, forces, and moments have on walking performance. During this period, Dr. Burnfield started teaching a course on prosthetics and orthotics in the Physical Therapist Assistant Program at Cerritos College (Norwalk, CA). Gait was a central component of the course.

Subsequent work on the Stroke Service, Gerontology Service, and in the Polio Clinic at Rancho expanded her knowledge related to the profound impact of weakness on movement disorders. It also highlighted the body's exquisite versatility to compensate when sensation and control mechanisms remained intact (eg, with polio). Dr. Burnfield developed a strong appreciation for the role of lower extremity orthoses in promoting independence, particularly when the device's capabilities were systematically matched to each patient's unique impairments. During this period, Dr. Burnfield engaged in formal training to become a Rancho Gait Instructor. She then led gait sessions at Rancho and started teaching workshops around the country for therapists and physicians.

In 1996, Dr. Burnfield left her position as Director of Physical Therapy at Rancho to commence graduate studies in Biokinesiology at the University of Southern California (USC). Her doctoral research, under the advisement of Dr. Christopher Powers, focused on human and environmental factors contributing to slips and falls during walking. She co-developed and taught the introductory and then advanced observational gait analysis courses for students enrolled in the clinical doctorate of physical therapy program at USC. At the time of her enrollment in USC's Biokinesiology program, she

also pursued a position as a research physical therapist working in the Pathokinesiology Laboratory at Rancho due to her desire to enhance her clinical research skills related to normal and pathologic gait. Her work in the latter environment focused on clinical and research studies quantifying gait abnormalities through the analysis of kinematic (motion), kinetic (moment) and electromyographic (muscle activity) data. The unique research and teaching experiences in the two environments complimented each other well and provided a framework for her role instructing Biomechanics and subsequently Kinesiology in the Physical Therapy Program at Mount St. Mary's College in Los Angeles.

Following completion of her doctoral studies, Dr. Burnfield engaged in a postdoctoral fellowship with Dr. Jacquelin Perry. This mentorship had a profound influence on Dr. Burnfield's professional development for which she is most grateful. This focused period of inquiry provided a strong foundation for their subsequent collaboration on the second edition of this book.

In October 2004, Dr. Burnfield joined Madonna Rehabilitation Hospital in Lincoln, Nebraska. She serves as Director of the Institute for Rehabilitation Science and Engineering, Director of the Movement and Neurosciences Center and the Clifton Chair in Physical Therapy and Movement Sciences. Dr. Burnfield has developed a dynamic research laboratory that capitalizes on the close proximity of patients and clinicians, a strong network of research collaborators internally as well as from universities and industry, and a diverse group of undergraduate and graduate students willing and eager to learn and contribute. Research efforts focus on developing and studying new treatments and technologies to help individuals with physical disabilities walk, exercise, and live more independently. The fully instrumented Chapin Gait and Motion Laboratory within the Movement and Neurosciences Center includes state-of-the art technology and software for conducting biomechanical and physiological analyses of movement function including a twelve-camera infrared motion analysis system, 16-channel and 10-channel portable electromyography technology, four force platforms, a footswitch system and plantar pressure mapping

technology, and a 30-meter walkway with an overhead safety support track and full-body harness system.

Dr. Burnfield holds adjunct faculty appointments at Creighton University, University of Nebraska– Lincoln, University of Nebraska Medical Center, and the University of Southern California. In addition to a vigorous research agenda, Dr. Burnfield continues to teach gait in the academic setting and presents both nationally and internationally on gait and rehabilitation related topics.

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Introduction

Walking is a convenient means for traveling short distances. In the absence of pathology, gait appears coordinated, efficient, and effortless. Disease or trauma, however, can disrupt the precision, coordination, speed, and versatility that characterize normal walking. The first edition of *Gait Analysis: Normal and Pathological Function* introduced a comprehensive and systematic means for analyzing pathological gait. The underlying framework was a solid understanding of normal gait mechanics, including joint motions, moments, and muscle activation patterns. The impact of pathology and impairments on function was systematically discussed.

Increasing knowledge related to the functional significance that can be derived from a systematic analysis of a patient's gait was the underlying stimulus to produce a second edition of *Gait Analysis: Normal and Pathological Function*. This was approached by reviewing each section to identify concepts that could be enhanced with the inclusion of recent research findings as well as identifying new topics that should be included.

The functions of 2 stance phases (initial contact and pre-swing) were clarified by new techniques that elucidated their force characteristics. Both phases previously had inconclusive descriptions that needed upgrading.

In the previous edition, the term *initial contact* was selected as a generic term to avoid designating a specific area of the foot as the contact site to initiate stance. As duration of the event was not identified in the first edition, some investigators objected to including this event in the list of gait phases. Recording the normal ground reaction forces using platforms with 100 to 200 Hz frequency responses (versus the customary 50 Hz) revealed a high intensity, sharp wave (1% to 2% of the gait cycle) superimposed on the ascending arm of the sagittal ground reaction force curve. This transient, high-amplitude wave is a clear indication that a significant

event accompanies initial contact and the early transfer of body weight onto the lead foot.

The significance of the sharp, intense burst of ankle plantar flexor power found in pre-swing just after the onset of terminal double support has been debated for decades. The engineering interpretation was a push-off force by the gastrocnemius and soleus to propel the body forward. The clinicians rejected this thought as no electromyographic (EMG) activity accompanied the rapid arc of ankle plantar flexion. A recent study clarified the debate by using a portable ultrasound sensor to visualize the muscle fascicles of the medial gastrocnemius as subjects walked. Simultaneous length measurements documented the response of the total muscle-tendon unit (MTU) and of just the muscle fascicles of the medial gastrocnemius to the ankle motion of late stance. Terminal stance dorsiflexion displayed a significant MTU length gain, no change by the muscle fibers, but intense EMG (acting isometrically). Pre-swing plantar flexion registered rapid MTU shortening and mild change in raw muscle fiber length without any EMG. The conclusion was that tendon recoil was providing the dynamic force.

The 8 phases of a gait cycle define the performance of one limb. The other limb repeats the same sequence of motions starting 50% of a gait cycle later. Comparing the reciprocal actions of the 2 limbs is a common clinical practice but there has been no model. To rectify this problem, a chapter defining the reciprocal relations of the 2 limbs has been added.

The number of clinical examples was increased to demonstrate the way joint pathology could direct the criteria for gait analysis and training. Recent orthopaedic research identified more accurately the pathology of arthritis and has provided useful guidelines for differentiating the force patterns and gait characteristics of patients disabled by the major sources of joint pain: osteoarthritis and rheumatoid arthritis. In addition, 4 other clinical areas were substantially expanded or added in that chapter.

The section on amputee gait now includes a generic classification of prosthetic components that groups the multitude of designs based on their mechanical properties in gait. The congenital clubfoot was added to introduce the significance of age, the qualities of

therapeutic stretch, and the timing of surgery. The section on stroke was expanded to include 4 case studies highlighting the varying impact of muscle weakness and selective control on gait function. Posterior tibialis tendon dysfunction, a not infrequent condition in the orthopedic community, was included to highlight the importance of early detection and management of this debilitating condition.

Two new chapters describe the mechanics of advanced patterns of gait: stair negotiation (ascent and descent) and running. Additionally, a chapter on pediatric gait has been included to describe the early maturation of walking. Thus, this volume has been enriched in several directions.

Section I

Fundamentals

Chapter 1

Gait Cycle

Walking uses a repetitious sequence of limb motions to simultaneously move the body forward while also maintaining stance stability. Because each sequence involves a series of interactions between 2 multisegmented lower limbs and the total body mass, identification of the numerous events that occur necessitates viewing gait from several different aspects. There are 3 basic approaches. Of these, the simplest system subdivides the cycle according to the variations in reciprocal floor contact by the 2 feet. A second method uses the time and distance qualities of the stride. The third approach identifies the functional significance of the events within the gait cycle (GC) and designates these intervals as the functional phases of gait.

RECIPROCAL FLOOR CONTACT PATTERNS

As the body moves forward, one limb serves as a mobile source of support while the other limb advances itself to a new support site. Then the limbs reverse their roles. For the transfer of body weight from one limb to the other, both feet are in contact with the ground. This series of events is repeated by each limb with reciprocal timing until the person's destination is reached.

A single sequence of these functions by one limb is called a GC.[3] With one action flowing smoothly into the next, there is no specific starting or ending point. Hence any event could be selected as the onset of the GC. Because the moment of floor contact is the most

readily defined event, this action generally has been selected as the start of the GC. Normal persons initiate floor contact with their heel (ie, heel strike). As not all patients have this capability, the generic term *initial contact* (IC) will be used to designate the onset of the GC.[7]

CYCLE DIVISIONS

Each GC is divided into 2 periods: stance and swing. *Stance* is the term used to designate the entire period during which the foot is on the ground. Stance begins with IC (Figure 1-1). The word *swing* applies to the time the foot is in the air for limb advancement. Swing begins as the foot is lifted from the floor (toe off).

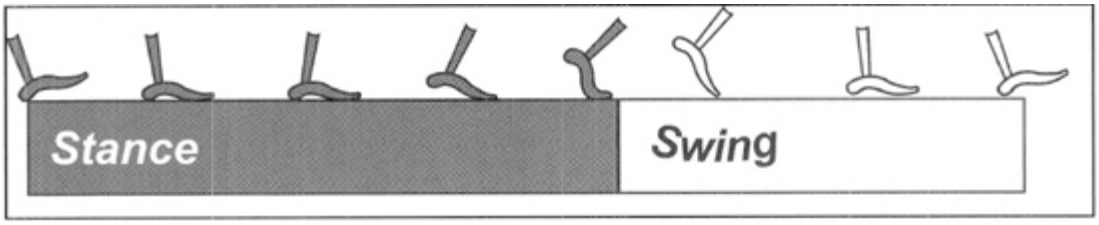

Figure 1-1. Divisions of the GC. The shaded bar represents the duration of stance. The clear bar is the duration of swing. Limb segments show the onset of stance with IC, end of stance by the roll-off of the toes, and end of swing as the instant before the foot contacts the ground.

Stance is subdivided into 3 intervals according to the sequence of floor contact by the 2 feet (Figure 1-2). Both the start and end of stance involve a period of bilateral foot contact with the floor (double stance), while the middle portion of stance has one foot contact (single limb support [SLS]).

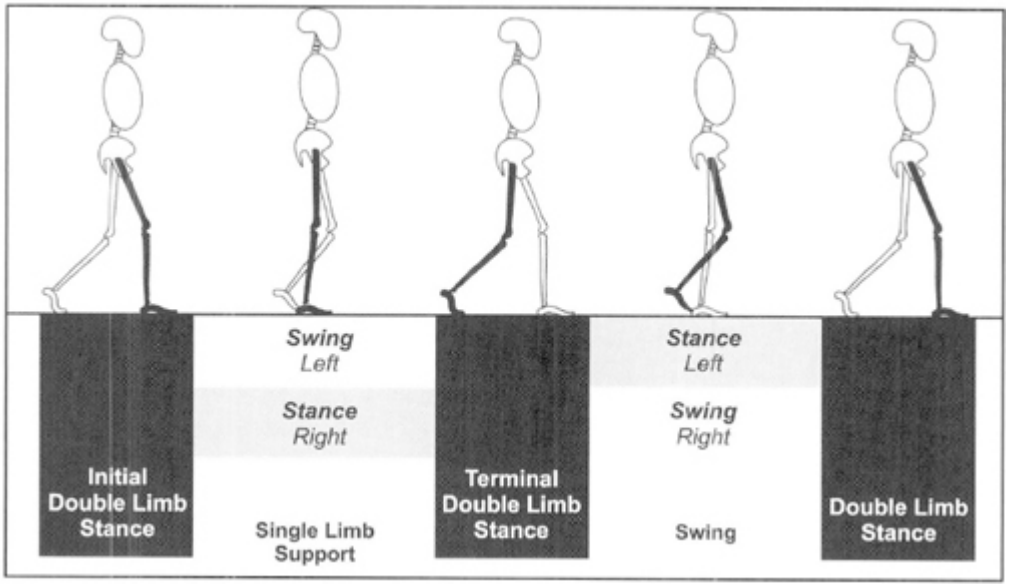

Figure 1-2. The subdivisions of stance and their relationship to the bilateral floor contact pattern. Vertical dark bars are the periods of double limb stance (right and left heel). Horizontal shaded bar is SLS (single stance). Total stance includes 3 intervals: the initial double stance, SLS, and the next (terminal) double stance. Note that right SLS is the same time interval as left swing. There is a left SLS during right swing. The third vertical bar (double stance) begins the next GC.

Initial double stance begins the GC. IC initiates this first period when both feet are on the ground. An alternate term is *double limb support*. This designation is to be avoided, however, as it implies an equal sharing of body weight by the 2 feet, which is not true during most of the double stance interval.

SLS begins when the opposite foot is lifted for swing. In keeping with the terminology for the double contact periods, this should be (and often is) called single stance. To emphasize the functional significance of floor contact by just one foot, the term *support* is preferred. The body's entire weight is resting on that one extremity during the SLS interval. The duration of single stance is the best index of the limb's support capability, with longer relative durations reflecting greater stability.

Terminal double stance is the third subdivision. It begins with floor contact by the other foot (contralateral IC) and continues until the original stance limb is lifted for swing (ipsilateral toe off). The term

terminal double limb support has been avoided as weight bearing is very asymmetrical.

Gait Cycle Timing

The generic normal distribution of the floor contact periods approximates 60% for stance and 40% for swing (Table 1-1).[3] The precise duration of these GC intervals, however, varies with the person's walking velocity.[1,6] At the customary 82 m/min (1.36 m/s) rate of walking, the stance and swing periods represent 62% and 38% of the GC, respectively, while each double stance interval is 12% of the GC (see Table 1-1).[7] The duration of both gait periods shows an inverse relationship to walking speed (ie, both total stance and swing times are shortened as gait velocity increases). The change in stance and swing times becomes progressively greater as speed slows.[1] A different relationship exits among the subdivisions of stance. Walking faster proportionally lengthens single stance and shortens the 2 double stance intervals.[4,5] The reverse is true as the person's walking speed slows. This pattern of change also is curvilinear. SLS of one limb equals swing of the other as they are occurring at the same time (see Figure 1-2).

Table 1-1

*Floor Contact Periods**

	Generic	*82 m/min*
Stance	60%	62%
Initial double stance	10%	12%
Single limb support	40%	38%
Terminal double stance	10%	12%
Swing	40%	38%

*Generic timing (expressed as % gait cycle) compared to the customary normal speed of walking, 82 m/min (1.36 m/s). (Adapted from Pathokinesiology Service and Physical Therapy Department. *Observational Gait Analysis*. 4th ed. Downey, CA: Los Amigos Research and Education Institute, Inc, Rancho Los Amigos National Rehabilitation Center; 2001.)

Having an interval when both feet are in contact with the ground for the limbs to exchange their support roles is a basic characteristic of walking. When double stance is omitted, the person has entered the running mode of locomotion.[2]

Stride and Step

The GC also has been identified by the descriptive term *stride*.[3] Occasionally, the word *step* is also used, but this is inappropriate.

Stride is the equivalent of a GC (Figure 1-3). It is based on the actions of one limb. The duration of a stride is the interval between 2 sequential initial floor contacts by the same limb (ie, right IC and the next right IC).

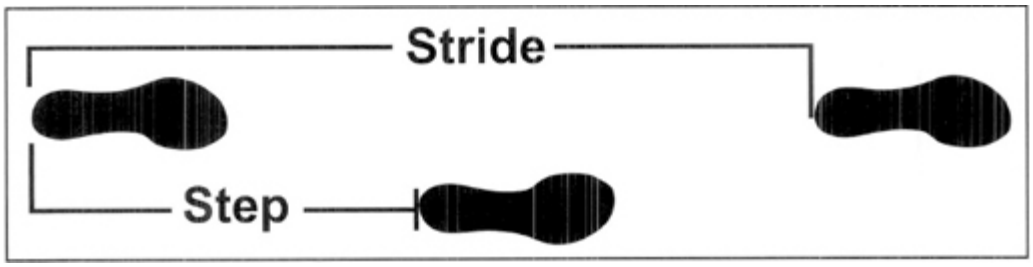

Figure 1-3. A stride versus a step. A left stride is shown (left heel contact to the next left heel contact). A right step is the interval from left heel contact to right heel contact.

Step refers to the timing between the 2 limbs (see Figure 1-3). There are 2 steps in each stride (or GC). At the midpoint of one stride, the other foot contacts the ground to begin its next stance period. The interval between an IC by each foot is a step (ie, left and then right). The same offset in timing will be repeated in reciprocal fashion throughout the walk.

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Chapter 2

Phases of Gait

In order to provide the basic functions required for walking, each stride involves an ever-changing alignment between the body and the supporting foot during stance and selective advancement of the limb segments in swing. These reactions result in a series of motion patterns being performed by the hip, knee, and ankle. Early in the development of gait analysis, the investigators recognized that each pattern of motion related to a different functional demand and designated them as the phases of gait. Further experience in correlating the data has progressively expanded the number of gait phases identified. It now is evident that each stride contains 8 functional patterns (phases).

It had been the custom in the past to use normal events as the critical actions separating the phases. While this practice proved appropriate for the amputee, it often failed to accommodate the gait deviations of patients impaired by paralysis or arthritis. For example, the onset of stance customarily has been called *heel strike*, yet the heel of persons with paralysis may never contact the ground or do so much later in the GC. Similarly initial floor contact may be by the whole foot (“foot flat”), rather than having a period of heel-only support followed by the forefoot contacting the ground. To avoid these difficulties and other areas of confusion, the Rancho Los Amigos Gait Analysis Committee developed generic terminology for the functional phases of gait.[1]

Analysis of a person’s walking pattern by phases more directly identifies the functional significance of the different motions occurring at the individual joints. The phases of gait also provide a means for correlating the simultaneous actions of the individual joints into patterns of total limb function. This is a particularly important

approach for interpreting the functional effects of disability. The relative significance of one joint's motion compared to the others varies among the gait phases. Also, a posture that is appropriate in one gait phase would signify dysfunction at another point in the stride because the functional need has changed. As a result, both timing and joint angle are very significant. This latter fact adds to the complexities of gait analysis.

Each of the 8 gait phases has a functional objective and a critical pattern of selective synergistic motion to accomplish this goal. The sequential combination of phases also enables the limb to accomplish 3 basic tasks. These are weight acceptance (WA), SLS, and swing limb advancement (SLA) (Figure 2-1).

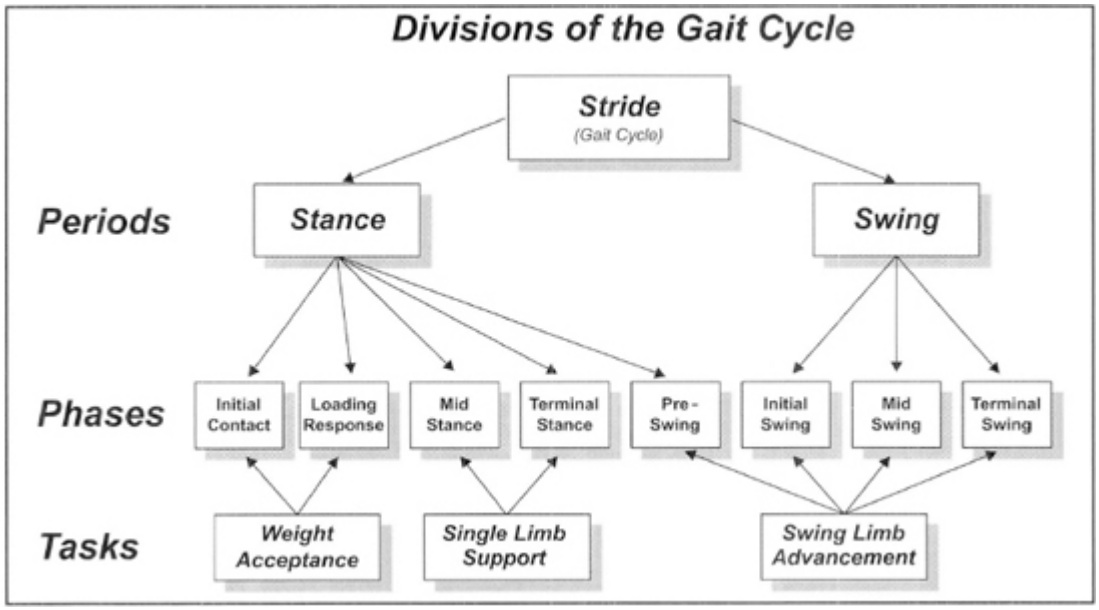

Figure 2-1. Functional division of the GC. A *stride* is the functional term for the GC. The periods show the basic division of the GC by foot contact. Each phase is determined by limb postures. The tasks show the grouping of the phases by the functions to which they contribute.

TASK: WEIGHT ACCEPTANCE

WA is the first task of stance. This is the most challenging task in the GC because 3 functional demands must be satisfied. These are

1) shock absorption, 2) initial limb stability, and 3) the preservation of progression. The challenge is the abrupt transfer of body weight onto a limb that has just finished swinging forward and has an unstable alignment. Two gait phases are involved, IC and loading response (see Figure 2-1).

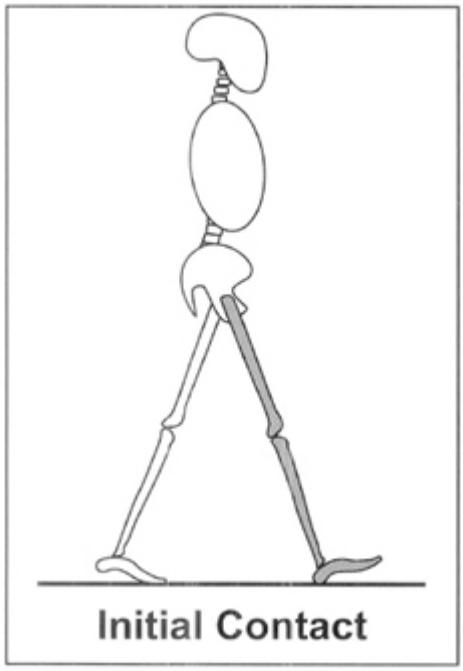

Figure 2-2. IC. The hip is flexed, the knee is extended, and the ankle is dorsiflexed to neutral. Floor contact is made with the heel. Shading indicates the reference limb. The other limb (clear) is at the beginning of pre-swing.

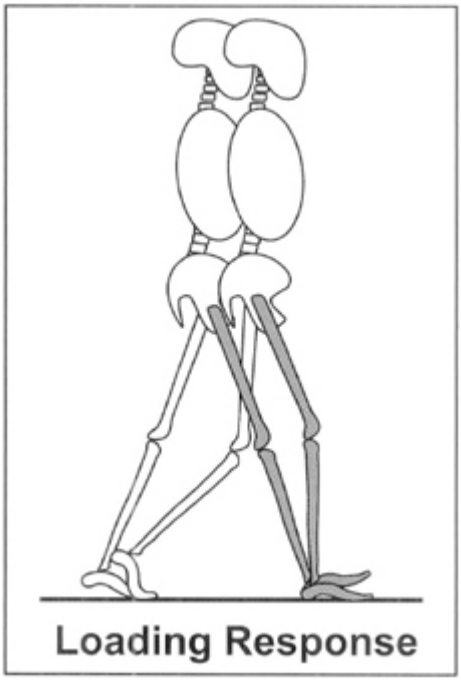

Figure 2-3. Loading response. Body weight is transferred onto the forward limb (shaded). Using the heel as a rocker, the knee is flexed for shock absorption. A brief arc of ankle plantar flexion (PF) interrupts the heel impact, but the heel rocker is preserved until the end of the phase.

PHASE 1. INITIAL CONTACT

Interval: 0% to 2% GC

This phase includes the instant the foot drops on the floor and the immediate reaction to the onset of body weight transfer. The joint postures present at this time determine the limb's loading response pattern (Figure 2-2).

Objectives:

Start stance with a heel rocker

Impact deceleration

PHASE 2. LOADING RESPONSE

Interval: 2% to 12% GC

This is the second phase contained in the initial double stance period. The phase follows the IC of the foot with the floor and continues until the other limb is lifted for swing (Figure 2-3).

Objectives:

Shock absorption

Weight-bearing stability

Preservation of progression

TASK: SINGLE LIMB SUPPORT

Lifting the other foot for swing begins the SLS interval for the stance limb. This continues until the opposite foot again contacts the floor. During the resulting interval, the one limb has the total responsibility for supporting body weight in both the sagittal and coronal planes while progression continues. Two phases are involved in SLS: mid stance and terminal stance (see Figure 2-1). They are differentiated primarily by their mechanisms of progression.

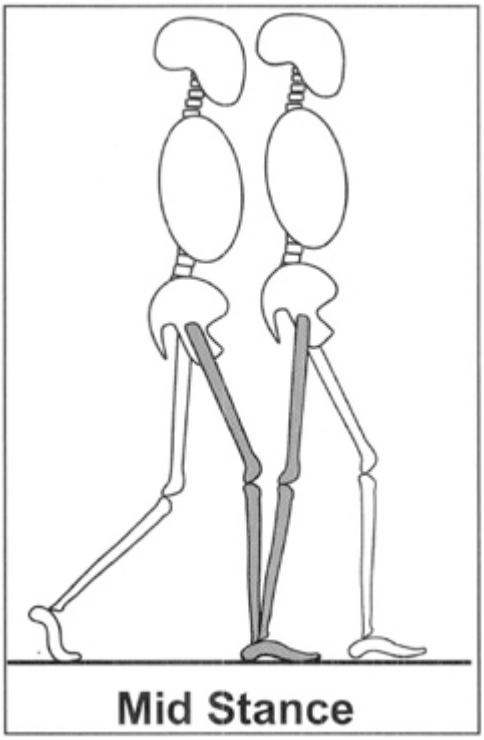

Figure 2-4. Mid stance. In the first half of SLS, the limb (shaded) advances over the stationary foot by ankle dorsiflexion (DF) (ankle rocker) while the knee and hip extend. The opposite limb (clear) is advancing through its mid swing phase.

PHASE 3. MID STANCE

Interval: 12% to 31% GC

This is the first half of the SLS interval. It begins as the other foot is lifted and continues until body weight is aligned over the forefoot (Figure 2-4).

Objectives:

Progression over the stationary foot

Limb and trunk stability

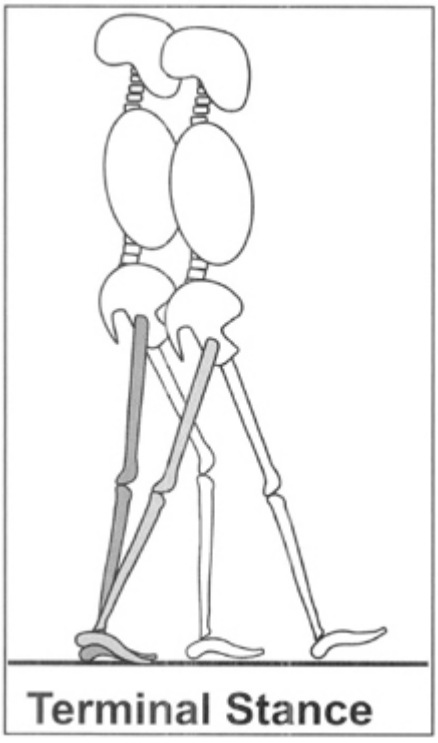

Figure 2-5. Terminal stance. During the second half of SLS, the heel rises and the limb (shaded) advances over the forefoot rocker. The knee completes its extension and then begins a new arc of flexion.

Increased hip extension and heel rise put the limb in a more trailing position. The other limb (clear) is completing terminal swing.

Phase 4. Terminal Stance

Interval: 31% to 50% GC

This phase completes SLS. It begins with heel rise and continues until the other foot strikes the ground. Body weight moves ahead of the forefoot throughout this phase (Figure 2-5).

Objectives:

Progression of the body beyond the supporting foot

Limb and trunk stability

TASK: SWING LIMB ADVANCEMENT

To meet the high demands of advancing the limb, preparatory posturing begins in stance. The limb then swings through 3 postures as it lifts itself, advances to complete the stride length, and prepares for the next stance interval. Four gait phases are involved: 1) pre-swing (end of stance), 2) initial swing, 3) mid swing, and 4) terminal swing (see Figure 2-1).

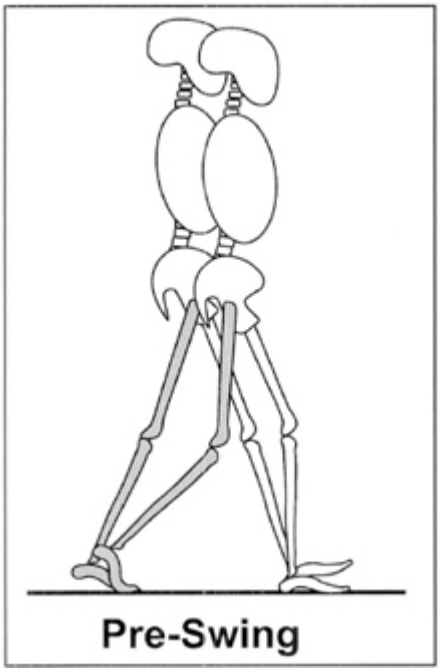

Figure 2-6. Pre-swing. Terminal double limb support is initiated by floor contact of the other limb (clear). The reference limb (shaded) responds to the initial weight transfer with increased ankle PF, knee flexion, and a reduction of hip extension. The opposite (clear) limb is in loading response.

PHASE 5. PRE-SWING

Interval: 50% to 62% GC

This final phase of stance is the second (terminal) double stance interval in the GC. It begins with IC of the opposite limb and ends with ipsilateral toe off. Weight release and weight transfer are other titles some investigators give to this phase. However, all the motions and muscle actions occurring at this time relate to progression. As the abrupt transfer of body weight rapidly unloads the limb, the trailing extremity contributes to progression with a forward “push” that also prepares the limb for the rapid demands of swing. Hence, the term *pre-swing* is representative of its functional commitment to initiating the forward motion that is used in swing (Figure 2-6).

Objectives:

Position the limb for swing

Accelerate progression

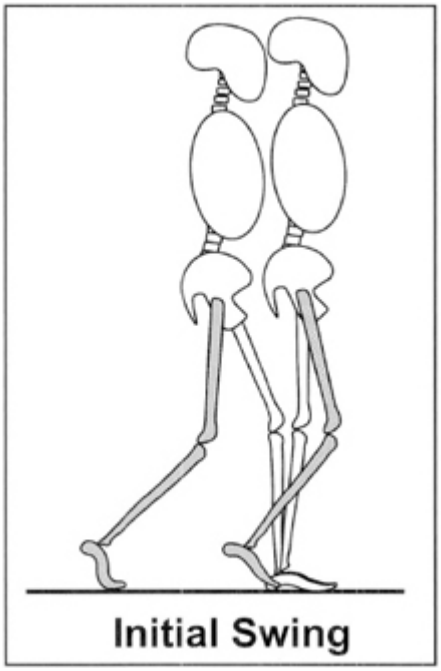

Figure 2-7. Initial swing. Increased knee flexion lifts the foot for toe clearance, and hip flexion advances the limb. Ankle DF is incomplete. The other limb (clear) is in early mid stance.

PHASE 6. INITIAL SWING

Interval: 62% to 75% GC

This first phase of swing is approximately one-third of the swing period. It begins as the foot is lifted from the floor and ends when the swinging foot is opposite the stance foot (Figure 2-7).

Objectives:

Foot clearance of the floor

Advancement of the limb from its trailing position

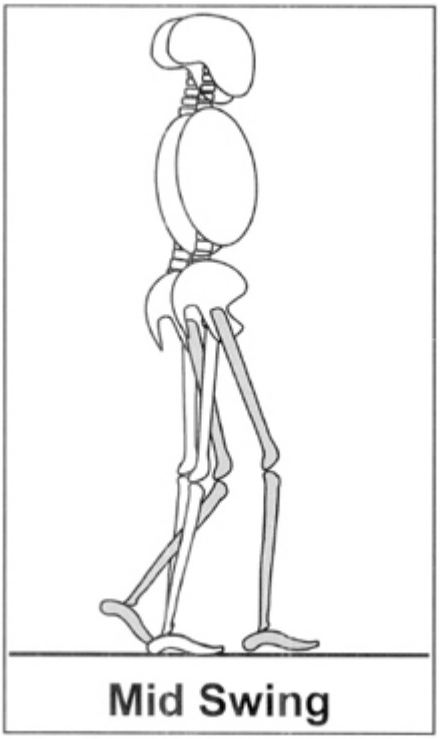

Figure 2-8. Mid swing. Advancement of the limb (shaded) anterior to the body weight line is gained by further hip flexion. The knee is allowed to extend in response to gravity while the ankle continues dorsiflexing to neutral. The other limb (clear) is in late mid stance.

PHASE 7. MID SWING

Interval: 75% to 87% GC

This phase, the middle third of the swing period, begins as the swinging foot is opposite the stance limb. The phase ends when the swinging limb is forward and the tibia is vertical (ie, hip and knee flexion postures are equal) (Figure 2-8).

Objectives:

Limb advancement

Foot clearance of the floor

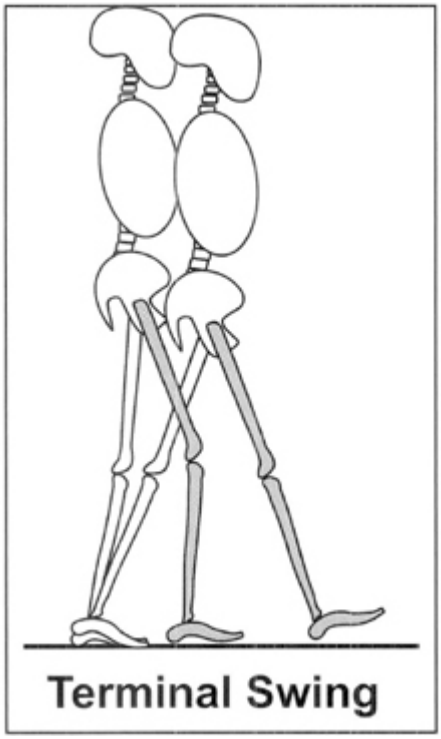

Figure 2-9. Terminal swing. Limb advancement is completed by knee extension. The hip drops slightly (to 20° flexion), and the ankle remains dorsiflexed to neutral. The other limb (clear) is in terminal stance.

PHASE 8. TERMINAL SWING

Interval: 87% to 100% GC

This final phase of swing begins with a vertical tibia and ends when the foot strikes the floor. Limb advancement is completed as the leg (shank) moves ahead of the thigh (Figure 2-9).

Objectives:

Complete limb advancement

Prepare the limb for stance

REFERENCES

1. Pathokinesiology Service and Physical Therapy Department. *Observational Gait Analysis.* 4th ed. Downey, CA: Los Amigos Research and Education Institute, Inc, Rancho Los Amigos National Rehabilitation Center; 2001.

Chapter 3

Basic Functions

Walking forward on level ground is the basic locomotor pattern. A change in direction increases the requirements on the locomotor system. Stairs and rough terrain further the demand. Running and various sports present even greater needs. Despite differences in complexity, they all represent adaptations of the fundamental pattern of walking.

BODY SUBDIVISIONS

During walking, the body functionally divides itself into 2 units: passenger and locomotor (Figure 3-1). While there is motion and muscle action occurring in each, the relative intensity of these functions is markedly different in the 2 units. Basically, the passenger unit is responsible only for its own postural integrity. Normal gait mechanics are so efficient that the demands on the passenger unit are reduced to a minimum, making it virtually a passive entity that is carried by the locomotor system. Alignment of the passenger unit over the limbs, however, is a major determinant of muscle action within the locomotor system.

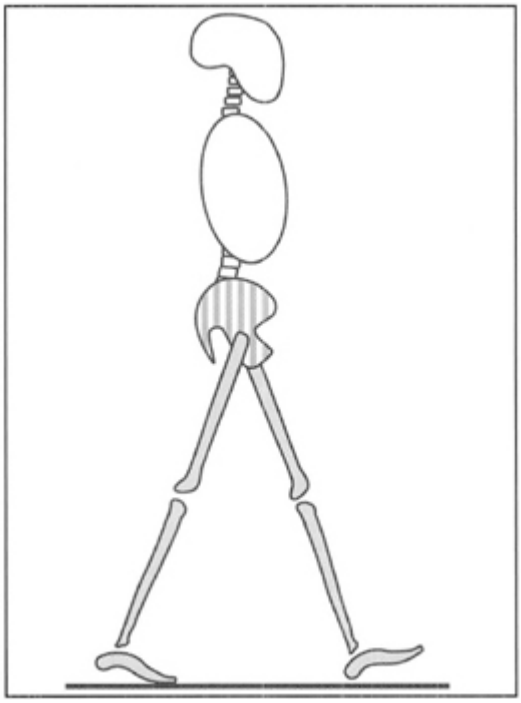

Figure 3-1. Functional division of the body. During walking, the upper body is a relatively passive passenger unit that rides on a locomotor system. The pelvis is part of both the passenger unit and locomotor system.

PASSENGER UNIT

The head, neck, trunk, and arms are grouped as a passenger unit because they are carried and do not directly contribute to the act of walking. Elftman introduced the term *HAT* (*H*ead, *A*rms, *T*runk) to represent this mass (ie, a structure on top of the locomotor apparatus).[11]

Muscle action within the neck and trunk serves to maintain neutral vertebral alignment and to minimize the transmission of postural change from the pelvis to the head. Arm swing involves both passive and active elements, but the action does not appear essential to the normal gait pattern. Experimental restraint of the arms registered no measurable change in the energy cost of walking.[38]

The structures comprising the HAT form a large and heavy mass that represents approximately 70% of body weight (Figure 3-2A).[10,43] Within this composite mass, the center of mass (COM) is located

approximately one-third of the way between the hip joint center and shoulder joint center (Figure 3-2B). As a result, balance of the passenger unit is very dependent upon the instantaneous alignment of the lower limbs to move the base of support under the HAT's momentary center of mass.

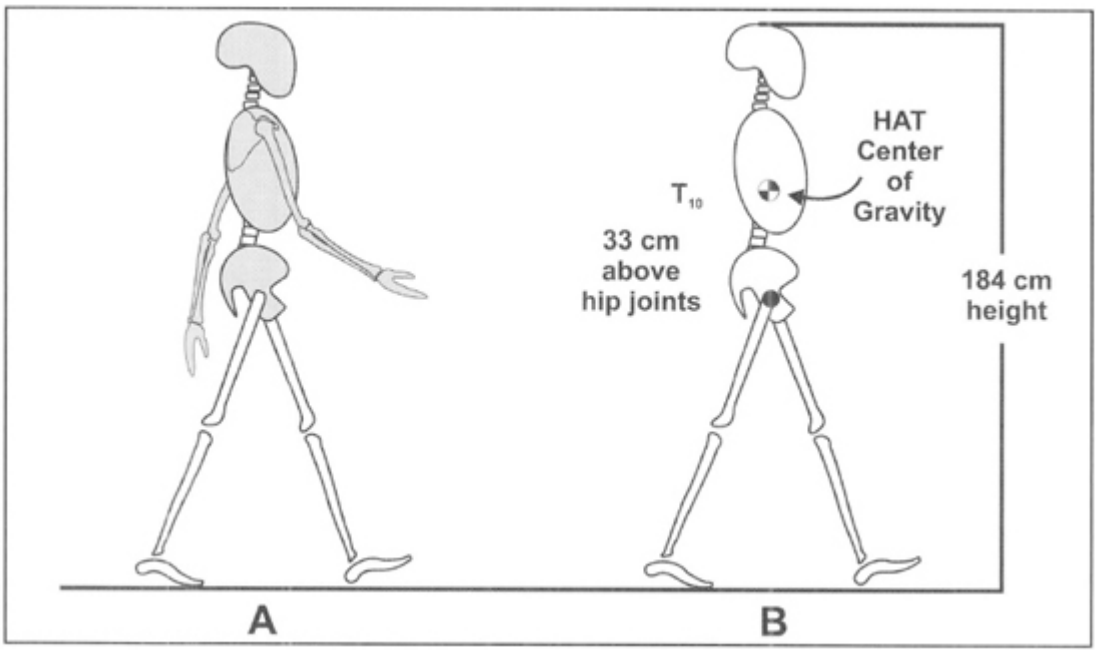

Figure 3-2. The passenger unit. (A) Components are the head, neck, arms, trunk, and pelvis (the HAT unit, shaded gray). (B) The HAT COG lies just anterior to the tenth thoracic vertebra (T10).[22] In a man of average height (184 cm), this point is 33 cm above the hip joint.

LOCOMOTOR UNIT

The 2 lower limbs and pelvis are the anatomical segments that form the locomotor system. Eleven principal articulations are involved: lumbosacral, bilateral hip, knee, ankle, subtalar, and metatarsophalangeal (MTP) joints (Figure 3-3). Timeliness and magnitude of motion in each limb are controlled by the finely modulated activity of 57 muscles functioning in a selective fashion. The bony segments (pelvis, thigh, shank, foot, and toes) serve as levers.

Each limb alternately assumes the responsibility to support the passenger unit in a manner that also carries it forward (Figure 3-4). Then, after being relieved of body weight, the limb rapidly swings itself forward to a new position and prepares to again provide progressive support (Figure 3-5).

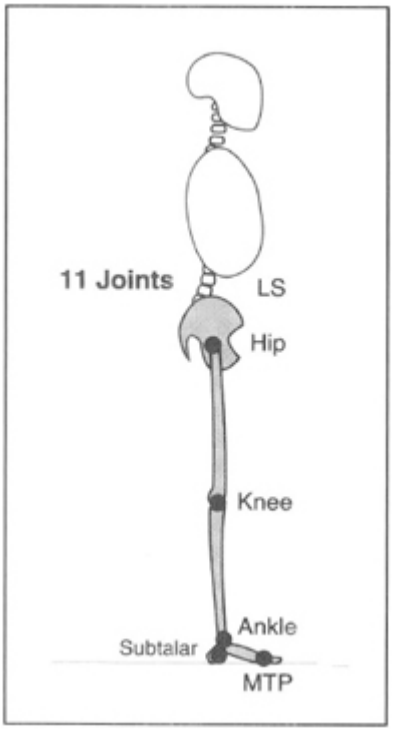

Figure 3-3. The locomotor system includes the pelvis and both lower extremities. This means the pelvis is dually considered a part of the passenger unit and the locomotor system.

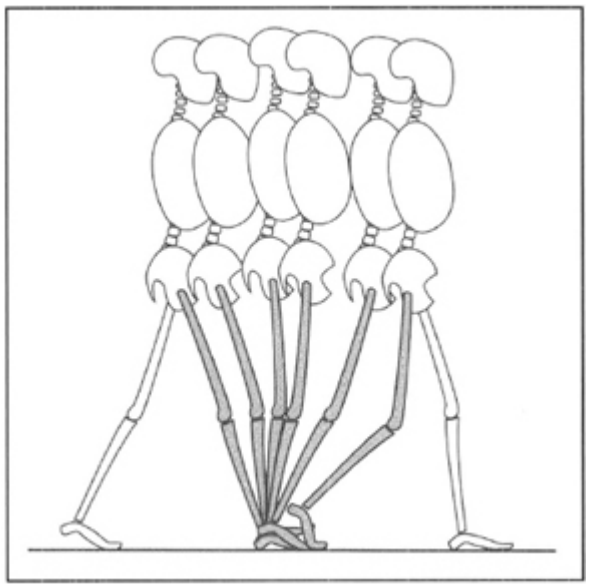

Figure 3-4. During stance, the supporting limb (shaded) provides an advancing base.

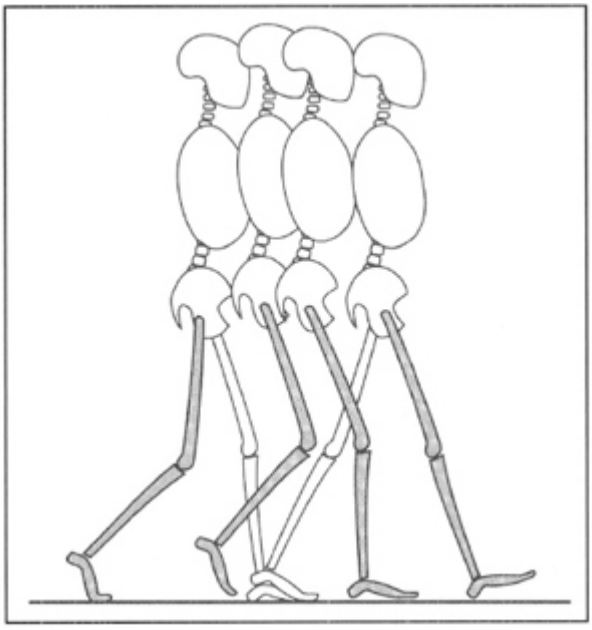

Figure 3-5. During swing, the limb (shaded) advances to initiate WA once again.

The pelvis has a dual role. As part of the locomotor system it is a mobile link between the 2 lower limbs. During swing, the pelvis also advances with the swing limb. The pelvis moves from a backwardly rotated position in terminal stance to a position of forward rotation in

terminal swing (Figure 3-6). In addition, the pelvis serves as the bottom segment of the passenger unit that rides on the hip joints.

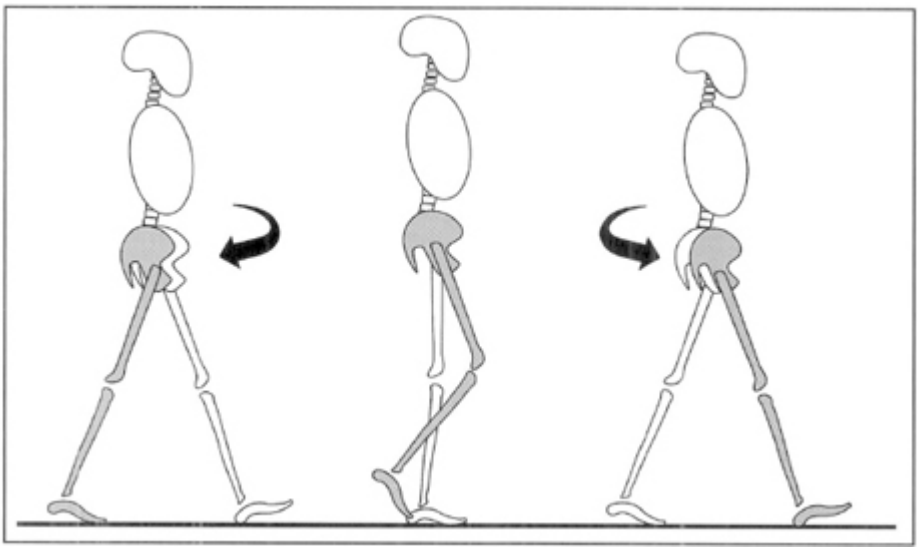

Figure 3-6. Pelvic mobility. Rotation of the pelvis with the swing limb adds to step length. The posterior rotation of the pelvis in terminal stance advances to neutral by the end of initial swing and progresses to forward rotation by terminal swing.

LOCOMOTOR FUNCTIONS

As the locomotor unit carries the body to its desired location, each weight-bearing limb accomplishes the following 4 distinct functions:

1. Upright stability is maintained despite an ever-changing posture.
2. Progression is generated by the interaction of selective postures, muscle force, and tendon elasticity.
3. The shock of floor impact at the onset of each stride is minimized.
4. Energy is conserved by these functions being performed in a manner that reduces the amount of muscular effort required (Table 3-1).

Table 3-1

Locomotor Functions

- Propulsion
- Shock absorption
- Stance stability
- Energy conservation

The simultaneous accomplishment of these 4 functions depends on distinct motion patterns, which present as a complex series of interactions between the HAT and the 2 multisegmented lower limbs. A prerequisite for walking is a stable standing posture.

UPRIGHT STABILITY

Stability in the upright position is determined by the functional balance between the alignment of the body and muscle activity at each joint. Each body segment is a weight that will fall toward the ground (through the pull of gravity) unless it is balanced over its supporting surface or is restrained. There is a balance point within each segment, the COM, that is representative of its weight. When the COM of the upper segment is aligned directly over the center of the supporting joint, the limb has passive stability.

Three anatomical situations challenge standing stability. First is the top-heavy relationship between the passenger unit and the locomotor system. Approximately 70% of body weight is resting on a support system that represents only 30% of the body mass.[10] Second is the multisegmented nature of the supporting limbs. Third is the contour of the lower limb joints.

Alignment of body weight is the dominant factor. The effect of body weight during standing and walking is identified by the ground reaction force vector (GRFV), or “body vector” (Figure 3-7). As body weight falls toward the floor, it creates a force in the floor of equal magnitude but opposite direction. During walking, the alignment of the body vector to the joints is continually changing. By relating the changing alignment of the body vector to the joint centers, the

magnitude and direction of instability can be defined. This provides insight into the demands on muscles and ligaments to either maintain stability or contribute to joint mobility.

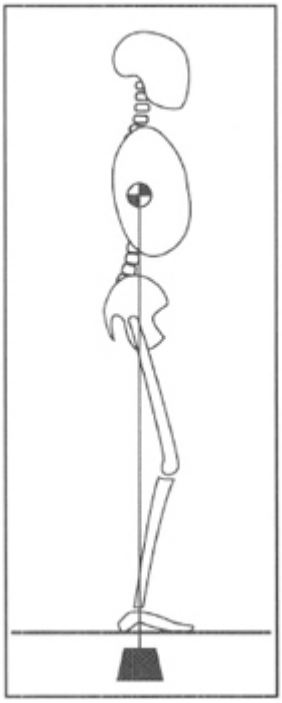

Figure 3-7. Body weight vector. With the flexed knee, the force vector falls behind the knee, requiring knee extensor control.

The ligamentous skeleton is built for mobility rather than stability. Each lower limb is a column of 3 major segments (thigh, shank, and foot). A simple model of the femur and tibia is 2 tall, slender rods aligned vertically over their square ends (Figure 3-8). Their dominance of length over width reduces the tolerance for tilt to less than 9° before passive stability is lost (see Figure 3-8).

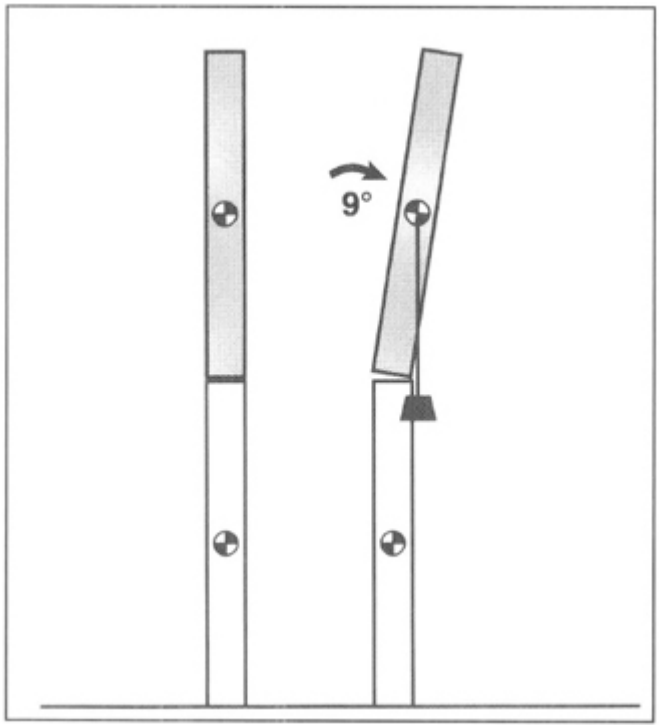

Figure 3-8. Tall rods provide a narrow base and a relatively high center of gravity (COG). Tilting of just 9° moved the COG beyond the base of support, creating an unstable alignment.

Even this margin of stability is not available in the lower limbs of the human skeleton. The slender mass of each segment is slightly top heavy, placing the COM approximately 7% above the anatomical midpoint.[10] The greater curvature of the femoral condyles compared to the tibial plateau enhances motion while reducing passive stability (Figure 3-9). Consequently, whenever the segments' COGs are not in line, the upper segment will fall unless there are controlling forces.

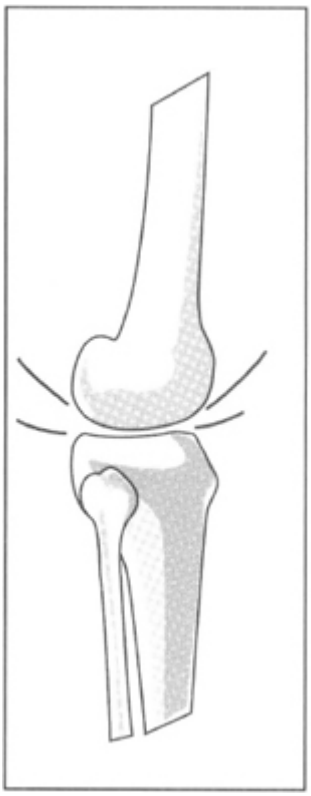

Figure 3-9. The rounded surfaces of the joint reduce the area of stability.

Passive stability at the hip and knee during standing, however, can be attained by substituting ligamentous tension and an opposing vector for absent muscle control. Both joints have a strong ligament on their flexor side. At the knee, it is a dense oblique posterior ligament. Mild hyperextension locks the knee by creating 2 opposing forces. The body vector of the passenger unit is aligned anterior to the knee joint, and the dense posterior ligament becomes taut (Figure 3-10). A similar situation exists at the hip, with its dense anterior iliofemoral ligament restricting extension.

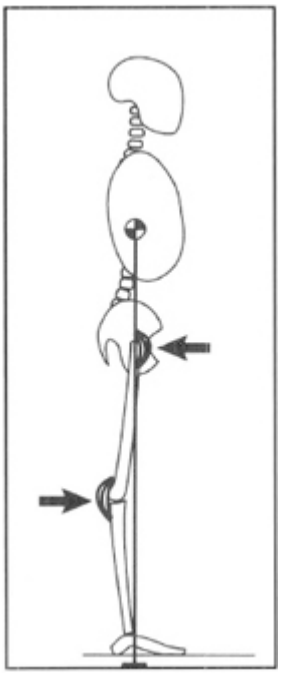

Figure 3-10. During quiet standing, passive stability at the hip and knee is gained by hyperex-tension. The stabilizing forces are ligamentous tension on one side of the joint and the vector on the opposite side. The ankle lacks passive stability.

The ankle, however, has no comparable source of passive stability. Both dorsiflexion and PF have a significant range of motion (ROM) beyond neutral (Figure 3-11). Also, the ankle joint is not located at the middle of the foot. Instead, it is far closer to the heel than to the metatarsal heads (see Figure 3-11). Heel length is further restricted by the support area being well-rounded calcaneal tuberosities, which are almost in line with the posterior margin of the ankle joint. This limits the margin of stability to approximately 1 cm (unpublished data based on informal comparison of skeletons by Dr. Jacquelin Perry). These inequalities further challenge standing stability. If the knee has hyperextension range, a stable balance can be achieved with the ankle slightly plantar flexed. When the knee lacks hyperextension, stable alignment of the limb requires 5° of ankle DF to advance the COG to the middle of the foot. The tilted tibia is now unstable at both the ankle and knee. Muscular control or bracing is essential.

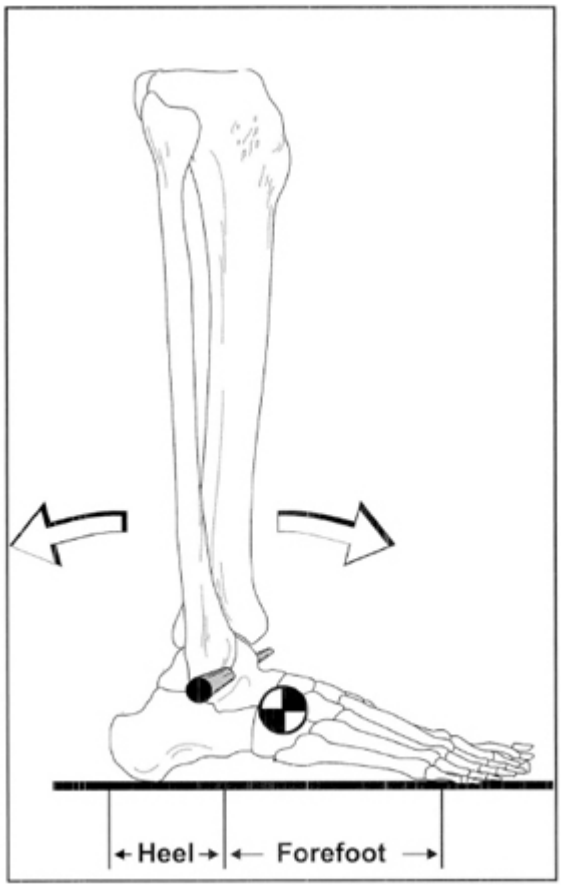

Figure 3-11. The ankle joint is located posterior to the center of the foot (COG marker). Hence, the heel lever is much shorter than the forefoot lever, which extends to the metatarsal heads.

Quiet Standing

As limb posture during quiet standing is similar to that used in mid stance, a person's ability to stand serves as a preliminary test of his or her ability to walk. Attainment of stable alignment requires functional integration of proprioception, joint mobility, and muscle control.

The area of the base of support is the distance between the lateral margins of the feet in the coronal plane and foot length in the sagittal plane. The usual 7° of toeing out by each foot makes the anterior (metatarsal) area wider than that provided by the heels. The mean distance between the medial malleoli during quiet standing averages approximately 9 cm.[33] People normally use 54% of the length

(sagittal plane) and 59% of the width (coronal plane) for voluntary postural deviations and still maintain upright stability.[33] This is considered the limit of postural versatility.

Balance beam measurements of nondisabled persons during quiet standing show that, in the sagittal plane, the body vector extends downward from the center of the head (ear canal), passes 1 cm anterior to the L4 vertebral body, and rests in the foot 1.5 to 5 cm anterior to the ankle (Figure 3-12).[2,3] Force plate measurements place the vector 5 cm (± 2 cm) anterior to the ankle axis.[1,33] The 40% standard deviation confirms considerable variability in knee and ankle mobility and/or the relative strength of the calf muscle group. The normal "easy standing" position uses only a minimal margin of stability, with the body's COG being just 0.6 cm posterior to the hip joint axis and anterior to the knee (see Figure 3-12). Muscle activity may be limited solely to the soleus and gastrocnemius.

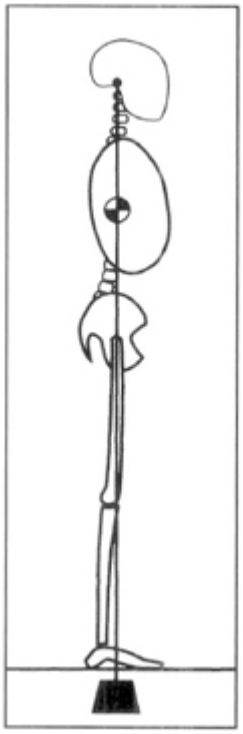

Figure 3-12. With balanced alignment, the body weight vector falls from the ear canal to the middle of the supporting foot (slightly anterior to the ankle joint). It passes slightly anterior to the thoracic spine, just anterior to the knee and barely posterior to the hip joint.

Equal sharing of body weight would place the body vector through the center of the support area. In reality, the normal quiet-standing posture tends to be shifted slightly to the right of midline (0.6 cm).[1,33] The average differences in weight bearing by the 2 limbs have varied with the technique of analysis. Scales placed under each foot revealed a mean 5.4-kg difference, reaching 12.2 kg at the 95% confidence level. In contrast, force plate measurements registered a 0.8-kg difference in vertical force.

Quiet standing is not totally stationary in either the sagittal or coronal plane. Recordings of postural sway reveal a slow, continual shift of body weight between the 2 limbs.[32] The average rate is 4 to 6 cycles per second and the arcs are small (5 to 7 mm laterally, 8 mm anteriorly).[1,33] Two mechanisms contribute to this subtle body instability: cardiac dynamics and the lack of absolute position sense (proprioception).[15,33]

Dynamic Stability

During walking, the body moves from behind to ahead of the supporting foot. At the same time, the area of support changes from the heel to flat foot and then the forefoot. These 2 variables mean that the body lacks passive stability throughout stance. The mean stride width during gait is approximately 7 cm for women and 8 cm for men, and the toe-out angle varies from 5° for women to approximately 7° for men (Figure 3-13).[29,30] Only in the mid point of the stance period does body alignment approximate that of a stable quiet-standing posture.

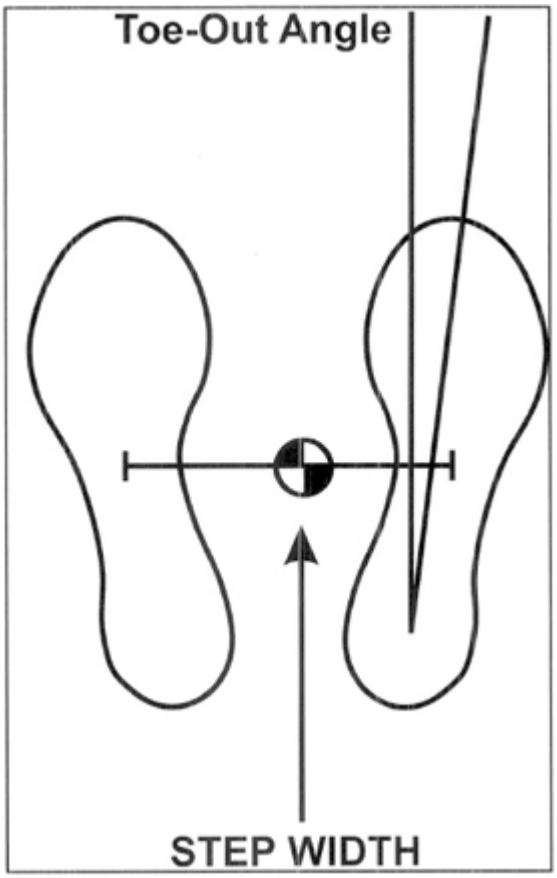

Figure 3-13. Foot alignment during walking is similar for men and women.

As the limb is loaded at the beginning of stance, the anterior placement of the foot relative to the trunk aligns the body vector anterior to the hip joint and posterior to the knee (Figure 3-14A). Response of the extensor muscles at both joints provides an extensor moment that restrains the fall of body weight. During mid stance, the body advances to a position over the supporting foot and the extensor moments approach zero (Figure 3-14B). Advancement of the body beyond this point introduces passive extension at the hip and knee as the trailing posture of the limb aligns the vector anterior to the knee and the erect alignment of the trunk contributes to a flexor moment. At the same time, body weight moves ahead of the ankle, initiating DF and a new area of postural instability. Active control by the plantar flexor muscles is needed to restrain the forward fall of body weight (Figure 3-14C). Thus, throughout stance, muscle action is directed toward decelerating the influences of

gravity and momentum. Extensor moments at the hip and knee during limb loading and a plantar flexor moment at the ankle during SLS reflect the muscular response to the threats to stability.

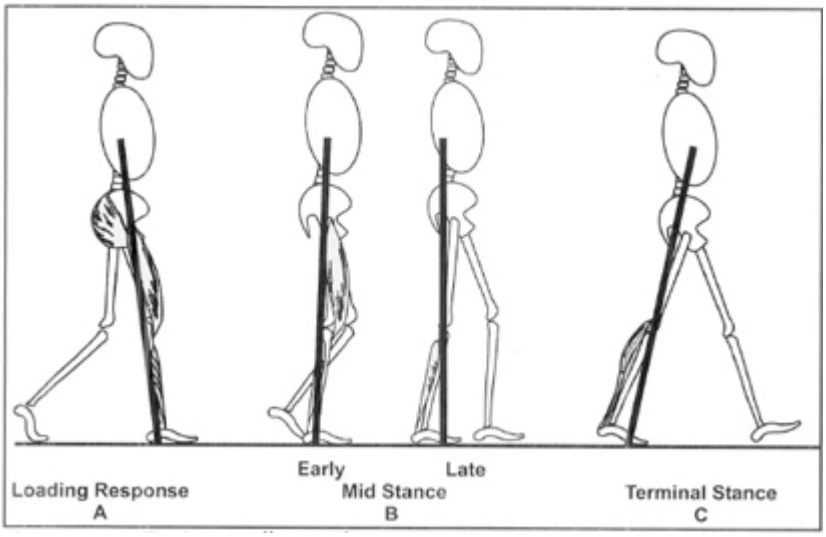

Figure 3-14. During walking, dynamic stability is modified by continual realignment of the vector to the joints. (A) Loading response: The vector is anterior to the hip and posterior to the knee and ankle. (B) Mid stance: At the onset of this phase (early), the body weight vector is slightly behind the knee but anterior to the ankle. By the end of the phase (late), the vector has moved forward of the ankle and the knee. At the hip, the trailing limb posture of the limb has moved the vector posterior to the hip. (C) Terminal stance: The vector is posterior to the hip, anterior to the knee, and maximally forward of the ankle.

Faster walking speeds increase the demands on the decelerating muscles[16,31] as the body vector becomes greater.[9] Conversely, within a limited range, a slower walking speed reduces the required intensity of muscular activity.[16,31] The limitation to this savings is the need for sufficient gait velocity to preserve the advantages of momentum, which is used as a substitute for direct extensor muscle action. An analysis of ankle muscle action demonstrated that the average intensity of muscle activity was equivalent to grade 3 by manual muscle test when walking at 80 meters per minute (m/min).[37] Fast walking (116 m/min) increased the intensity of muscle action to

3+. Walking slowly (56 m/min) reduced the effort to grade 3-.[37] These strengths would be 15%, 25%, and 10% of maximum strength by Beasley's quantified scale.[5]

Single Limb Support

When both feet are in contact with the ground, the trunk is supported on either side (Figure 3-15). This balance is abruptly lost as one foot is lifted for swing. Now the center of the HAT is aligned medial to the supporting limb and the connecting link is a highly mobile hip joint. Two preparatory actions are essential to preserve standing balance over a single limb. These are lateral shift of the body mass and local muscular stabilization of the hip joint to keep the pelvis and trunk erect (Figure 3-16).[39]

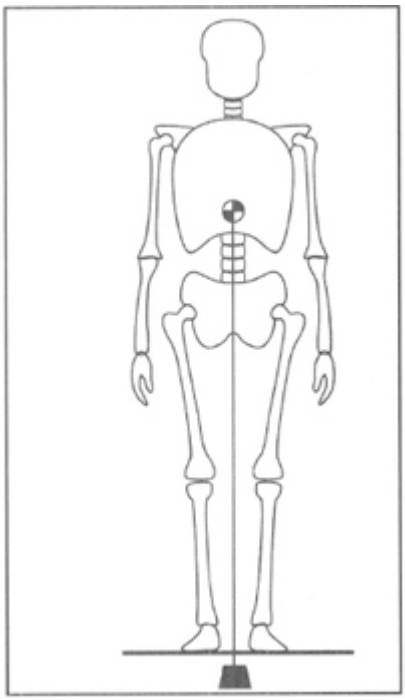

Figure 3-15. In the coronal plane, during quiet standing the body vector (weight line) passes through the middle of the pelvis and between the 2 feet.

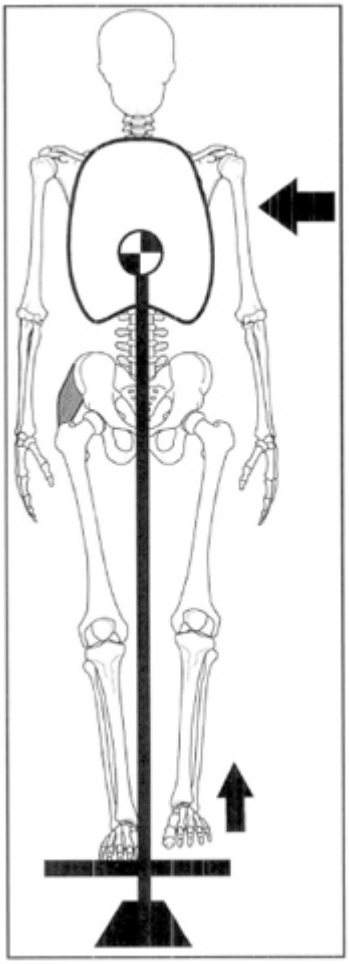

Figure 3-16. Lifting the opposite limb for a step removes the support for that side. Instability is avoided by a shift of the body vector toward the stance limb and strong contraction of the hip abductors to support the unstable pelvis.

During quiet standing, the lateral shift places the center of the trunk over the foot. Both foot and knee valgus are used. Less stability is sought for walking as the swinging limb will be prepared to catch the falling body at the onset of the next step and knee valgus is less.

PROGRESSION

The basic purpose of walking is to move the body from its current site to a new location so the hands and head can perform their

numerous functions. This task is accomplished by a series of events that enable each limb segment to continually roll forward. The major determinants of progression are the initiation of the first step, the action of the foot rockers, forward fall of body weight, and generation of propulsive forces.

The Initial Step

The seemingly simple task of transitioning from standing to walking is really a sequence of motion patterns that are best differentiated by the directions of the center of pressure (COP) and COM displacements.[7,24,26] From its beginning location midway between the stationary feet, the COP sequentially moves in 3 directions (Figure 3-17). First is a lateral and posterior movement toward the "to be" swing limb. Second is a sharp reversal of COP motion medially toward the developing stance foot. Third is anterior displacement of the COP. The first motion pattern has been named "preparation."[7] Logical functional titles for the others are "weight-shift" and "progression."

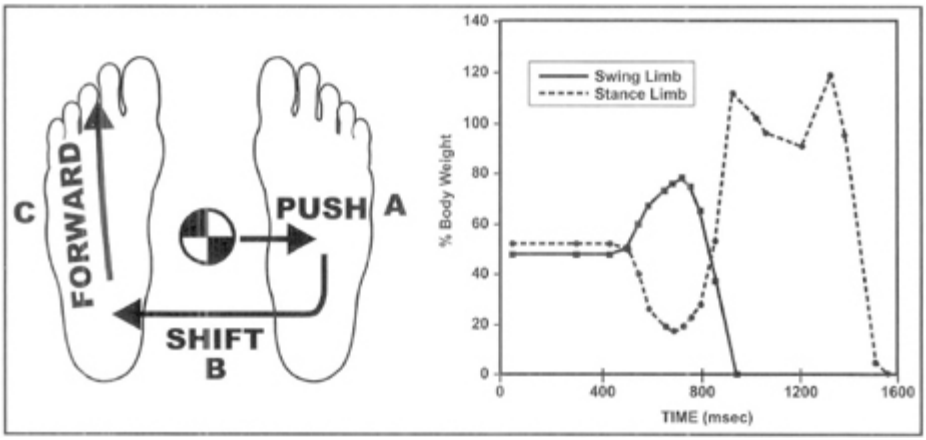

Figure 3-17. Step initiation COP and vertical GRF patterns. (A) Preparation: From the mid-floor position during quiet standing, the COP first moves toward the foot to be lifted and the vertical GRF rises under that limb. (B) Weight shift: Then the COP shifts toward the stance foot and the GRF begins to increase under the stance limb. (C) Progression: The COP moves forward across the stance limb following toe-off of the swing limb. (Adapted from Nissan W,

Whittle MW. Initiation of gait in normal subjects: a preliminary study. *J Biomed Eng*. 1990;12:165-171.)

Preparation

The first motion pattern starts with an inconspicuous change at the ankle by means of bilateral relaxation of the soleus muscles[7] and activation of the anterior tibialis (Figure 3-18).[24] The relative timing between soleus inhibition and anterior tibialis activation was inversely correlated to the velocity of gait.[8] This allows the tibia to fall forward and releases the knees from their stance posture. At the same time, the hip abductor and peroneal muscles of the "to be" swing limb become active,[24] while the muscles of the "to be" stance limb relax and allow the hip and knee to drop into slight flexion.[7] The result is an increase in the force beneath the "to be" swing limb and a reciprocal reduction under the stance limb (see Figure 3-17A).[34]

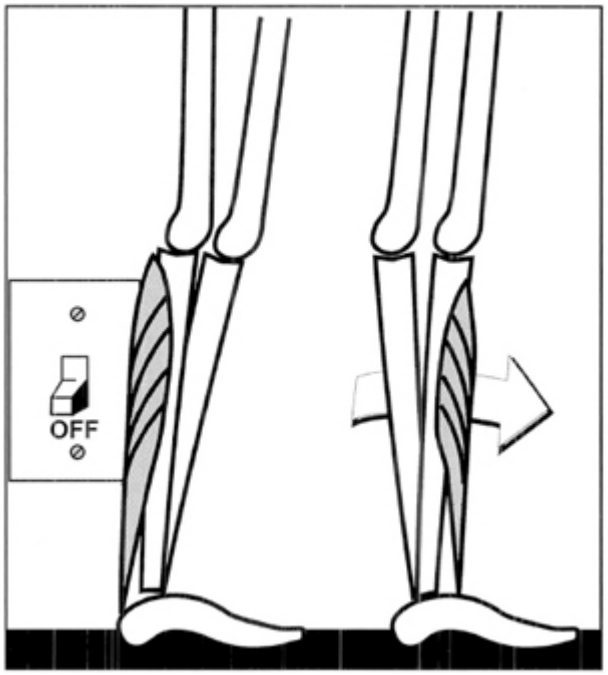

Figure 3-18. Tibial posture in quiet standing determines the initial ankle muscle action. If the tibia is forward with the COP anterior to the ankle, the soleus decreases action (off switch) so passive alignment draws the tibia forward. With the tibia back and the knee

relatively hyperextended, the tibialis anterior (and other pretibial muscles) act to advance the tibia.

The purpose of the COP moving toward the swing limb has been difficult to explain. At the time of the early investigations, displacement of the COP was attributed only to changes in body alignment. This led to the interpretation that the lateral and then medial shifts represented a preliminary test of balance before starting single limb stance.[7,24] Subsequent study of the motion of the body's COM found no lateral displacement (Figure 3-19A).[26] Further biomechanical studies revealed that activation of foot muscles could influence the COP. The current conclusion is that the recorded posterior-lateral displacement of the COP represents the generation of a push force in preparation for the weight shift.

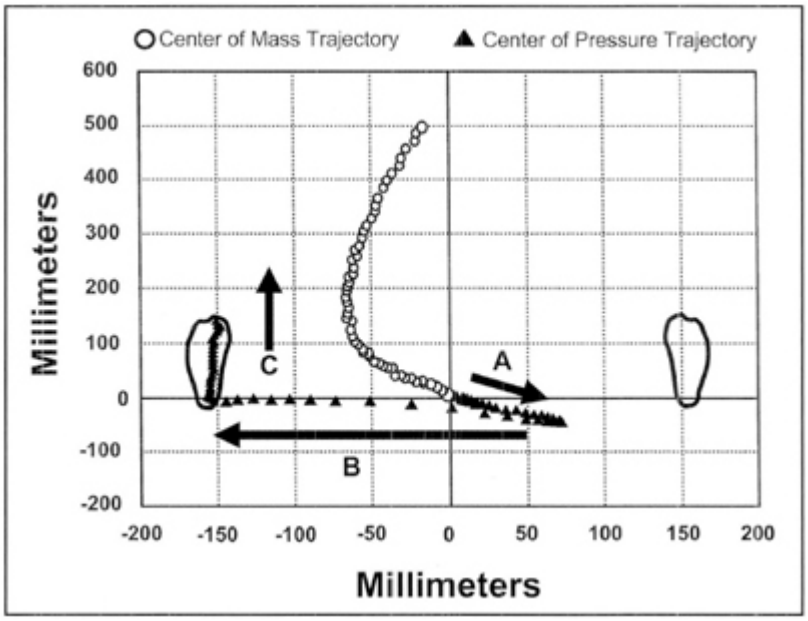

Figure 3-19. Relationship of center of pressure (▲, COP) to center of mass (O, COM) during step initiation. (A) During preparation, only the COP shifts toward the future swing limb. (B) During weight shift, the COP and COM both move toward the stance limb, but the COM does not shift as far. (C) During progression, both COM and COP move forward. The greater shift of the COM reflects the large forward displacement of the pelvis beyond the supporting foot.

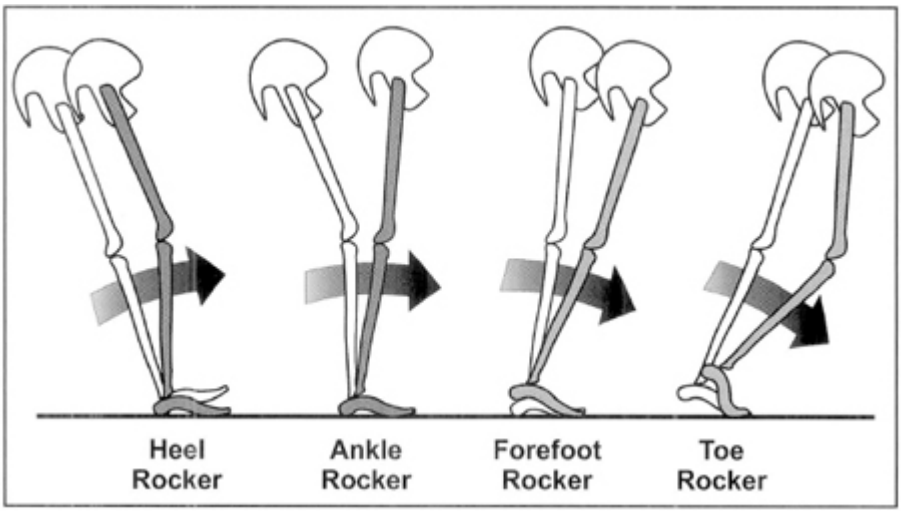

Figure 3-20. Progression (arrows) over the supporting foot is assisted by the actions of the 4 functional rockers: 1) heel rocker; 2) ankle rocker; 3) forefoot rocker; and 4) toe rocker.

Weight Shift

During the second motion pattern, the force under the swing limb is reduced at a rate twice as fast as push force development.[34] Stance limb load is correspondingly increased and its hip abductor and peroneal muscles become active to provide the necessary weight-bearing stability. By the middle of this pattern, the swing limb begins rapid knee flexion followed by a slower arc of hip flexion and continuing ankle DF in anticipation of toe-off.[24,34] Throughout the weight shift, the COP moves medially (see Figure 3-17B).[7,24] The COM accompanies the COP throughout the medial movement (Figure 3-19B).

Progression

Toe-off of the swing limb initiates the third motion pattern. The COP advances the length of the foot (see Figure 3-17C), while the COM progresses anteriorly at a faster rate as the pelvis follows the swing limb.[19] During its forward travel, the COM gradually drifts toward the swinging limb in preparation for the next stance event (Figure 3-19C).

The aim of the gait initiation process is to advance the subject to a steady gait within the first step.[6] From toe-off to heel strike of the first

swing, the velocity of the COM reaches 91% of the rate accomplished by the second and subsequent steps.[19] A steady step rate is established by the second step.

Foot Rockers

Once walking has been initiated, advancement of the body over the supporting foot depends on stance limb mobility. The challenge begins at the onset of stance with the rapid drop of body weight onto the forward limb following initial foot contact. The thigh is flexed only 20°, consequently, the force is primarily directed toward the floor. Preservation of the body's forward progress requires that some of this force be redirected in a manner that combines progression and stability. The foot responds by providing a pivot system. In serial fashion, the heel, ankle, forefoot, and toes serve as rockers, which allow the body to advance smoothly (Figure 3-20).

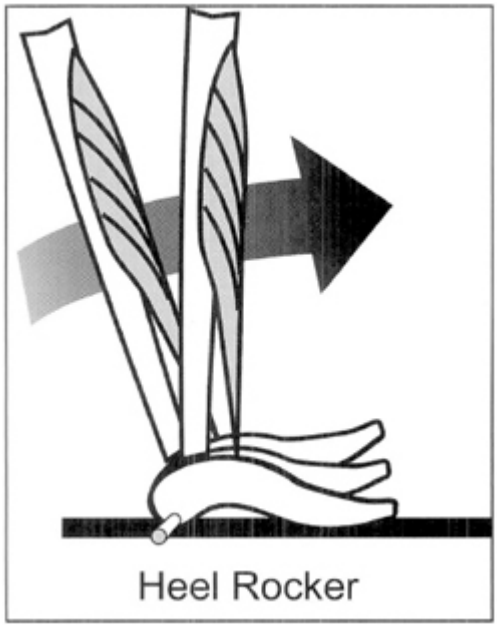

Figure 3-21. Heel rocker. Using the heel as the fulcrum (rod designates motion axis), the foot rolls through a short arc of PF. Pretibial muscles decelerate foot drop and draw the tibia forward, preserving the heel rocker until the end of loading response.

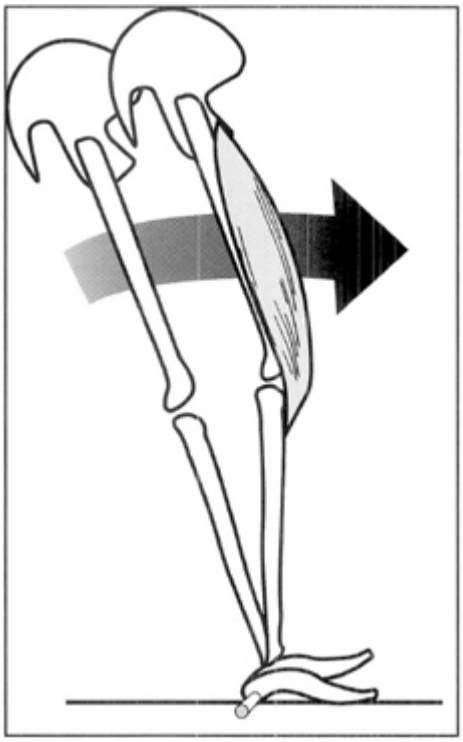

Figure 3-22. Quadriceps action draws the femur forward at a slower rate than the tibia advances.

Heel Rocker

As body weight is dropped onto the stance limb, the momentum generated by the forward fall is preserved by the heel rocker (Figure 3-21). Floor contact is made by the rounded surface of the calcaneal tuberosities. The bony segment between this point and the center of the ankle joint serves as an unstable lever that rolls toward the ground as body weight is dropped onto the foot. Action by the pretibial muscles to restrain the rate of foot drop creates a tie to the tibia that draws the leg forward. This progressional effect is transferred to the thigh by the quadriceps (Figure 3-22). While acting to restrain the rate of knee flexion, the quadriceps muscle mass also ties the femur to the tibia. In this manner, the heel rocker facilitates progression of the entire stance limb. As a result, the force of falling, rather than being totally directed toward the floor, has a significant portion realigned into forward momentum. By the end of the heel rocker (12% GC), the whole foot will be in contact with the ground, the tibia will be vertical, and the knee will have flexed to 20°.

Ankle Rocker

Once the forefoot strikes the floor, the ankle becomes the fulcrum for continued progression. With the foot stationary, the tibia continues its advancement by passive ankle DF in response to the momentum present (Figure 3-23). The body vector advances along the length of the foot to the metatarsal heads during this period. A critical aspect of the ankle rocker is the yielding quality of the soleus musculotendinous complex. As it contracts to make the tibia a stable base for knee extension, the soleus assisted by the gastrocnemius also allows tibial advancement.

Figure 3-23. Ankle rocker. With the ankle as the fulcrum (rod designating the axis of motion), the tibia (and whole limb) rolls forward in response to momentum (arrow). The rate of tibial progression is decelerated by the soleus muscle.

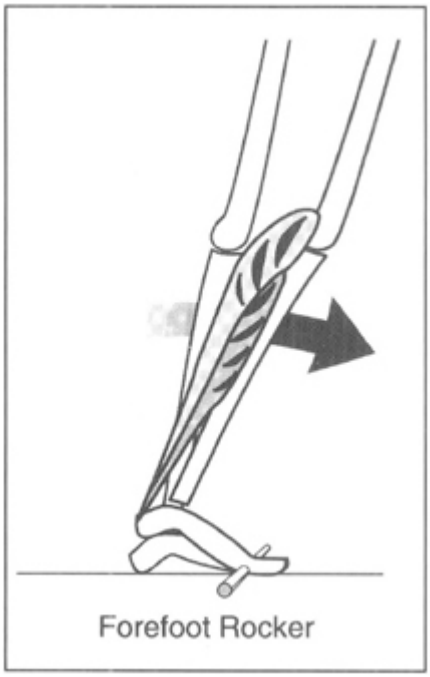

Figure 3-24. Forefoot rocker. Tibial progression (arrow) is continued over the forefoot rocker (rod as the axis). The gastrocnemius and soleus are active to stabilize the ankle.

Forefoot Rocker

As the base of the body vector (center of pressure) reaches the metatarsal heads, the heel rises. The rounded contour of the metatarsals serves as a forefoot rocker (Figure 3-24). Progression is accelerated as body weight falls beyond the area of foot support (Figure 3-25). This is the strongest propelling force during the GC. The body mass is a passive weight at the end of a long lever and its rate of fall is restrained by vigorous action of the gastrocnemius and soleus.

Toe Rocker

In pre-swing, the most anterior margin of the medial forefoot and the great toe serves as the base for accelerated limb advancement. Elastic recoil of the plantar flexors thrusts the tibia forward (see Figure 3-20).[12]

Propulsive Forces

One basic force available to advance the stance limb is forward fall of body weight (see Figure 3-25). Also, hip flexion, in the process of lifting and advancing the swing limb, creates an anterior (pulling) force that encourages progression of the body vector. Faster rates of hip flexion add acceleration, which increase walking speed (Figure 3-26).[25,27] Active knee extension in swing provides an additional pulling force.

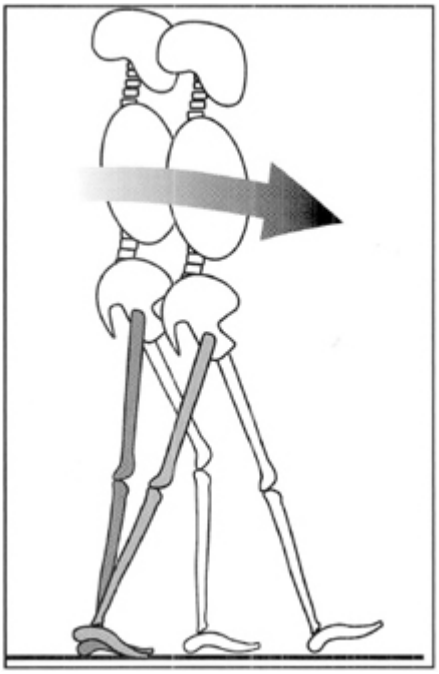

Figure 3-25. Forward fall of body weight (arrow) is a primary force contributing to progression.

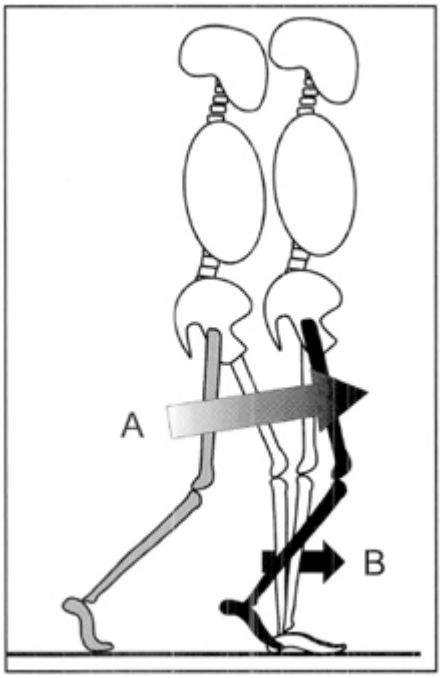

Figure 3-26. A progressional force (arrow) also is provided by the swinging limb (gray).

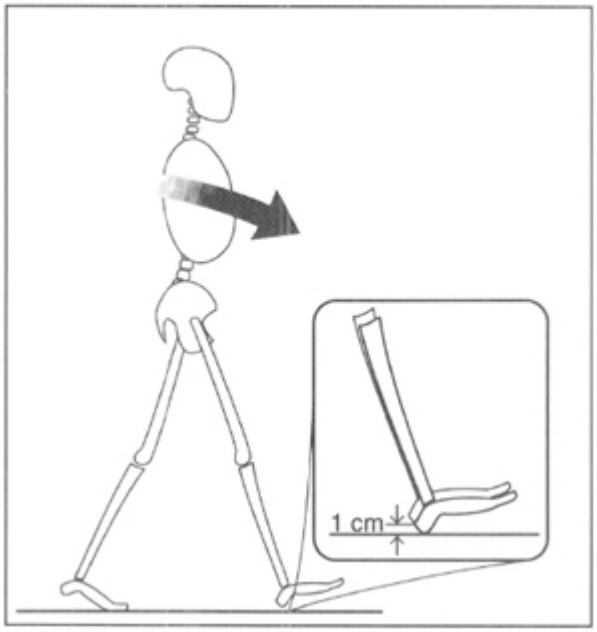

Figure 3-27. Floor contact is abrupt because body weight had a free fall (arrow) for about 1 cm (insert). This is the distance between the heel and the floor at the end of terminal swing.

Normal walking velocity, however, utilizes additional propulsion forces generated by the timely interplay of vector alignment and

muscle action. At critical intervals, eccentric muscle activity (which provides stance stability) changes to concentric action to accelerate or preserve progression. This transition is not identified by electromyography (EMG), as muscle recordings detect neural stimulation, not the mechanics of contraction. Power analysis, which integrates the moments of force with joint velocity, is a measure that is sensitive to the transition from eccentric to concentric muscle activity.[43] Positive peaks of power indicate concentric muscle action is parallel with the motion.

During the typical GC, there are 4 phases where continuity of function is augmented by a positive peak of power. At the end of loading response, there is a small power boost associated with the initiation of hip extension. In early mid stance, completion of knee extension occurs in association with a quadriceps power burst of similar magnitude. The largest power burst occurs at the ankle during pre-swing. As body weight is abruptly transferred to the contralateral limb, release of the taut soleus and gastrocnemius musculotendinous complex generates elastic recoil, which prepares the limb for swing.[12] This action is commonly called *push-off*. Rapid onset of hip flexion at the beginning of swing is the fourth power burst.

Shock Absorption

Transfer of body weight from the trailing limb to the forward foot begins as an abrupt exchange even though it occurs during a double stance interval. At the end of the single support period, body weight has moved beyond the margin of stability available from the trailing forefoot. The resulting loss of stability causes the body to fall forward and down. At the start of the fall the foot of the weight-accepting limb, while positioned for stance, is still about 1 cm above the floor's surface (Figure 3-27).[28] Hence, the body is in a virtual free fall for a short period. The full intensity of the floor impact is reduced by shock-absorbing reactions at the ankle, knee, and hip.

At the ankle, heel contact initiates a 5° arc of PF before the pretibial muscles catch the foot (Figure 3-28).[31,36] Then the pretibial

muscles rapidly restrain the ankle and delay forefoot contact with the floor until the 12% point in the GC. Loading the limb is so rapid that 2 ground reaction force (GRF) patterns are generated. First is an instantaneous “heel transient,” which partially interrupts the initial ascending arm of the GRF (60% body weight in 0.02 seconds) (Figure 3-29).[40,42] Weight transfer then continues to the first peak (F1) of the GRF record, which occurs at the end of loading response.

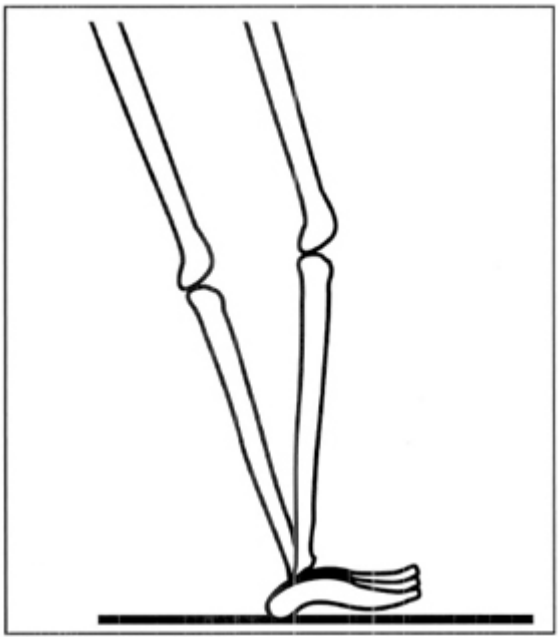

Figure 3-28. The immediate shock-absorbing reaction to floor impact is a small arc of ankle PF and subtalar eversion following heel contact. Response of the pretibial muscles preserves the heel rocker.

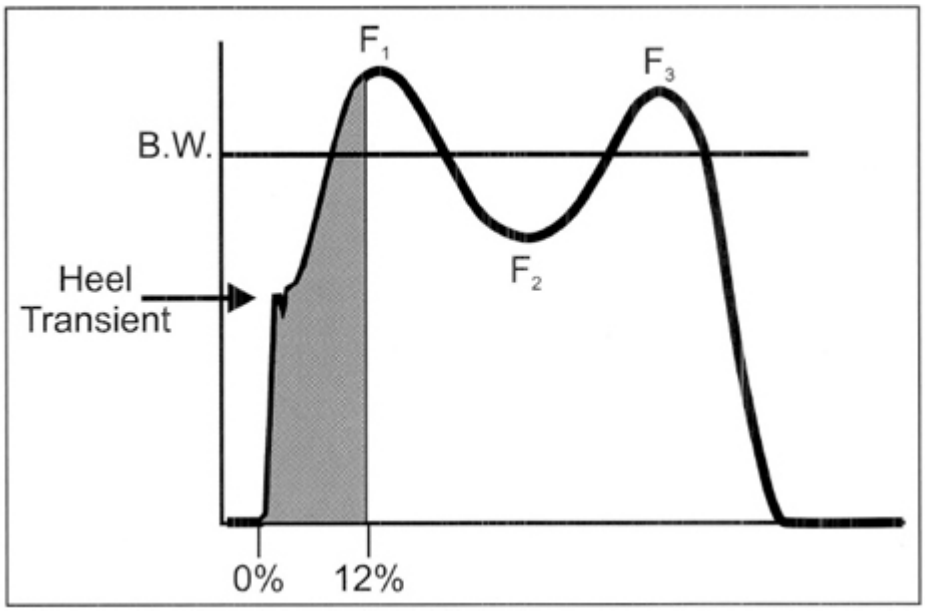

Figure 3-29. The effect of terminal swing free fall is an abrupt floor impact (heel transient). F1 and F3 are the 2 peaks in the vertical GRF, while F2 is the valley. B.W. = body weight.

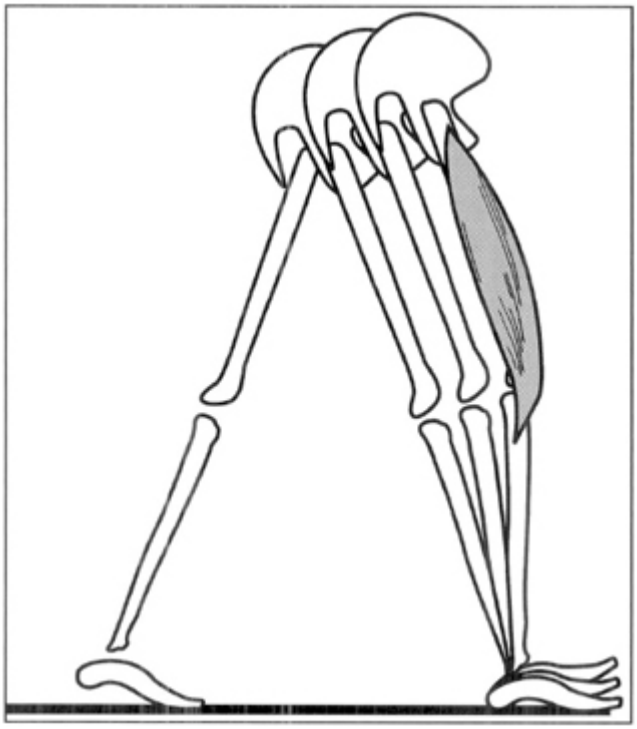

Figure 3-30. Knee flexion, restrained by the quadriceps, is the second shock-absorbing reaction to floor contact.

Knee flexion is the second and largest shock-absorbing mechanism. This motion also is a reaction to the heel rocker initiated by floor contact. As the pretibial muscles restrain the falling foot, their tendinous attachments to the tibia and fibula cause the leg to follow the foot. Forward roll of the tibia initiates rapid knee flexion as the joint center moves anterior to the body vector. As the quadriceps contracts to decelerate the rate of knee flexion, the muscle also absorbs some of the loading force (Figure 3-30). Both the joint-loading force and floor impact are reduced. The shock experienced by the limb is documented as the F1 of the sagittal plane GRF record (see Figure 3-29). At the normal walking speed, the acceleration from the fall raises the F1 peak to approximately 110% of body weight.

Abrupt loading of the WA limb also reduces support beneath the trailing limb, contributing to a contralateral pelvic drop. Prompt response by the lead limb's abductor muscles absorbs some of the impact as they minimize the displacement (Figure 3-31).

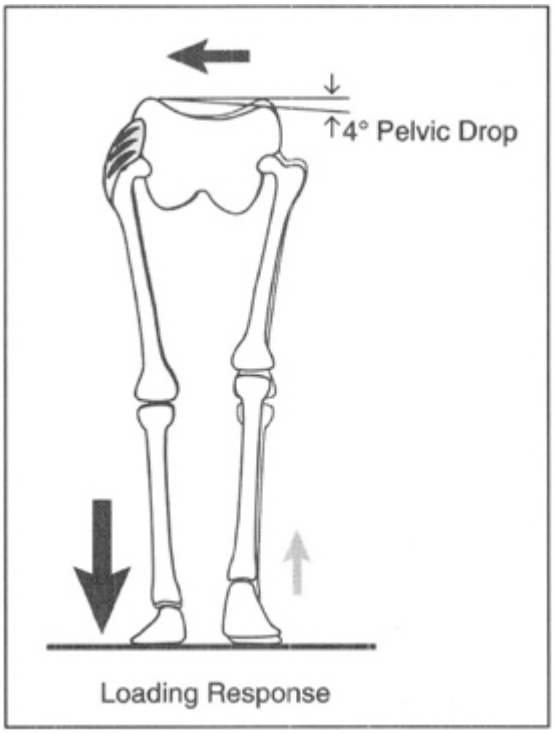

Figure 3-31. Contralateral pelvic drop decelerated by the hip abductors provides an additional shock-absorbing maneuver. This

occurs as weight is rapidly dropped onto the loading limb (large arrow) as the other limb is being lifted (small arrow).

The sum of these muscular reactions to the abrupt passive destabilization of the ankle, knee, and hip by the free fall of body weight reduces the force of limb loading. Each joint between the HAT and the foot, including the lumbosacral joint, has received some protection from the potential injury of abrupt impact.

Energy Conservation

The efficiency of doing any activity is the ratio between the work accomplished and the energy expended.[4] During walking, preservation of stance stability by selectively restraining falling body weight and facilitating body advancement over each supporting foot for the desired distance constitutes the work being performed. Engineers define “work” as the product of a force times the distance the force acts. Physiologically, the concern is the amount of muscular action required, both magnitude and duration. The relative intensity of the muscular effort expressed as a percent of each muscle’s maximum capacity indicates the person’s capability of performing the task. Intensity combined with the duration of muscular effort required determines the energy cost.

Unlimited endurance requires that the energy cost of walking be less than the cardiopulmonary mid-point in a person’s maximum energy production capacity.[4,41] This energy threshold is expressed as 50% maximal aerobic capacity (VO_2 max). Normal walking at the average speed of 82 m/min uses energy at a rate that is 38% of the maximum. The fact that walking requires less than 50% VO_2 max, and thus does not reach the anaerobic threshold, accounts for why healthy adults perceive that walking requires only minimal effort.[41]

To maintain the normal walking velocity, 2 mechanisms for conserving energy are used. These are COG alignment modulation and selective muscular control. Both serve to reduce the intensity and duration of the muscular action involved.

Center of Gravity Alignment Modulation

Minimizing the amount the body's COG is displaced from a level line of progression is considered to be a major mechanism for reducing the muscular effort of walking and consequently for saving energy. The least energy would be used if the weight being carried remained at a constant height and followed a single central path. No additional lifting effort would then be needed to recover from the intermittent falls downward or laterally.

Dependence on reciprocal bipedal locomotion, however, presents 2 potentially costly situations during each stride: changes in lateral alignment and height of the COG. As the right and left limbs alternate their support roles, the body must shift from one side to the other to preserve balance. The limbs also change their vertical alignment between double and single support intervals, causing a change in the height of the pelvis. As a result, the body mass moves up and down. The body is at its lowest point when the limbs become obliquely aligned during the 2 double support periods (initial and terminal). Then in mid stance, the body is raised to its highest position when the supporting limb (right or left) is vertical (Figure 3-32).

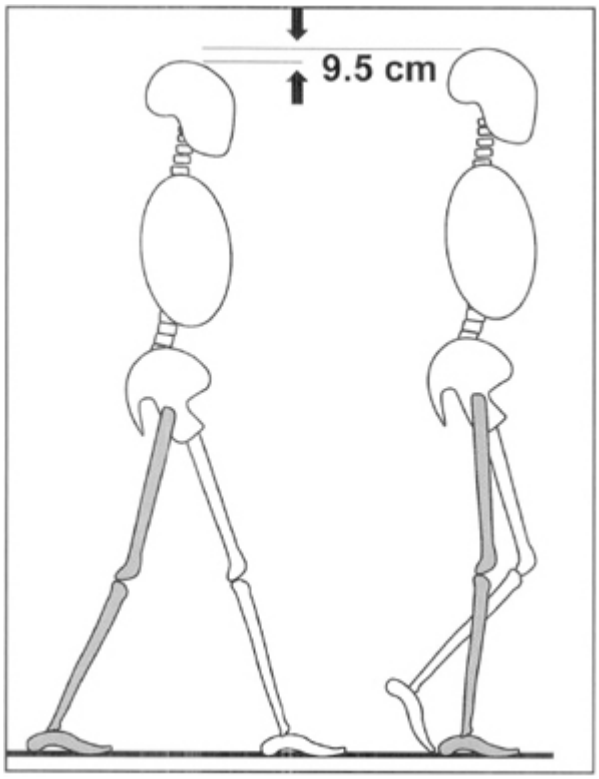

Figure 3-32. The change in body height between double and SLS would be 9.5 cm if no modifying action were performed.

Saunders, Inman, and Eberhart, pioneers in comprehensive gait analysis, identified 6 motions that they called the "Major Determinants of Gait."[39] Cameras recorded the motion of each joint in the 3 orthogonal planes. The displacement of bone and surface markers was determined by manually measuring each frame of film. Relationships were calculated with slide-rulers. From this intense, 250 man-hours of data acquisition per stride, the authors concluded that energy of walking was conserved by a combination of motions that reduced the magnitude and abruptness of the directional changes. They calculated a 9.5-cm potential difference in hip height throughout the GC and an 8-cm side-to-side displacement of the body's COG.[39] It was concluded that these magnitudes of repeated body displacement would quickly introduce fatigue. By conceptually applying their 6 determinants of gait, they determined that the magnitude of these costly vertical and horizontal displacements could be reduced to just 2.3 cm in each direction for a total arc of 4.6

cm (Figure 3-33).[39] This represents more than a 50% improvement. In addition, abrupt changes in direction are avoided.[18]

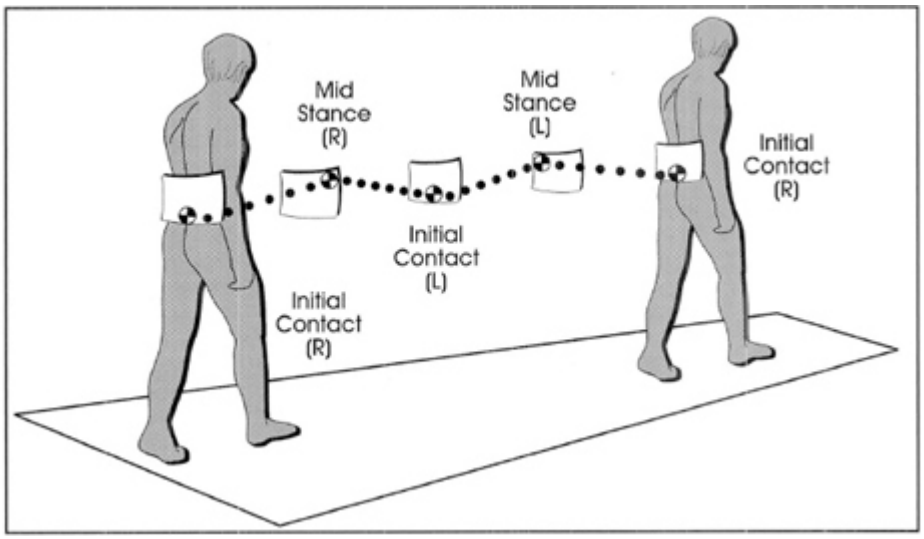

Figure 3-33. The normal path of the COG (black/white circle) illustrated by a stride beginning with the right foot. From the low, central point in double limb support (right initial contact), the COG moves upward and laterally (right mid stance), drops to a second central low point (left initial contact), rises to a peak again (left mid stance), and drops once more (second right initial contact).

More recent analysis of the COG kinetics during normal gait, using modern photography and computer programming of the motions, showed similar responses but slightly smaller magnitudes of displacement.[17] Lateral shift of the COG averaged 3.5 ± 0.9 cm, while the mean vertical displacement was 3.2 ± 0.8 cm. Individual values varied mostly due to the impact of gender differences on body size and gait velocity.[17,35]

The more precise timing of events provided by modern technology, however, contradicted the earlier reports on two of the determinants. While “pelvic list” and “knee flexion” do selectively lower peak trunk height, the timing of their peak effect is too early.[13,14] Both motions lower the trunk height during loading response (12% GC); however, peak trunk height occurs later at the end of mid stance (29% GC). At that time, the effect of pelvic list and knee flexion is only 2 to 4 mm. The pelvic list and knee flexion

motions during loading response, however, introduce a 10% to 15% phase shift that smooths the trunk displacement curve.

Two of the remaining “determinants” (rotation and transverse shift) modify the pattern of pelvic motion (Figure 3-34). The pelvis rotates forward 5° in the transverse plane as it follows the swinging limb (see Figure 3-34A). This advances the hip joint of the swing limb slightly anterior to the hip of the stance limb. Conceptually, the distance between the 2 hips is lengthened, resulting in an increase of the functional limb length between foot contact and the center of the base of the HAT. Pelvic rotation also moves the hips closer to the midline. Both effects are predicted to reduce the amount of limb obliquity that is needed to accomplish the desired step length. Trunk displacement is reduced and the curve is smoothed.

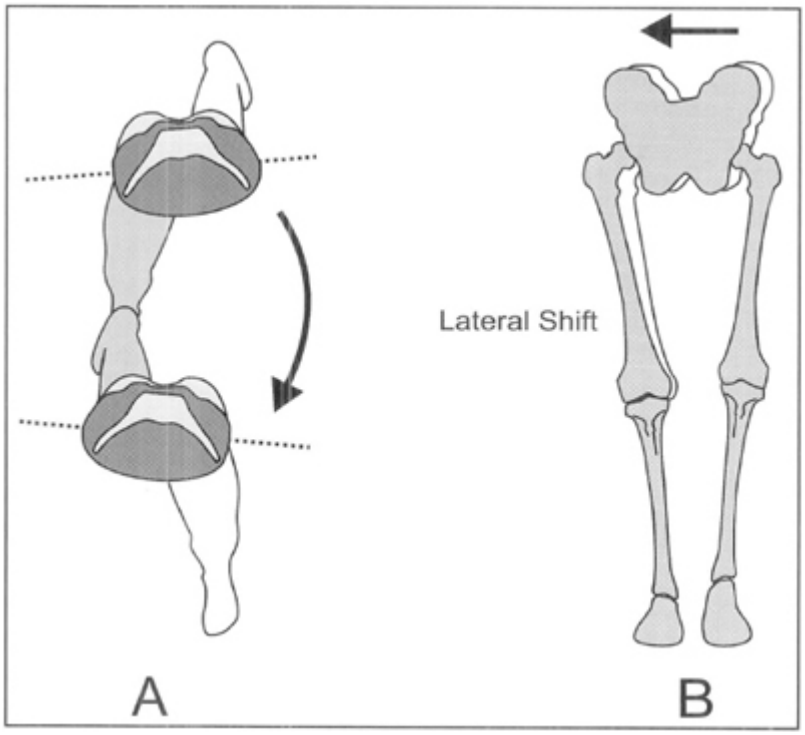

Figure 3-34. Pelvic maneuvers to minimize COG displacement in a stride include (A) transverse rotation and (B) lateral shift.

Lateral displacement of the pelvis during WA involves 2 factors (see Figure 3-34B). First is the natural valgus angle between the femur and the tibia, which places the knees (and supporting feet) closer to each other than a vertical line down from the hip joints

would. Anatomical width between the hip joints approximates 20 to 25 cm. Normal step width is 8 cm.[29] As the limb is loaded, there is a slight increase in knee abduction that moves the body's COG nearer to the supporting foot.

In addition to the anatomical narrowing of step width, the body does not fully align itself over the supporting foot as would occur in single limb standing. The potential imbalance is controlled by inertia. By the time body weight loses its lateral momentum and is about to fall to the unsupported side, the swing limb has completed its advancement and is prepared to accept the load.

The final determinant relates to the contribution of the ankle to smoothing the pathway of the trunk. At the onset of double limb support when the trunk height is least, the position of the ankles adds limb length. The neutral ankle position at initial contact makes the heel the most distal segment of the forward-reaching limb (Figure 3-35). Heel rise lengthens the trailing limb in terminal stance. This advantage has been noted by the clinical observation that the ipsilateral pelvis drops when heel rise is lacking.

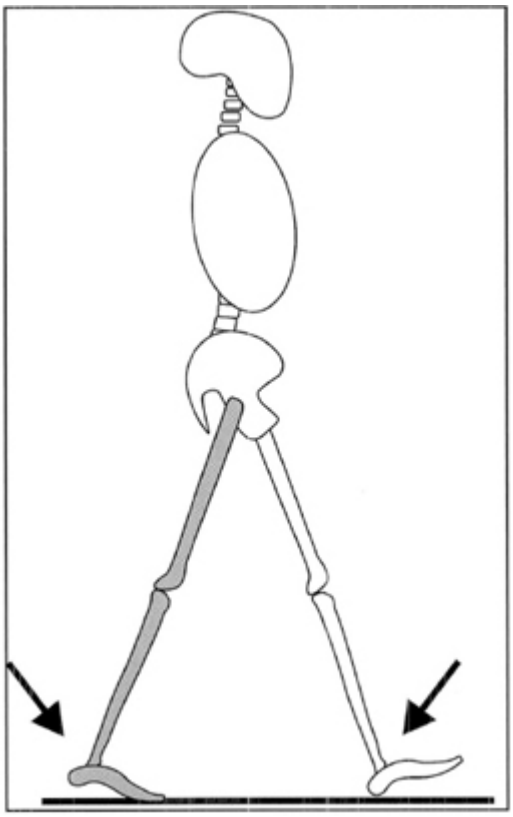

Figure 3-35. Ankle actions to elevate COG. Heel rise in terminal stance preserves pelvic height while ankle DF in terminal swing minimizes the need for a drop at initial contact.

In summary, the magnitude of trunk displacement is minimized by multiple small postural changes at the pelvis, knee, and ankle. As a result, the body's COG follows a smooth three-dimensional sinusoidal path, which combines vertical and horizontal deviations.

Selective Muscular Control

The ability to move one joint in a particular direction and at a designated speed or to combine antagonistic motions such as flexion of one joint and extension of a second joint in the limb is selective control. During gait, muscles are selectively activated based on their contributions to movement in all 3 planes and across multiple joints. Walking is voluntarily initiated but once the motion pattern is learned, it is stored and can be performed subconsciously as needed.

Each muscle is activated at a specific time and at a select intensity to control the path of the limb segments for a designated duration. To begin the action the motor control system must know the current position of each joint in the limb as well as the position of the body in order to select the muscles to be activated. Secondly, the system must be aware of the ongoing changes in the postural relationships of the limb segments to complete the movement.

Immediate and continuing position and motion awareness of each limb segment and the body as a whole are essential to selective control. This information is provided by the kinetic sensory system (proprioception and kinesthesia). Specialized sensory organs in the tendons, muscles, ligaments, and skin identify and monitor each segment from onset to cessation. Pertinent factors include the starting positions, relative weight of the limb segments, status of the controlling muscles, and rate of motion. Persons who lack proprioception often have difficulty walking, even though they can move their joints easily.[20,21] Information regarding body balance in relation to the environment is sensed by the inner ear and assists with planning movement.

Clinical assessment of these sensory functions, however, is fairly gross. Only 3 levels of sensation are determined (normal, impaired, and absent) and the grade is dependent on the accuracy of the patient's awareness. Subtle gradation in single limb stance and double limb stance obscure the boundaries, and loss of body image masks the lack of balance. Hence, lesser levels of sensory impairment are interpreted from imprecise performance.

Selective control is essential for the normal sequence of motion during gait, and the functional muscle groups alter their timing and intensity in an asynchronous manner to achieve the different phases of stance (Figure 3-36) and swing (Figure 3-37). WA combines activity of the hip[23] and knee extensors with action of the ankle dorsiflexors. During early mid stance, activity of the calf muscles replaces pretibial muscle activation, the thigh is controlled just by the quadriceps, and the hip extensors cease to be active. By the onset of terminal stance, only the plantar flexors are required to stabilize the ankle, knee, and hip. During initial swing, the flexors of the hip, knee, and ankle are activated, while in mid swing flexors at only the hip and ankle are required. Terminal swing changes hip and knee control to the extensors, while the dorsiflexors continue to control the ankle.

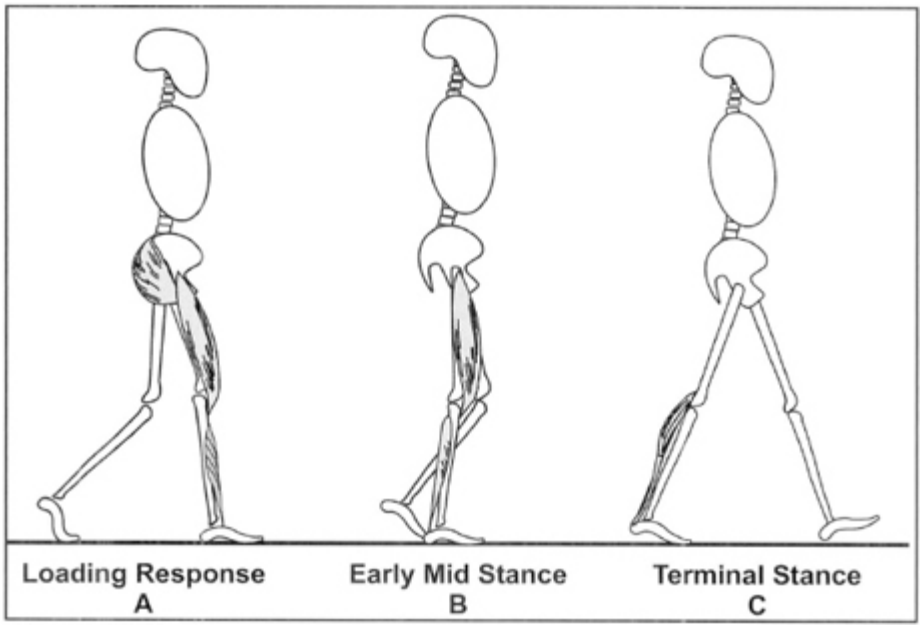

Figure 3-36. Selective control of gait during stance. Loading response requires selective control of hip and knee extensors with ankle dorsiflexors. During early mid stance, limb control is reduced to the quadriceps and calf. By the onset of terminal stance, limb control requires only the calf.

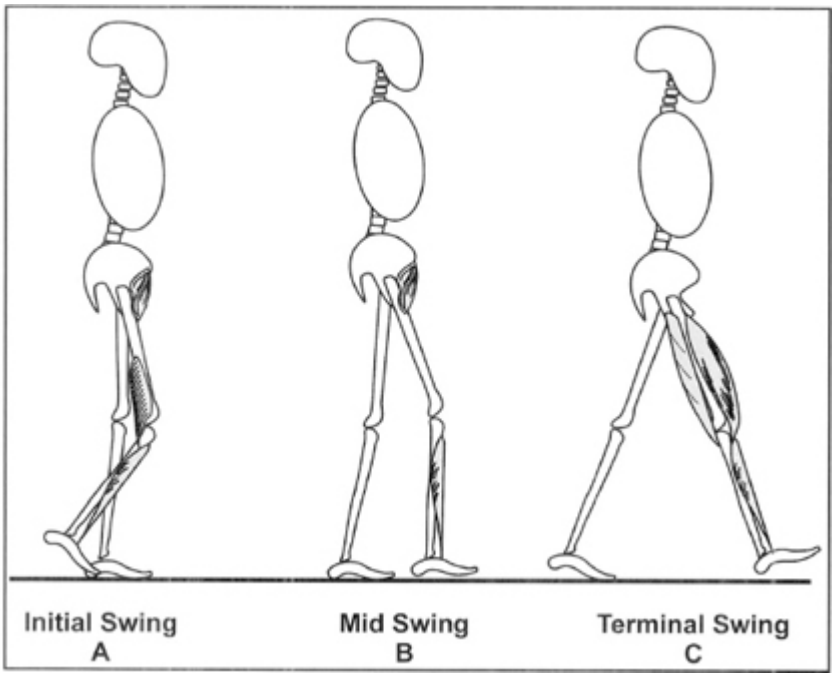

Figure 3-37. Selective control of gait during swing. During initial swing, the flexors are active at each joint, while in mid swing, flexors are active primarily at the hip and ankle. Terminal swing requires a combination of extensor (hip, knee) and flexor (ankle) muscles.

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Section II

Normal Gait

Chapter 4

Ankle-Foot Complex

The starting point for observing gait often focuses on the changing pattern of foot contact with the floor. The analysis extends to include the motions of the foot and ankle and considers the unique balance between muscle action and walking function.

FOOT SUPPORT PATTERNS

Differences in timing of floor contact by the heel and forefoot create 3 periods of foot support. These normally occur in the following sequence: heel, foot flat (heel and forefoot), and forefoot (Figure 4-1). The last areas of the forefoot in contact with the ground are the first metatarsal and great toe as body weight rolls onto the other foot (see Figure 4-1). Equivalent Latin terminology is calcaneograde (heel only), plantigrade (flat), digigrade (forefoot), and unguligrade (toe tips).[4]

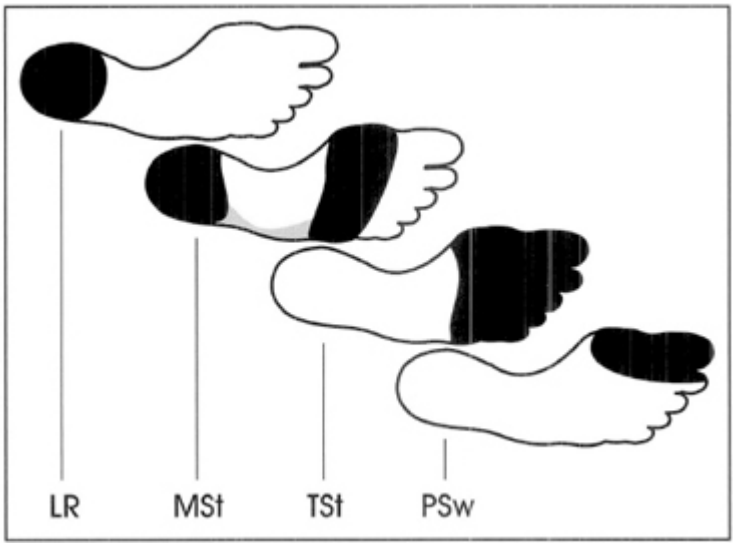

Figure 4-1. Sequence of foot support areas during stance. Heel only in loading response (LR). Foot flat in mid stance (MSt). Forefoot and toes in terminal stance (TSt). Medial forefoot in pre-swing (PSw). (Adapted from Barnett CH. The phases of human gait. *Lancet*. 1956;82(9/22):617-621.)

HEEL SUPPORT (CALCANEOGRADE)

The stance period normally is initiated by just the heel contacting the floor. Rapidity of this action has led to the term *heel strike*. After contacting the floor, the heel continues as the sole source of support for the first 6% to 12% of the GC.[4,21,27,51-53] Consistency among studies has followed the development of more responsive equipment. In contrast, the early pedographs indicated a heel support time to be 15% GC.[2] Timing also is influenced by the size and location of the sensors used, as contact is first made by the posterior margin and followed by a quick roll onto the center of the heel.[2,57]

FOOT-FLAT SUPPORT (PLANTIGRADE)

Forefoot contact terminates the heel-only support period and introduces a plantigrade or foot-flat posture. This persists for

approximately 20% of the GC. The manner of forefoot contact varies among individuals. Most commonly (71%), the fifth metatarsal head is the first part of the forefoot to touch the floor, resulting in at least a 0.1-second interval of H-5 support. The average is 10% GC.[27,51] A moderate number of subjects (22%) make contact with the total forefoot (H-5-1), and 8% of the subjects initiate forefoot support with the first metatarsal (H-1). Regardless of the means of starting forefoot contact, all segments from fifth to first rays soon are involved (H-5-1). Recent studies with a segmented force plate show quick progression from the lateral side of the foot (fifth and fourth metatarsal heads) to the medial side (first metatarsal head).[27] Less than 1% omit the foot-flat interval.

Forefoot Support (Digigrade)

Heel rise changes the mode of foot support to the forefoot. This occurs at the 31% point in the GC and persists until the end of stance. All the metatarsal heads are involved, although the simplified foot switch indicates only the fifth and first metatarsals. While the onset of heel rise is subtle and difficult to observe, footswitches provide a tool for documenting the timing.

Toe contact with the floor is quite variable. Scranton identified very early onset while Barnett found that toe contact followed isolated forefoot support by 10% of the stance period.[2,54] It is common for the toe to be the last segment to lift from the floor at the end of stance.[4,51] Bojsen calls this the “unguligrade” phase of gait.[4] Simultaneous first metatarsal and toe departure also is a normal finding.

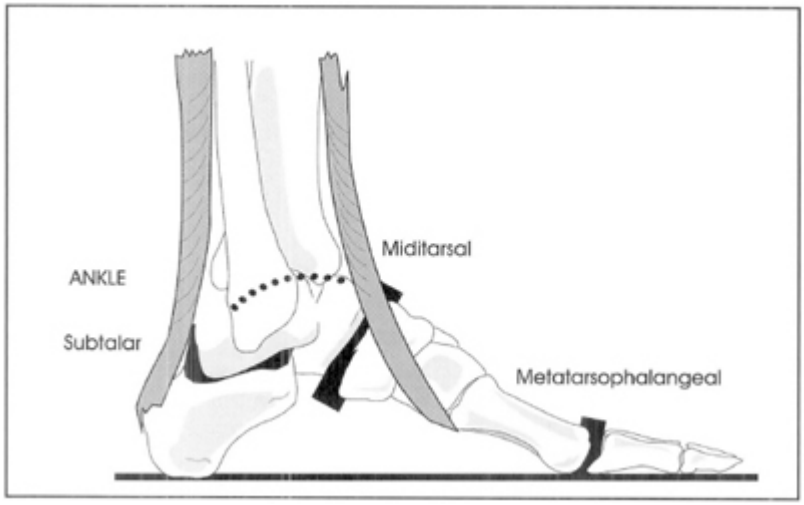

Figure 4-2. The joints in the ankle and foot with major functional significance during walking (black areas): ankle joint (tibiotalar), ST, MT, and MTP. The talus has no muscular attachments as the tendons from the lower leg instead attach to the more distal bones.

ANKLE AND FOOT ARTICULATIONS

The junction between the shank (tibia) and foot commonly is called the ankle, and the motions at this joint are flexion and extension. These concepts, however, are too limited. Movement between the tibia and foot involves a complex of 2 critical articulations created by the interposition of the talus between the tibia and the calcaneus (Figure 4-2). The talus serves as a weight-bearing link. Vertically, the weight-bearing column is divided into 2 joints (tibiotalar and subtalar [ST] or calcaneotalar). By custom, the term *ankle* refers just to the tibiotalar joint. While it has the largest ROM, the ankle cannot function independent of the ST articulation.

The insertion sites of the ankle muscles reinforce the skeletal synergy between the “ankle” (tibiotalar) and ST joints. No tendons insert on the talus. Instead, they all cross both articulations to attach to the calcaneus, navicular bone, and more distal sites (see Figure 4-2). Hence, even nonweight-bearing control of the ankle involves the foot joints.

Inman emphasized the interplay between the ST and tibiotalar articulations under the term *joints of the ankle*.[28] This concept is correct, but redefining a commonly used term has proven futile in other endeavors and is not attempted here. The original usage is too well preserved in prior writing and customs. The term *ankle* refers to the junction between the tibia and the talus (tibiotalar joint), while the inferior joint continues to be called *subtalar.* The actions of the other major joints within the foot (midtarsal [MT] and metatarsophalangeal [MTP], particularly) are included as part of the functional complex (see Figure 4-2).

ANKLE GAIT DYNAMICS

TERMINOLOGY

The clinical terms for motion in the sagittal plane are *flexion* and *extension*. While these terms also identify ankle motion,[1] the definition of the ankle's motion has varied. Some authorities follow the general rule that "flexion" is a motion that decreases the angle between the 2 bones (ie, flexion is an upward motion of the foot toward the tibia) and "extension" indicates relative straightening of the limb (foot moving away from the leg). Other clinicians consider upward motion of the foot to be extension as it is consistent with the direction the toes move.[1] Following the same logic, downward motion of the foot is called *flexion*.

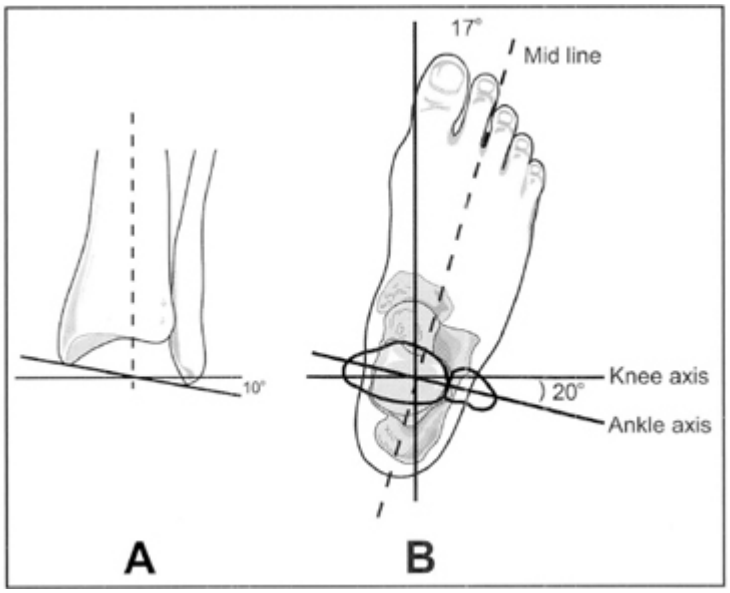

Figure 4-3. Axis of the tibiotalar joint in the (A) coronal plane and (B) transverse plane.

Neurologically, the first terminology is correct. Upward movement of the foot is part of the primitive flexor synergy (ie, it accompanies hip and knee flexion). Likewise, downward motion of the foot is part of the extensor synergy. This confusion has been resolved by substituting the terms *dorsiflexion* to signify upward travel of the foot and *plantar flexion* for the ankle's downward motion. This is the policy in this text.

While the dominant motions at the ankle joint are DF and PF, the axis of the tibiotalar joint is neither truly transverse nor horizontal. The axis is oblique to both the sagittal and coronal planes (Figure 4-3). The mean alignment of the ankle axis (which lies just below the 2 malleoli) is a 10° downward tilt in the coronal plane with the lateral end inferior to the horizontal (see Figure 4-3A). The lateral end is 20° posterior to the medial end in the transverse plane (see Figure 4-3B).[28,43] The result of this dual obliquity of the ankle axis is biplanar motion. PF is accompanied by mild inversion and, conversely, DF includes eversion.

Many of the numerical values for individual arcs of motion cited in this second edition may differ from much of the published reference data.[10,14,37,48,62] These changes represent technical progress in motion documentation. Three dimensional (3-D) gait analysis

systems have replaced all of the previous techniques, which were limited to two-dimensional representation of gait motion. The Rancho motion values cited in this text were recorded with a 3-D system.

The advantage provided by 3-D computer-driven, “multi-camera” motion analysis is the ability to define and track the longitudinal axes of the limb segments rather than relying on surface approximations (see Chapter 20 for details). At the foot, 3-D analysis avoids errors such as artificial equinus and varus implied by the conspicuous lateral border or the difference in forefoot and heel width. A disadvantage is the precision of computer calculations. Mean values can be extended to any decimal point fraction, yet physical measurement of joint motion is questionably accurate even in 1° increments due to skin mobility and parallax caused by the distance between a surface marker and the center of motion. Clinical experience in correlating measured motion with function strongly implies that 5° increments are more realistic than single integers or decimal fractions. Consistent with this philosophy, the characteristic joint positions for each phase have been rounded to the nearest 5° value in the text of this book. The graphs of joint motion reflect the continuous nature of transitions across phases and the more precise values at each point within the GC. Appendix A contains joint position data (mean and standard deviation) for each percent of the GC for those seeking greater detail.

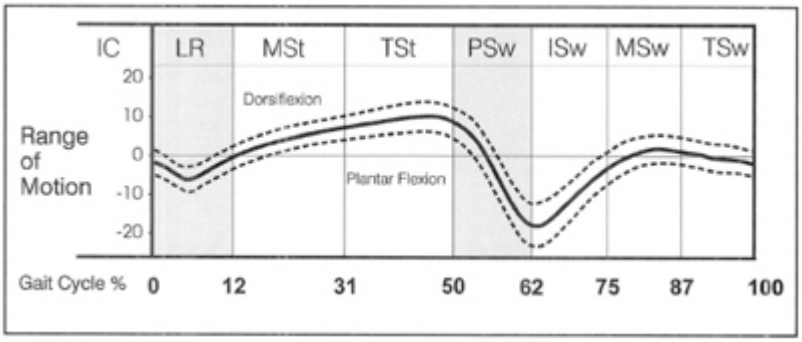

Figure 4-4. Ankle motion: Normal range during a stride. Black line = mean, dotted line = one standard deviation. Vertical bars designate the gait phase divisions.

Arcs of Motion

During each GC, the ankle travels through 4 arcs of motion (Figure 4-4). They alternate between PF and DF.[10,34,48,58,64] The first 3 arcs of motion occur in stance (PF, DF, PF). Although none of these motions are large, they are critical for progression and shock absorption. The fourth arc (DF) occurs in swing and contributes to foot clearance. The entire range of ankle motion used during each stride averages 25°.[8,34,48]

The ankle begins its first arc of motion as a reaction to the abrupt impact of initial floor contact. With the ankle at neutral DF and the limb reaching forward, initial floor contact is by the heel. This places the ground reaction vector posterior to the ankle. The sudden drop of body weight onto the heel initiates a rapid arc of ankle PF. Approximately half of the total PF arc that occurs during WA is accomplished during the first 2% of the GC. This amount of foot drop does not cause the forefoot to contact the floor, but the quick motion provides shock absorption and briefly decelerates tibial advancement by interrupting the rate of floor impact. The heel remains the site of foot support.

The PF initiated by heel contact continues through the first half of loading response to peak at 5° and then immediately reverses toward DF. By the end of the phase, the ankle has regained neutral DF. The ankle motion is masked by the conspicuous downward roll of the foot on the heel rocker.

During mid stance, the ankle gains 5° DF as the tibia advances over a stationary foot. Both the heel and forefoot are in contact with the floor. This also is the first phase of SLS. By the end of the phase, the body vector has advanced to the forefoot and the heel begins to rise.

In terminal stance, the ankle continues to dorsiflex slowly, reaching a peak of 10° at 45% GC. It stays in this position throughout the final 5% of terminal stance. During this same period, the heel rises 3.5 cm.[47] Thus, the source of tibial advancement in terminal stance is greater from heel rise than ankle DF. A 15° arc of

DF occurs over a period of 3 gait phases (loading response, mid stance, and terminal stance).

Abrupt floor contact by the opposite foot signifies the onset of pre-swing and double limb stance. Rapid transfer of body weight to the forward limb follows. The corresponding reduction of weight on the trailing limb allows the ankle to planter flex. The ankle moves from 10° DF to 15° PF throughout pre-swing as the foot rotates over the great toe. A 25° arc of motion is accomplished during a 12% GC interval. Also the limb begins to reduce its trailing posture.

Table 4-1

Physiological Cross-Sectional Areas and Moment Arms of Dorsiflexor Muscles

	Muscle Cross-Sectional Area (cm^2)	*Sagittal Plane Moment Arm (cm)*
TA	13.5	4.2
Extensor digitorum longus	4.6	4.0
Extensor hallucis longus	2.4	4.0
Peroneus tertius	1.0	Variable

At the onset of initial swing, rapid ankle DF is initiated to lift the foot for floor clearance. While neutral alignment is not quite achieved in initial swing (5° PF at 75% GC), a dragging toe is avoided as the swinging limb passes the contralateral stance limb.

Neutral alignment is typically attained in the early part of mid swing (79% GC) and then slightly exaggerated (2° DF). The visible posture is a vertical tibia with the foot horizontal.

As the limb reaches forward for full step length in terminal swing, the ankle appears neutral; however, it may experience a minor drop into PF (2° at 100% GC). This is generally interpreted as preparation for stance.

MUSCLE CONTROL

As the ankle joint moves in a single plane, all the controlling muscles function either as dorsiflexors or plantar flexors. Timing of ankle muscle action is very phasic. The plantar flexor muscles are consistently active in stance. The dorsiflexor muscle group, however, participates in the IC and loading response phases of stance to decelerate PF as well as provides foot control in swing.

The relative functional potential (strength) among the ankle muscles is proportional to their size (physiological cross-sectional area) and leverage. Both of these values have been well defined in the literature.[31,60] Knowing each muscle's potential is an important factor in understanding the pattern of ankle control that occurs during gait. While muscle leverage is modified by joint position, a useful relative scale can be developed by comparing the moments available with the ankle at neutral. The soleus, as the largest ankle muscle, was selected as the reference model to which others are related.

Dorsiflexors

The 4 muscles anterior to the ankle joint are the primary dorsiflexors. Three (the tibialis anterior [TA], extensor digitorum longus [EDL], and extensor hallucis longus [EHL]) are consistently present (Figure 4-5). A fourth potential dorsiflexor is the peroneus tertius (PT), but it is small and inconsistent (10% to 20% absence),[33] varies in its insertion along the metatarsal shaft, and blends with the lateral margin of the EDL. While the tendons of PT and EDL are distinct, there are no surface landmarks to differentiate their muscle bellies. This prevents the use of surface EMG to isolate their actions. Thus, routine recording of the PT is not done unless by fine wire EMG.

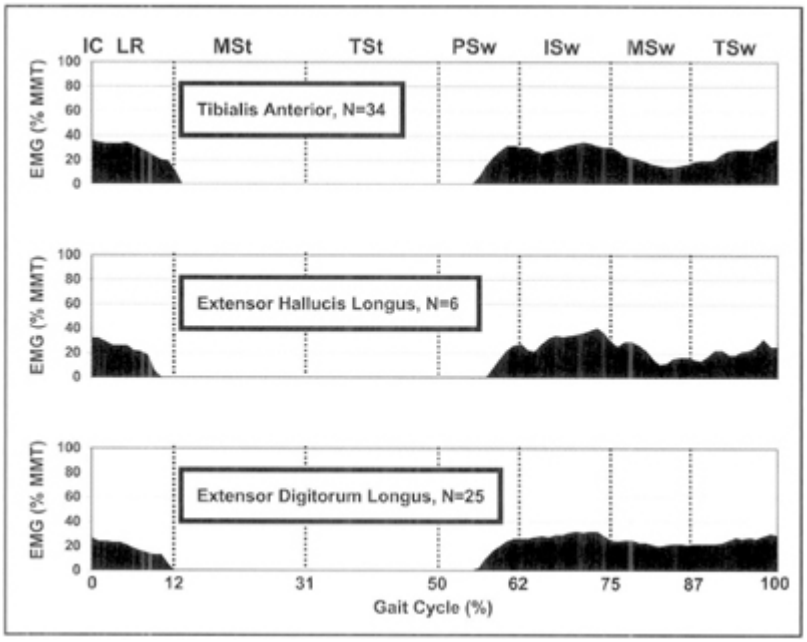

Figure 4-5. Ankle dorsiflexor muscles. Normal mean intensity and timing during free walking (quantified EMG). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. The dark shading indicates the activity pattern of the majority of subjects. Vertical bars designate the gait phase divisions. N = samples included in data.

The DF potential of the individual muscles is far from equal (Table 4-1). Each has a lever arm of similar length, but size differs markedly. The TA has the largest cross-section, while the EDL is only one-third as large and the EHL is half the size of the common extensors. The combined muscle mass of the PT and EDL is approximately 40% of the TA and very visible.

The typical pattern of dorsiflexor muscle action is biphasic, with initial swing and loading response containing the intervals of peak intensity (see Figure 4-5). The timing of muscle activity is similar for the 3 primary dorsiflexors. The onset of activity starts in pre-swing, and cessation is toward the end of loading response. The intensity of their peak EMGs is also similar (35% MMT); however, timing of peak effort differs. The anterior tibialis is most intense at IC, while the peak activity of the toe extensors is at the end of initial swing.

All of the pretibial muscles are strongly active at IC and continue this intensity until the end of loading response is near. This muscle

action is initially eccentric, opposing the rotational demand of the forefoot to “slap” the ground, which was created by the heel lever. Concentric activity follows in the second half of loading response as the tibia is drawn forward by the pretibials.

The second period of peak activity is during initial swing. The intensity of anterior tibialis activity rises rapidly, reaching 34% MMT. Then during mid swing, muscle action commonly diminishes (14% MMT). In terminal swing, the intensity gradually rises again to prepare for the challenging impact of IC (37% MMT). Relative intensity of the EHL and EDL peaks during initial swing to ensure clearance of the ground by the toes. Pretibial muscle action is concentric during initial swing and isometric in the latter 2 phases.

Table 4-2

Physiological Cross-Sectional Areas and Moment Arms of Plantar Flexor Muscles

	Muscle Cross-Sectional Area (cm^2)	*Sagittal Plane Moment Arm (cm)*
Triceps Surae		
Soleus	84.1	5.2
Gastrocnemius	57.3	5.2
Perimalleolar		
TP	16.9	0.5
Peroneus longus	13.9	1.0
FHL	13.4	1.0
FDL	5.5	2.0
Peroneus brevis	6.7	0.7

Plantar Flexors

Seven muscles pass posterior to the ankle and thus, are aligned to serve as plantar flexors (Table 4-2 and Figure 4-6). Their actual capacity, however, varies markedly. The soleus and gastrocnemius, which insert on the posterior superior area of the calcaneus, produce 93% of the plantar flexor torque. In contrast, the 5 perimalleolar muscles generate only 7% of the plantar torque.[22] This means there

are 2 distinct functional groups of plantar flexors: the triceps surae and the perimalleolar muscles.

Triceps Surae

The soleus and 2 heads of the gastrocnemius (medial and lateral) are the primary ankle plantar flexors. Their combined size (cross-section) is 73% of the total posterior muscle mass (see Table 4-2). In addition, they have a moment arm that is approximately 5 times larger than those of the perimalleolar muscles (Figure 4-7). The soleus is the largest muscle in the calf and the gastrocnemius is approximately 68% the size of the soleus.

While the triceps surae anatomical function is well defined, their contribution during walking is still in debate. The first problem is the clinical measure of plantar flexor muscle strength. Manual testing by resistance against the forefoot generates only 19% of the EMG recorded by the maximum isometric effort against a stationary dynamometer.[46] The EMG of a maximum heel rise, however, is equivalent to the peak plantar flexor muscle action recorded in late terminal stance. Hence, the normalizing factor for soleus or gastrocnemius EMG is percent maximum heel rise (% MHR). This intensity is 60% of the value recorded during a maximal isometric test using a dynamometer.[45]

The second issue is the function of the plantar flexors during the late phases of stance. The visual appearance of a trailing limb posture and the heel rise suggests that the soleus and gastrocnemius muscles propel the body forward by generating push-off. But the anatomical location of the gastrocnemius and soleus muscles on the posterior surface of the tibia contradicts this assumption. Their activation would plantar flex the foot, yet throughout terminal stance the ankle is slowly dorsiflexing. Recent research has shown that the plantar flexor pull of the soleus and gastrocnemius “locks” the ankle so the limb and foot rotate on the forefoot rocker. The segment between the ankle and metatarsal heads is a lever that supports body mass as it follows the advancing limb.

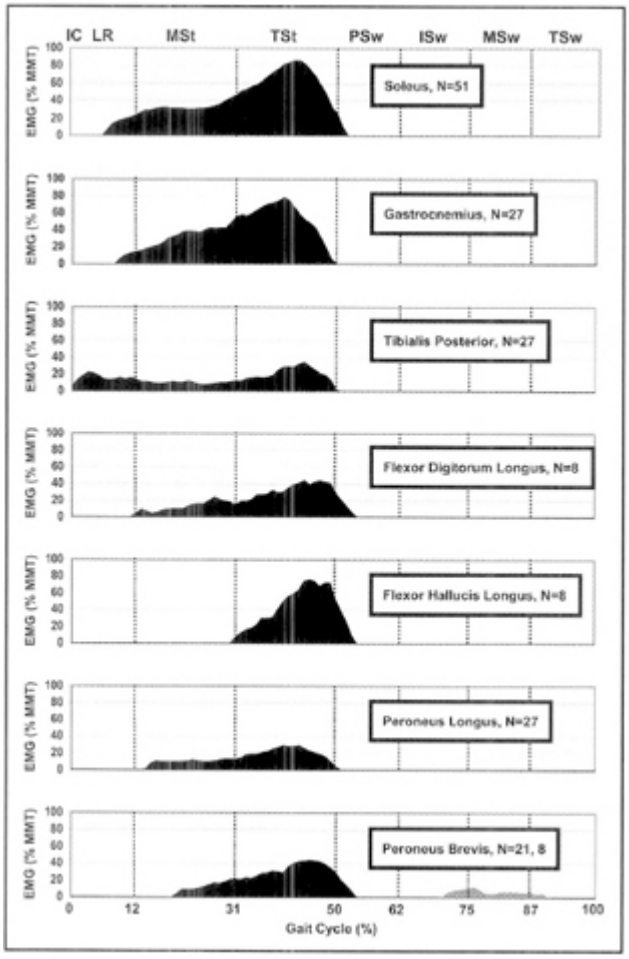

Figure 4-6. Ankle plantar flexor muscles including the triceps surae (soleus, gastrocnemius) and perimalleolar (TP, FDL, FHL, peroneus longus, peroneus brevis). Normal mean intensity and timing during free walking (quantified EMG). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in the data (dominant pattern, less frequent pattern if present).

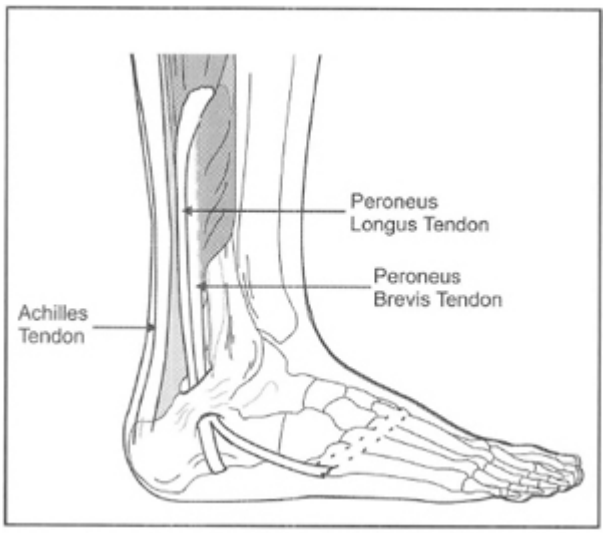

Figure 4-7. Lateral view of the ankle showing the relative alignment of the plantar flexor tendons to the ankle joint. Note the marked difference in lever length between the peroneals (which tightly hug the lateral malleolus) and the triceps surae (which insert on the posterior aspect of the calcaneus).

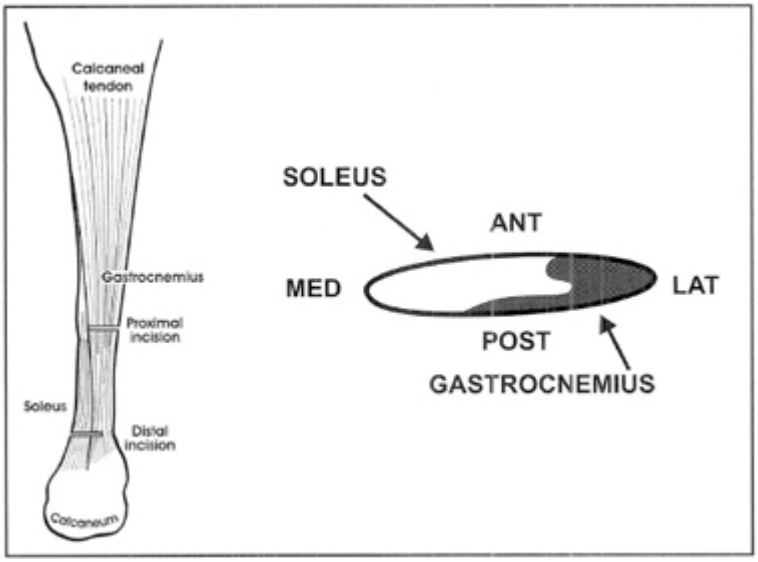

Figure 4-8. Insertion pattern of the tendo-Achilles. The soleus forms the larger portion of the tendon and inserts predominately posterior and medial, making it a strong invertor of the ST joint. The insertion site of the gastrocnemius is primarily lateral, contributing to its function as an evertor. (Adapted from Cummins EJ, Anson BJ, Carr BW, Wright RR. The structure of the calcaneal tendon (of Achilles) in

relation to orthopedic surgery. *Surg Gynecol Obstet.* 1955;99:107-116.)

Triceps Surae Function

The gastrocnemius and soleus muscles differ functionally due to their anatomical relationships to the ST joint and knee. As the gastrocnemius and soleus merge to form the Achilles tendon, both groups of fibers rotate transversely. The majority of the fibers of the tendo-Achilles are provided by the soleus, which inserts predominately medially, while the gastrocnemius inserts primarily laterally (Figure 4-8).[13] Thus the soleus has a strong inversion capability while the gastrocnemius acts as an evertor.

Anatomical differences at the knee also contribute to functional variations between the gastrocnemius and soleus. While both muscles contribute to the Achilles tendon, the gastrocnemius spans the knee in addition to crossing the ankle. This difference in muscle origin (distal femur for the gastrocnemius and proximal tibia and fibula for the soleus) modifies the function that is identified by their EMG patterns.

Soleus muscle action follows 2 intensity patterns. After a quick start during the latter half of the loading response (7% GC), the soleus rises quickly to 30% MHR and maintains this level of effort throughout mid stance (see Figure 4-6). With the approach of terminal stance (31% GC), its muscular effort rises rapidly, reaching an amplitude of 86% MHR during the latter half of the phase (43% GC). Then the intensity of soleus action declines with similar speed, dropping to 30% MHR by the onset of double stance and ceasing early in pre-swing (52% GC).

The EMG of the gastrocnemius displays a more consistent rise in EMG. While the onset of gastrocnemius action (9% GC) quickly follows the soleus, its intensity rises continuously throughout SLS to a peak of 78% MHR in the middle of terminal stance (40% GC). This is followed by a decline that is 3 times as fast as the rise to cease by the onset of pre-swing. A brief contraction of the gastrocnemius in mid swing is a common finding, but the reason is not clear.

Other gait characteristics that increase calf muscle action are a lengthened stride[24] and greater speed.[6] Both qualities were studied during treadmill walking. Hof et al found a linear relationship between stride length and peak calf muscle action by surface EMG.[24] In Hof et al's study, gait velocity was not a statistically significant factor, although there was a strong trend. When step rate was the means of increasing gait velocity, no relationship between calf muscle action and speed of walking was found.[24]

Perimalleolar Muscles

The 5 smaller muscles wrap closely around the medial and lateral malleoli as their tendons turn from their vertical alignment along the tibial shaft to extend horizontally along the plantar side of the foot. The flexor hallucis longus (FHL) gains a longer plantar moment arm as it follows the more posterior margin of the tibial articular surface. Control of the joints within the foot is the function of the perimalleolar muscles. At the ankle, their only effect is a low level of stability from the compression created by the tendons' abrupt change in alignment, but no significant motion.

Perimalleolar Function

The other 5 muscles crossing the ankle posteriorly have low plantar flexor capability because they are aligned for a different primary role during walking (ie, to control the ST joint and other articulations within the foot). In the process of providing their basic functions, these muscles also create a force at the ankle that should be considered (see Figure 4-6).

The tibialis posterior (TP) becomes active shortly after IC (0% GC) and continues through single stance.[58] This is promptly followed by the flexor digitorum longus (FDL) (13% GC) and, finally, the FHL (31% GC). Contralateral foot contact (50% GC) is the signal for the TP to relax while the toe flexors (FDL, FHL) continue briefly into pre-swing (54% GC). Sutherland found that both toe flexor muscles may show much earlier action, and there may be prolonged action of the FHL and TP.[58]

Peroneal muscle action starts at the 15% point in the GC.[9,20,58] The timing and relative intensity of the peroneus brevis and peroneus longus are very similar.[25,26,32,49,50] Both muscles tend to relax in early pre-swing (51% to 55% GC).

Forces

The functional demands placed on the ankle joint during the 3 arcs of motion occurring during the stance phase follow the load imposed on the limb, the alignment of the body vector, and the speed of motion. In swing, primarily the weight of the foot and speed of motion influence ankle forces.

Throughout stance, the base of the body vector (COP) advances along the length of the foot from the heel to the MTP joint and proximal phalanges.[35,44] This creates 2 alignments of the body vector in relation to the ankle joint axis.

The weight line (COP) is centered in the heel at IC. This places the vector behind the ankle, and a low amplitude dorsiflexor moment is required early in the phase to control foot lowering (peak of 0.18 N•m/kg•m at 4% GC; Figure 4-9). The rapid response of the ankle dorsiflexor muscles to eccentrically control foot lowering generates an immediate peak of absorptive power (0.15 W/kg•m at 3% GC). As limb loading continues, the COP advances rapidly. By the end of loading response (12% GC), the vector passes anterior to the ankle joint and the ankle dorsiflexor moment is reduced to zero. Low amplitude power generation during this period reflects concentric control of the pretibials as they function to draw the tibia forward.

Transition to SLS is accompanied by development of an increasing plantar flexor moment as the COP progressively moves ahead of the ankle joint. This continues at virtually the same rate until the end of terminal stance is approached, just before the other foot strikes, at which time the plantar flexor moment peaks (1.40 N•m/kg•m at 47% GC). This high internal moment limits ankle DF to 10°, thereby preserving the height of the center of mass and the location of the vector over the metatarsal heads. Power absorption predominates until the final half of terminal stance (peak 0.54

W/kg•m at 40% GC), reflecting the eccentric control provided by the plantar flexors throughout the majority of SLS.

During pre-swing, the rapid unloading of the trailing limb that follows floor contact by the other foot releases tension in the gastrocnemius and soleus musculotendinous unit, which had been pulled tight by terminal stance progression. This generates a strong burst of positive power (3.7 W/kg•m at 54% GC) through elastic recoil of the stretched tendon and contributes to rapid PF. This event is commonly called *push-off*. The force is sufficient to initiate forward swing of the limb. Many investigators consider this the prime propulsive force advancing the limb into swing.

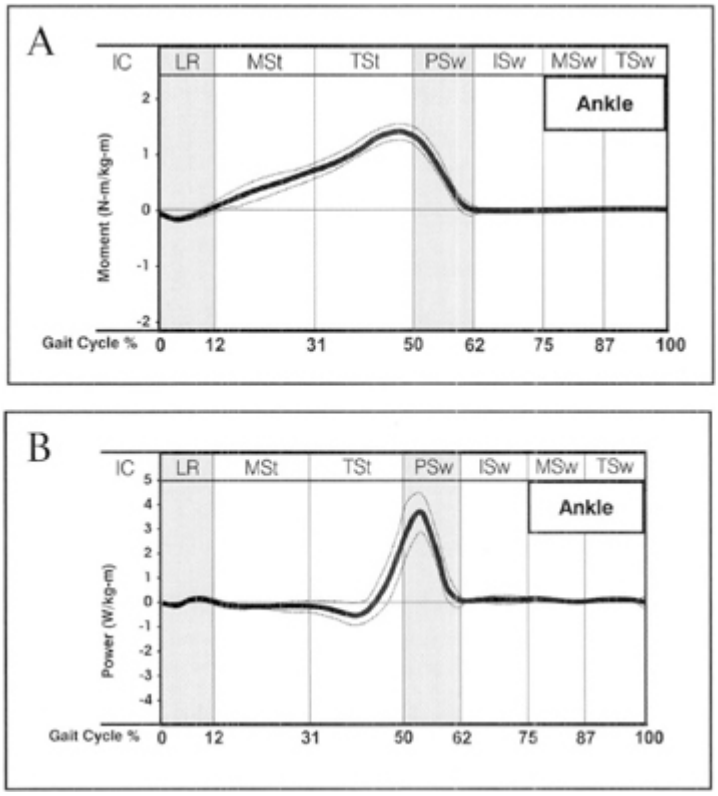

Figure 4-9. Ankle forces: (A) The brief dorsiflexor moment (-) at the onset of stance is followed by a plantar flexor moment (+) that peaks at the end of terminal stance and then progressively declines through the remainder of stance. A very low-level dorsiflexor moment is present during swing. (B) Ankle joint power demonstrating periods of power absorption (-) and generation (+).

At the onset of swing, a small dorsiflexor moment is generated (0.03 N•m/kg•m at 62% GC). This lifts the foot for clearance during swing.

FOOT GAIT DYNAMICS

TERMINOLOGY AND ARCS OF MOTION

There are 3 articulations within the foot that have measurable arcs of motion during walking. These are the ST, MT, and the group of 5 MTP joints between the forefoot and toes. The other tarsal and metatarsal articulations are tightly bound together by dense ligaments and display only a jog of motion when manipulated manually. Yet this minimal flexibility provides shock absorption and adaptation to irregular surfaces (see Figure 4-2).

Subtalar Joint

The ST joint is the junction of the talus and calcaneus. This places it within the vertical weight-bearing column between the heel and tibia. ST action adds coronal and transverse plane mobility to the sagittal plane function available at the ankle.

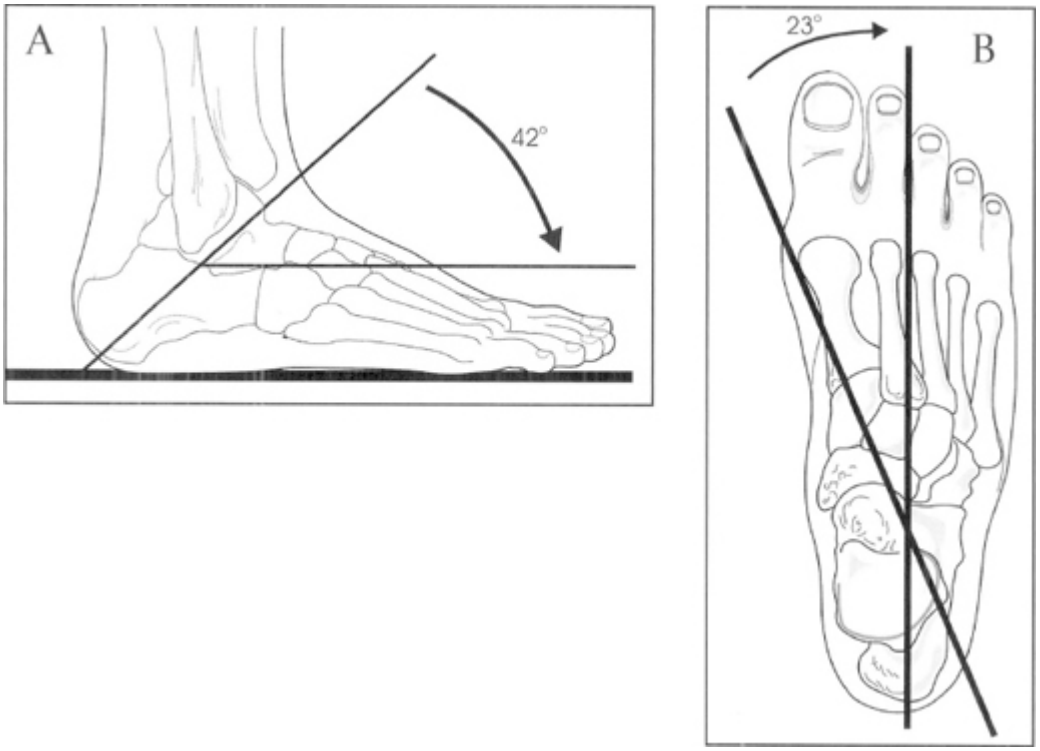

Figure 4-10. ST joint axis projected onto the (A) sagittal plane and (B) transverse plane. (Adapted from Inman VT. *The Joints of the Ankle*. Baltimore, MD: Wilkins & Wilkins Company; 1976.)

The ST joint has a single, obliquely oriented axis that allows the foot to tilt medially (inversion) and laterally (eversion). The mean alignment of the ST axis deviates 42° vertical and 23° medial from the midline of the foot (Figure 4-10). The standard deviations of 9° vertical and 11° medial indicate that the variation among subjects is large.[28] While ST motion occurs in both stance and swing, the stance phase motions are more significant as they influence the weight-bearing alignment of the whole limb.

IC occurs with the ST joint in neutral (Figure 4-11A). The lateral offset of the calcaneus relative to the weight-bearing axis of the tibia results in the calcaneus everting 5° as the limb is loaded (Figure 4-11B).[11,64] During mid stance and early terminal stance, this posture remains relatively unchanged. In late terminal stance, however, there is a progressive reduction of eversion to approximately 2° of eversion (ie, relative inversion) by the end of SLS (Figure 4-11C). During pre-swing, the ST joint achieves a neutral position and maintains this alignment through the remainder of swing.

Midtarsal Joint

The MT (or transverse tarsal) joint is the junction of the hind and forefoot. It is formed by 2 articulations: talonavicular (TN) and calcaneocuboid (CC). MT motion contributes to the shock absorption of forefoot contact.

MT mobility is customarily just observed. Recently, the use of a small field and 3-D measurement identified 5° DF. A decrease in medial arch height (ie, DF) follows forefoot contact at the onset of mid stance and SLS. Restoration of the arch is observed with heel rise, implying the MT DF has been reversed.

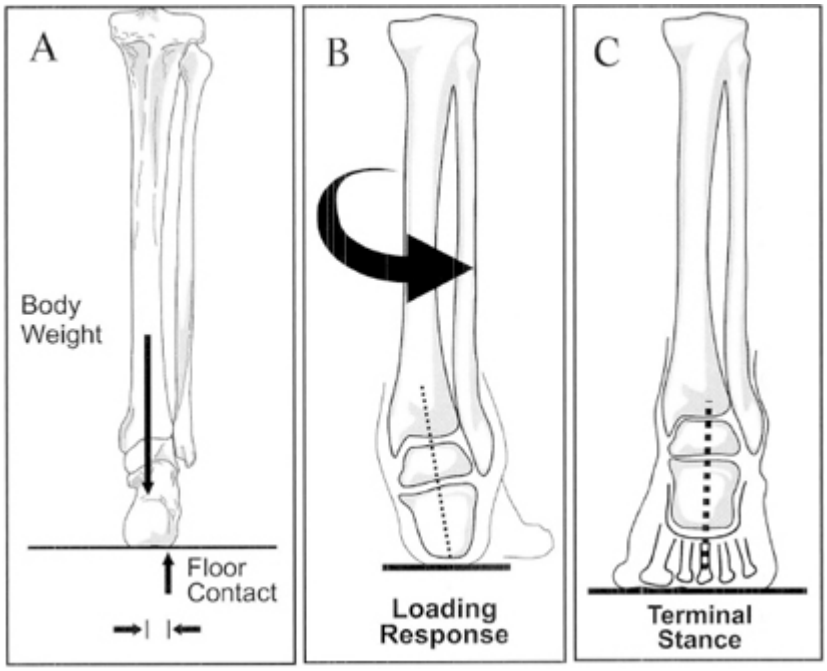

Figure 4-11. (A) IC: Vertical alignment of the tibia, talus, and calcaneus. (B) Loading response: IC by the lateral area of the calcaneus in combination with the medial projection of body weight everts the calcaneus, reduces support for the talus, and causes internal rotation of the tibia. (C) Terminal stance: As the heel rises, eversion of the calcaneus reduces toward neutral.

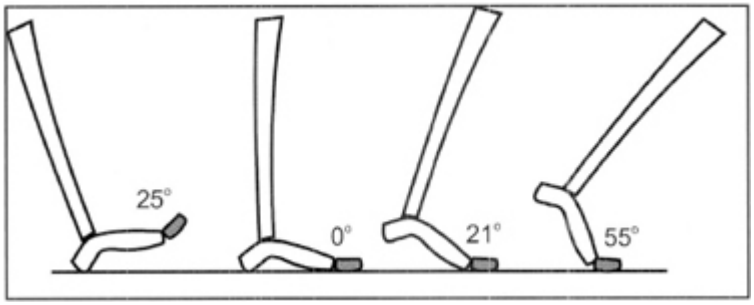

Figure 4-12. MTP joint motion during stance. Shaded area represents the toes.

Metatarsophalangeal Joint

The MTP joints form the “toe break” that allows the foot to roll over the metatarsal heads, while the toes provide variability in the shape of the toe rocker. The 5 metatarsal heads provide a broad area of support across the forefoot. In addition, the proximal phalanges allow an adjustable lengthening of the forefoot for progressional stability as needed.

At IC, the MTP joints are in 25° DF with the toes up (Figure 4-12). The toes drop toward neutral alignment following forefoot contact at the end of loading response and maintain this position throughout mid stance. With heel rise in terminal stance, the MTP joints dorsiflex (extend) 21°.[4] The toes remain in contact with the floor during this motion and the metatarsal shafts angle upward as the hind foot is lifted. This motion continually increases throughout pre-swing to a final position of 55° extension just before toe-off.

Lifting the foot for swing allows the toes to drop toward the line of the metatarsal shafts. Slight DF is maintained in mid swing. Then the MTP joints increase their DF (toes up) in preparation for IC.

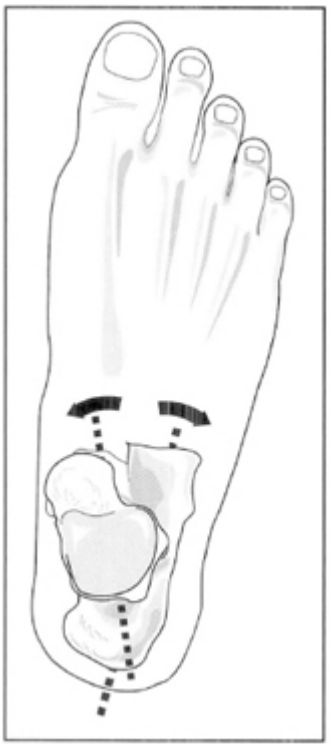

Figure 4-13. Internal rotation of the talus as the calcaneus everts during loading response.

Synergies of the Ankle and Foot

Triplanar mobility of the ST joint impacts function of both the ankle and the midfoot. Shock absorption and stability are modified as the ST joint moves in and out of eversion.

Ankle-Subtalar Synergy

A significant function of ST eversion is to prevent excessive strain at the ankle as the limb is loaded. The body moves from behind to ahead of the supporting limb throughout stance. This creates a rotational torque on the supporting joints. The externally rotated alignment of the ankle axis (20°) is not compatible with the longitudinal path of the body's progression in loading response.

ST joint eversion is initiated passively by the lateral offset of the calcaneus relative to the vertical axis of the tibia (see Figure 4-11A).[51] Abrupt loading of the heel at IC causes the calcaneus to tilt as the ST joint everts (see Figure 4-11B). Anterior support for the

head of the talus (sustentaculum tali) is reduced, causing internal rotation of the talus (Figure 4-13). The closely fitted rectangular-shaped tibia-fibula ankle mortise causes the tibia to follow the talus.[36] Hence, the load imposed on the talus at IC causes eversion at the ST joint and subsequent realignment of the ankle joint for compatible progression during loading response and early mid stance.[44]

As the body moves ahead of the ankle during mid and terminal stance, ST motion is reversed toward inversion (see Figure 4-11C). This lifts the head of the talus and restores the externally rotated alignment of the ankle axis. Again, strain from body weight torque is avoided at the snugly contoured, single-axis ankle joint. Rotational moments measured at the floor are small but functionally significant as intra-articular shear is poorly tolerated.

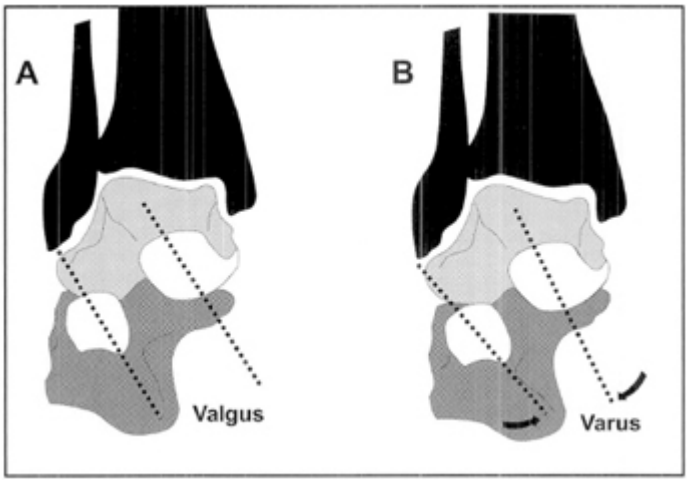

Figure 4-14. MT joint reactions. (A) TN and CC joint axes (dotted lines) become parallel with ST eversion. (B) As eversion is reduced (relative inversion), the axes converge. (Adapted from Mann RA, Mann RJ. Biomechanics of the foot. In: Goldberg B, Hsu J, eds. *Atlas of Orthoses and Assistive Devices.* 3rd ed. St. Louis, MO: Mosby; 1997:135-152.)

Subtalar-Midtarsal Synergy

The ST joint controls MT mobility by altering the relative alignment of the TN and CC joints.[43] With ST joint eversion, the axes of the TN and CC joints are parallel (Figure 4-14A). This frees the MT joint to

serve as a shock-absorbing region as the forefoot is loaded in mid stance. Then to meet the demand for mid foot stability, the ST joint reverses its everted motion toward inversion (Figure 4-14B) and the MT joint is locked.

Muscle Control

The sequence of muscular control within the foot progresses from the hind foot to the forefoot and then the toes. The same 10 muscles initially classified as ankle dorsiflexors and plantar flexors also control ST inversion and eversion. For their action within the foot, these muscles are classified according to their relationship to the ST joint axis. The 5 muscles that pass medial to the axis provide inversion, while the 5 on the lateral side create eversion (the PT is excluded). Two factors determine the timing of muscle activation: their primary sagittal function (ankle and toes) and the demands of the ST joint. The small plantar intrinsic muscles both originate and terminate within the foot and are activated by the location of the GRF.

Invertor Muscles

The muscles that cross the ST joint medially differ considerably in their inversion lever lengths (Figure 4-15). From the longest to the shortest, the sequence is TP, TA, FDL, FHL, and soleus. All but the TA lie posterior to the ankle. Their onset of activity during stance occurs in a serial fashion and continues relative to their need by more distal joints.

TP activity has demonstrated considerable variability in the Rancho data, but the dominant pattern is early ST control (see Figure 4-6). Following the onset with initial heel contact (0% GC),[58] there are 2 periods of peak intensity. The first is a 22% MMT effort at the beginning of loading response (3% GC). The middle of terminal stance (44% GC) is the time of the second TP rise (34% MMT). Contralateral foot contact (50% GC) is the signal for the TP to cease activity. Close and Todd[12] and Sutherland[58] reported good TP

activity, but Gray and Basmajian[20] and Basmajian and Stecko[3] did not.

The TA, active in swing, markedly increases its intensity following IC to 36% MMT (see Figure 4-5). Then there is a rapid decline and relaxation by the initiation of mid stance (13% GC).[51]

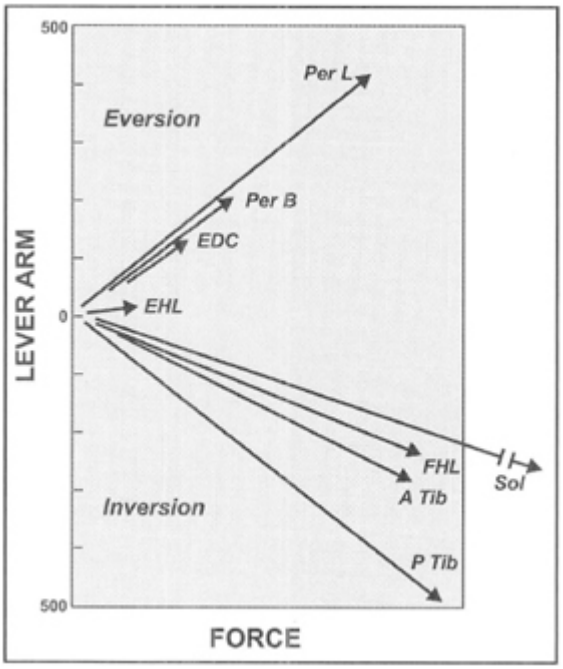

Figure 4-15. Relative inversion and eversion torque capability of the ST muscles. Horizontal axis = force. Vertical axis = lever arm length. Diagonal arrow length = relative torque.

Table 4-3

Physiological Cross-Sectional Areas and Moment Arms of Invertor Muscles

	Muscle Cross-Sectional Area (cm^2)	*Coronal Plane Inversion Moment Arm (cm)*
Invertor and Dorsiflexor		
TA	13.5	2.6
Extensor hallucis longus	2.4	0
Invertor and Plantar Flexor		
TP	16.9	3.4
FDL	5.5	1.4
FHL	13.4	1.2
Soleus	84.1	1.1

Soleus muscle activity starts during the latter half of loading response (7% GC) in preparation for the demands of SLS (see Figure 4-6). The muscle demonstrates 2 levels of control. There is a sustained 30% effort during mid stance. This is followed by a progressive rise in terminal stance to 86% MHR. In pre-swing, soleus muscle action promptly declines. Only by its large cross-section does the soleus dominate (Table 4-3). Its inversion lever is short. Timing and intensity are dictated by the demands at the ankle.

Table 4-4

Physiological Cross-Sectional Areas and Moment Arms of Evertor Muscles

	Muscle Cross-Sectional Area (cm^2)	*Coronal Plane Eversion Moment Arm (cm)*
Evertor and Dorsiflexor		
Extensor digitorum longus	4.6	1.7
Peroneus tertius	1.0	2.0
Evertor and Plantar Flexor		
Peroneus longus	13.9	3.1
Peroneus brevis	6.7	2.6
Gastrocnemius	57.3	1.1

The FDL becomes active at the transition to mid stance (13% GC), while onset of FHL activity occurs later (31% GC) (see Figure 4-6). Both muscles progressively increase their intensity through terminal stance and then rapidly decline during the first half of pre-swing. Sutherland found that both toe flexor muscles may show much earlier action and there may be prolonged action of the FHL.[58]

Evertor Muscles

Five muscles lie on the lateral side of the ST joint axis. Two (the EDL and EHL) are anterior. Posterior to the ankle are the gastrocnemius, peroneus longus, and peroneus brevis. Eversion leverage is also determined by size and lever length (see Figure 4-15; Table 4-4).

The EDL and EHL, both active during swing, continue their activity into the latter half of loading response (see Figure 4-5). The EDL has the best eversion capability by leverage and cross-section among the smaller muscles. The EHL is visibly very obvious because of its bowstring tendon. Both its small size and alignment over the ST axis, however, minimize its effect on ST function.

The gastrocnemius, as the other large ankle plantar flexor muscle, dominates by size even though its leverage for eversion is small. Its action progressively increases after onset in late loading response (9% GC; see Figure 4-6). Peak action (78% MHR) is reached during the middle of terminal stance (40% GC), followed by rapid decline and cessation at the end of terminal stance (50% GC).

The peroneus longus and brevis begin their activity during mid stance (15% GC and 20% GC, respectively) and reach peak intensity during terminal stance (see Figure 4-6).[12,20,58] Cessation follows contralateral foot contact. Earlier investigators identified slightly different timing. The differences relate to improvements in technique gained over the 30-year interval.[9,20,25,26,32,49,50,58]

Muscle Synergies

The inverting muscles lessen the shock of floor impact by decelerating ST eversion. TA restraint of the ST joint to maintain the heel rocker is assisted by the early onset of the TP (0% GC). The TA exerts peak intensity with IC. The first peak effort for the TP occurs in early loading response (3% GC). By the time of peak eversion (5°, 20% GC), the TA has relaxed as its primary function as a dorsiflexor no longer is needed. At the same time, the soleus has reached a moderate level of action. The dominant functions of the TA and soleus relate to ankle control, but both muscles also have an inversion lever (see Table 4-3). The much larger size of the soleus compared to the TP (5x) makes it a significant inverter, even though the TP has a significantly longer inversion lever arm (see Figure 4-15 and Table 4-3). The variability in TP activity may relate to the wide range of ST joint mobility.

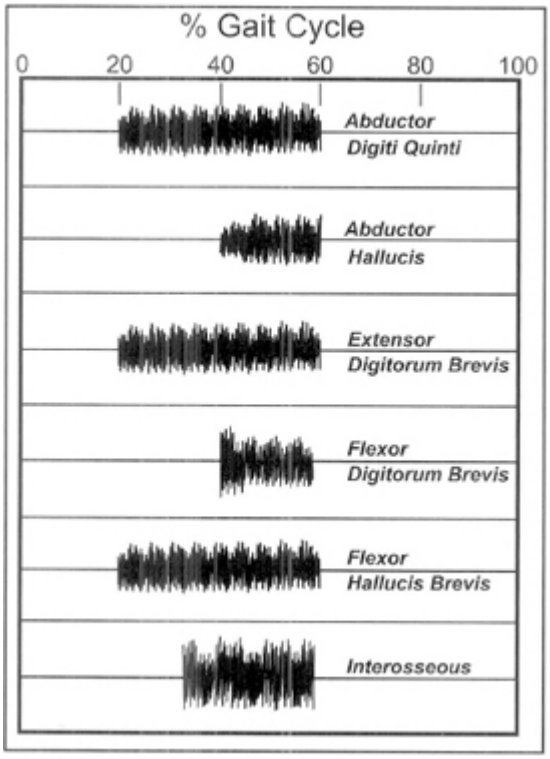

Figure 4-16. Intrinsic foot muscle action during stance. (Adapted from Mann R, Inman VT. *J Bone Joint Surg*. 1964;46A:469-481.)

In terminal stance, the 4 plantar-inverting muscles (soleus, TP, FDL, and FHL) markedly increase their intensity to provide the added stability needed for forefoot support. The deep intrinsic

muscle layer has the advantage of proximity, but the other muscle groups provide greater force due to their larger mass (see Table 4-3). Excessive inversion is avoided by the synergistic action of the 2 peroneal muscles. Their activity peaks as the heel rises from the ground (40% to 46% GC) and continues into pre-swing.

Dynamic support of the MT joint is provided by several longitudinal muscles. First are the FDL (onset 15% GC) and lateral plantar intrinsic (onset 20% GC) muscles, which precede any change in toe position. Subsequent action by the FHL (25% GC) provides additional support.

The intrinsic muscles of the foot differ in their times of onset; however, they show similar times of cessation (Figure 4-16).[42] Three begin their activity in early mid stance (20% GC). These are the abductor digiti quinti, extensor digitorum brevis, and flexor hallucis brevis. These are followed by the interossei in early terminal stance (35% GC). The last 2 muscles (abductor hallucis, flexor digitorum brevis) are activated later in terminal stance (40% GC) as heel rise becomes prominent. The pattern of loading appears to parallel the rate of loading of the forefoot. Activation of the intrinsic muscles increases with foot pronation.

The plantar fascia offers passive support to the mid foot and MTP joints in mid stance, terminal stance, and pre-swing. Extending from the calcaneus to the fascia about the base of the toes (proximal phalanges), the plantar fascia becomes taut as MTP DF serves as a windlass during walking (Figure 4-17). Its effect in routine walking is limited, however, as 50% shortening of the aponeurosis requires 30° of MTP DF. Only two-thirds of this range of DF is reached by the end of terminal stance when maximum stability is needed.[4] Planter fascia stability, however, would be of significance in more vigorous activities.[18] A force plate study showed that in maximum DF, the toe flexor force was twice that available by maximum voluntary contraction of the flexor muscles.

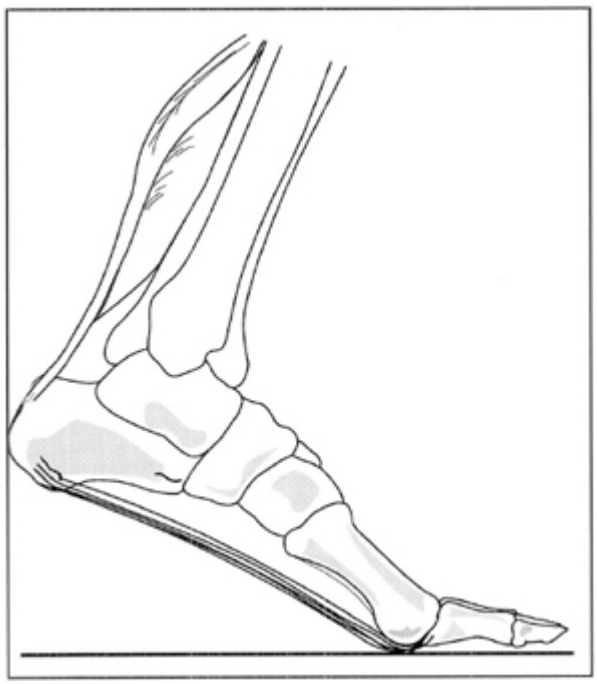

Figure 4-17. Plantar fascia of the foot (superficial to the intrinsic foot muscles in the arch). Fascia tensed by MTP joint DF.

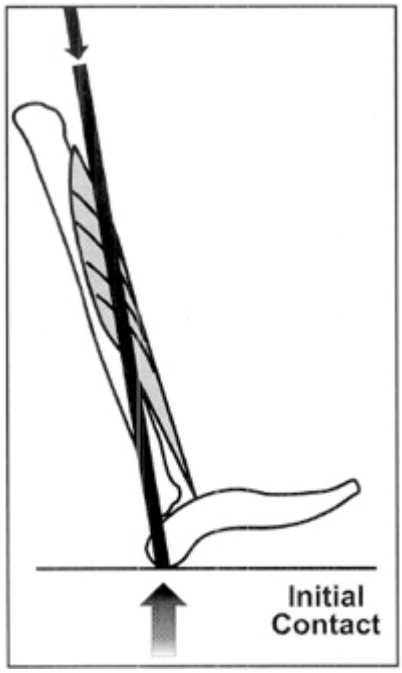

Figure 4-18. IC posture of the ankle. The vector is within the heel, controlled by the TA.

FUNCTIONAL INTERPRETATION OF THE ANKLE AND FOOT

The complex interactions of the ankle and foot are best appreciated when functional demand and response are coordinated. This approach is facilitated by integrating motion, muscle control, and the involved forces according to the GC phases.

INITIAL CONTACT (0% TO 2% GC)

Posture: Ankle and foot at neutral
Function: Initiate the heel rocker
Impact deceleration

Initial floor contact begins with the ankle and foot held in neutral by the pretibial muscles. The forward reach of the limb tilts the distal tibia upwards 15° and positions the heel as the lowest segment of the foot (Figure 4-18). The abrupt drop of body weight onto the heel generates an immediate but brief peak in the GRF pattern, called a heel strike transient (HST).[61] The intensity ranges between 50% to 125% body weight.[56] Further analysis documented the variability of the HST.[59] In 76% of the subjects tested (n = 75), the force was sharp and intense, lasting no more than 10 milliseconds (ms). Another 13% of the subjects displayed a smoother reaction of 20 ms duration, while the remaining 11% exhibited no HST.[59] A significant correlation was identified between the sharp transient and a delay in the time of quadriceps and anterior tibialis EMG activation. This relationship was not evident in the other subjects. These findings indicate that for most individuals, IC with the floor is a forceful event. The rapid transfer of body weight onto the forward limb, with the heel as the only area of support, drives the forefoot toward the floor. Immediately ankle PF and ST eversion are initiated to decelerate the impact of falling body weight.

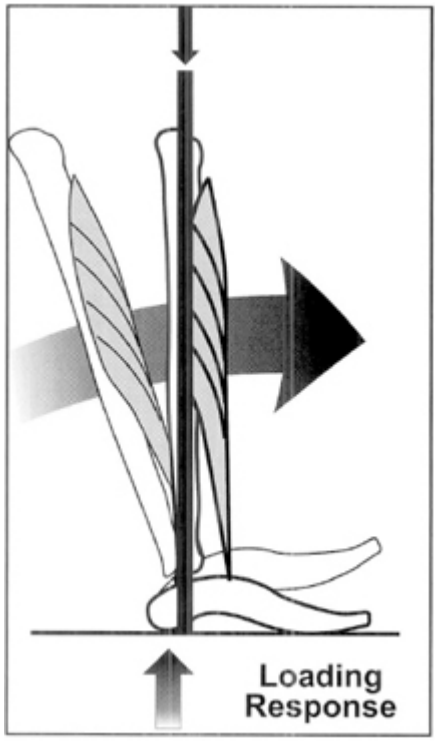

Figure 4-19. Loading response pattern of motion, muscle control, and vector alignment at the ankle. The pretibial muscles reverse the initial PF, preserving the heel rocker until the tibia advances to neutral. Arrow indicates the direction of motion.

LOADING RESPONSE (2% TO 12% GC)

Motion:	Ankle PF
	ST eversion
Function:	Heel rocker initiation of progression
	Realignment of the ankle axis

Both the ankle and ST joints respond to the impact of initial heel contact (Figure 4-19). At the ankle, the location of the body vector at the heel produces a posterior lever arm. In response to the rapid loading of body weight on the heel lever (70% body weight by 2% GC), the foot is promptly driven to the floor. Intense, eccentric activity of the pretibial muscles (TA and long toe extensors) generates an opposing dorsiflexor moment. The foot drops to 5° by 6% GC. In vivo studies of the TA suggest that tendon stretch may contribute to the 5° of DF that is later recovered by elastic recoil.[38-40] Two purposes

are served by this dynamic response: 1) the heel support period is extended, and 2) the tibia is drawn forward as the foot drops. Both actions contribute to limb progression. The combined effects of the controlled movement of the ankle into PF and active tibial advancement roll the body weight forward on the heel. Hence, the term *heel rocker*.

The 5° of PF motion that occurs during loading response actually reduces (rather than contributes to) the heel rocker effect so that the tibia will not advance too fast. If the ankle had remained in a 90° position, the tibia would accompany the foot through its rapid arc of motion during the first half of loading response. The loose coupling of the foot and tibia during WA reduces the rate of knee flexion.

Shock absorption is the second advantage of restrained ankle PF during the loading response. This, too, is facilitated by the pretibial muscle action. As a greater dorsiflexor moment is required to control the rate of ankle PF than was used to support the foot in swing, there is a lag in muscle effectiveness. The result is an initial arc of rapid ankle PF.[47] This augments the brief “free fall” period for body weight. The vertical height of the ankle is lowered by the rapid drop of the foot as it rolls freely on the rounded undersurface of the calcaneus (heel). Once the pretibial muscle force becomes sufficient, the downward motion of the foot is slowed, making forefoot contact a quiet event. In this manner, the heel rocker redirects some of the body’s downward force to the pretibial muscles as they restrain ankle motion. These actions absorb some of the shock that accompanies rapid limb loading.

Continuing progression of the body vector across the heel then quickly reverses the PF toward DF as the GRF advances. Continued contraction of the pretibial muscles preserves the heel rocker during the last half of loading response. By the time forefoot contact terminates loading response, the ankle is at neutral.

At the ST joint, the impact of abrupt loading is a fast arc of eversion. Lateral rotation of the calcaneus depresses support of the head of the talus, which causes internal rotation of the talus and the tightly fitted ankle mortise. Rotation of the ankle realigns the joint’s axis closer to the body’s longitudinal path of progression. Also,

eversion unlocks the MT joint. Forefoot contact is softened by yielding DF of the unlocked MT joint.

Both the ankle and ST actions interrupt the impact of abrupt limb loading. The pretibial and inverting muscles absorb some of the loading shock that accompanies rapid limb loading as they restrain the ankle and foot. Exact timing varies with the person's gait velocity.

Mid Stance (12% to 31% GC)

Motion:	First arc of single stance DF
	MT joint DF
Function:	Ankle rocker progression
	MT shock absorption
	Tripod foot support for stability

Floor contact by the heel and the first and fifth metatarsal heads provides a stable foot-flat posture. Progression of the limb is continued by DF of the tibia over the articular surface of the talus (Figure 4-20). This ankle rocker (also called the second rocker)[16,17] advances the body vector from behind to in front of the ankle joint axis, while the tibia moves from an initial 5° plantar flexed position to 5° DF. The heel and forefoot remain in contact with the floor.

The propelling force is assumed to be residual momentum from the push-off power generated while the opposite limb was in pre-swing.[63] Also, it may be a response to the forward swing of the other limb.

Eccentric soleus and gastrocnemius muscle action provides control for the ankle's simultaneous progression and stability (see Figure 4-20). Soleus activity is the dominant decelerating force (compared to the gastrocnemius) because it provides a direct tie between the tibia and calcaneus. Also, the soleus is the largest plantar flexor muscle. In contrast, the gastrocnemius has no direct tie to the tibia because it arises from the distal femur. Consequently, until body weight is anterior to the knee joint axis (late mid stance), the gastrocnemius also acts as a knee flexor as it lies posterior to the knee joint axis. This increases the demand on the quadriceps but that is not a problem for persons with normal strength and control.

Sutherland's data demonstrate a distinct delay in the onset of gastrocnemius activity.[58] The Rancho data also show a slight delay in gastrocnemius onset compared to that of the soleus.

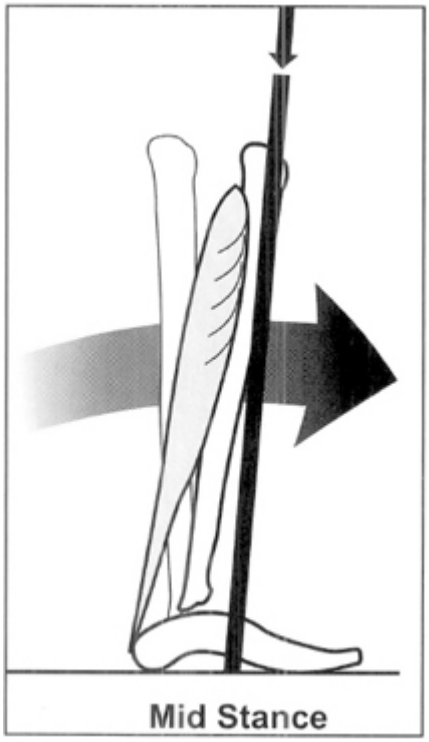

Figure 4-20. Mid stance pattern of motion, muscle control, and vector alignment at the ankle. Passive DF is restrained by the soleus and gastrocnemius muscle action. Arrow indicates the direction of motion during the ankle rocker.

The EMG of the soleus muscle maintains a steady intensity at 30% MHR through the middle of mid stance (see Figure 4-6). Shortly before the end of the phase, the soleus increases intensity. In contrast, the EMG pattern of the gastrocnemius is a slow continuous rise throughout mid stance. The combined eccentric action of the soleus and gastrocnemius muscles reduces the rate of tibial advancement to half of its former speed.

TP and soleus activity reverse ST eversion toward inversion (20% GC), enhancing stability of the MT joint (see Figure 4-6). Progression and stability are both served by the normal balance between passive mobility and muscular control.

TERMINAL STANCE (31% TO 50% GC)

Motion: Heel rise
Continued ankle DF
Reduced ST eversion to lock the MT joint
Function: Forefoot rocker for progression

Heel rise, as recorded using footswitches, signifies the onset of terminal stance (Figure 4-21). Motion toward ST inversion (end position of 2° eversion) has locked the MT joint, and the forefoot is now the sole support for body weight. The rounded contours of the small metatarsal heads and the base of the phalanges provide a forefoot rocker and allow the body vector to advance for continued progression.

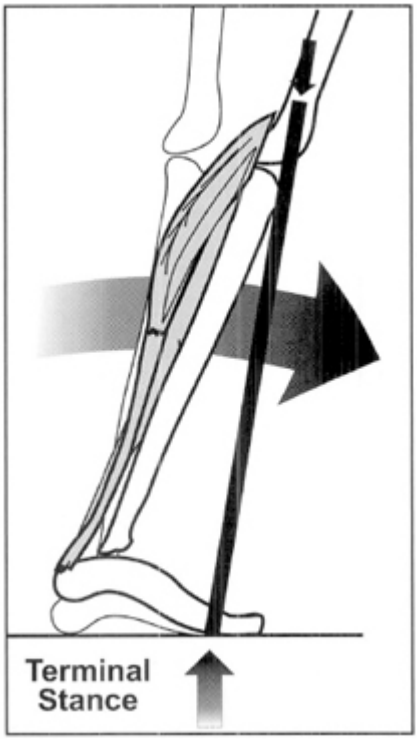

Figure 4-21. Terminal stance pattern of motion, muscle control, and vector alignment at the ankle. The vector at the metatarsal head initiates progression over the forefoot rocker (arrow). Strong soleus and gastrocnemius muscle action stabilizes the dorsiflexing ankle and allows heel rise.

The foot segment between the ankle (fulcrum for the limb) and the metatarsal heads (base for the COP) serves as a lever positioned to preserve leg height as the COM advances over the forefoot. At the onset of terminal stance (31% GC), the COM is at its highest point and the swing limb is anterior to the COM. Thus, the propelling force is falling body weight (ie, potential energy arising from gravity) and the momentum generated by forward swing of the opposite limb.

The function of the calf muscles is to preserve the forefoot rocker. As the tilt of the body vector increases, the plantar flexor moment rapidly increases. This presents a high demand on the soleus (86% MHR) and gastrocnemius (78% MHR) to provide the plantar flexor force necessary to control the tibia (see Figure 4-6).[55,58] Eccentric action of the plantar flexor muscles virtually locks the ankle joint by isometric action, while stretch of the Achilles tendon by the plantar flexor moment provides the “ankle” motion. The ankle experiences a 5° increase in DF to reach a final position of 10° DF. All other posterior muscles also contribute to the stability of the forefoot rocker. The peroneals and TP control the ST and MT joints and the long toe flexors stabilize the MTP joints.

Rotation of the foot around the forefoot rocker elevates the hind foot and maintains height of the COM. When ankle control is deficient (eg, with a weak calf), the COM and limb advance at the same site (ankle DF) and the COM is lowered.[55,58]

There is a serial decline in gastrocnemius and soleus muscle function during the last half of terminal stance (Figure 4-22). The relatively early decline in EMG intensity of the gastrocnemius (40% GC) and soleus (43% GC) suggests sensory feedback of imminent instability. Both the plantar flexor moment and GRF reach their peak intensities at 47% GC. These changes imply a major disruption of stance stability. It is assumed that the foot no longer is an adequate weight-bearing base.

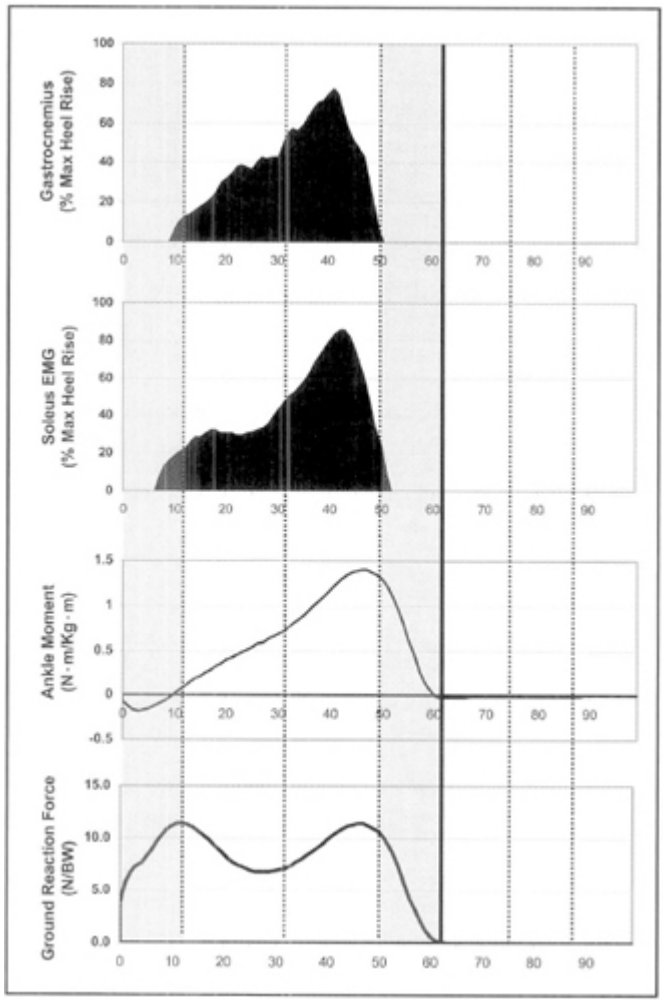

Figure 4-22. Peak activity of the gastrocnemius (40% GC) and soleus (43% GC) occurs just prior to the peak in the plantar flexor moment and GRF. All rapidly decline following the peak, implying that the need for forceful stabilization of the ankle and foot has passed.

During terminal stance, biomechanical studies identify deceleration rather than an increasing rate of tibial advancement.[19,55] A careful study relating the pattern of body advancement to the external vector demonstrated the plantar flexor muscles restrain the body's momentum rather than propelling it forward.[55] Direct shank accelerometry found a deceleration force in terminal stance about equal to that occurring in loading response.[19] These findings contradict Hof et al's conception of an elastic after-force catapulting the body forward.[24] In reality, the plantar flexor

muscles provide critical ankle stabilization, which allows both the foot and tibia to roll forward on the forefoot rocker. There are 2 significant effects: a reduction in the amount of fall by the body's COG and enhancement of progression.[55] Both relate to the increase in relative limb length gained from using the forefoot rather than the ankle as the fulcrum. Hence, *roll-off* (over the forefoot rocker) is the more appropriate term at this time. Push-off will occur in pre-swing.

Controlled mobility of the MTP joints is essential for optimum forward roll across the forefoot. As the heel rises with continued forward progression, body weight advances across the forefoot and DF increases at the MTP joints. Stance phase action of the toe flexors controls the shape and stability of the forefoot rocker. The total area can be expanded by inclusion of the toes (see Figure 4-21). This allows a greater forward roll if the MTP joints yield appropriately. In addition, the compressive force of the toe flexor muscles stabilizes the MTP joints. Supplementing the muscles is the passive compression by the plantar fascia, which has been tightened by MTP joint DF.[7]

A further stabilizing force that improves the forefoot support area is the action of the peroneus longus on the first metatarsal. By plantar flexing the first ray, the weight-bearing capacity of the medial side of the forefoot (ie, hallux) is improved.[30] This is particularly significant as body weight rolls toward the first ray in preparation for WA by the other limb. As a result, the location of the body vector is between the first and second MTP joint.

Freedom for the foot to roll across the rounded metatarsal surfaces depends on the presence of adequate passive mobility of the MTP joints and yielding (eccentric) control by the flexor muscles. The small leverage available to the long toe flexor muscles allows them to maintain the desired compressive force for MTP joint stability and still permit progressive DF. This enables the COP to move beyond the end of the metatarsal head, thereby adding to the length of the stride. Ground contact by the other foot ends terminal stance.

PRE-SWING (50% TO 62% GC)

Motion: Second arc of ankle PF

Functions: Propulsion

Initiation of knee flexion for swing

This is a very complex phase of gait. The actions of the ankle are related to progression rather than weight bearing; however, continued forefoot contact assists with balance. During this period of terminal double foot-to-floor contact, body weight is rapidly transferred from the trailing limb to the forward limb. The abrupt onset of the transfer is confirmed by the impact (50% to 125% body weight in 10 to 20 ms) experienced by the forward limb at IC.[56,61]

The trailing limb is unloaded at the same rate of speed that the lead limb is loaded. The need for forceful stabilization of the ankle and foot has passed. As a result, soleus and gastrocnemius action parallels the rapid decline in the GRF (see Figure 4-22). The perimalleolar muscles respond similarly. Despite early cessation of gastrocsoleus EMG during pre-swing, the ankle continues to plantar flex. Ultrasound studies suggest that the abrupt burst of plantar flexor power during this period is elastic recoil of the Achilles tendon following the quick release of the previously tense soleus and gastrocnemius.[5,15,23,29,41] This allows the forces of the plantar flexor muscles to prepare the trailing limb for swing (Figure 4-23).

As the trailing limb maintains terminal contact of the metatarsal heads and toes with the ground, it provides a fourth rocker (toe rocker) to advance the limb. The velocity of the recoil thrust rapidly introduces ankle PF to 15° (a 25° arc from the starting 10° DF). The foot is free to plantar flex because the body vector is located in the forefoot. The residual plantar flexor muscle action thrusts the tibia forward as the toes are stabilized by floor contact. The effect is rapid knee flexion to 40° (Figure 4-24). The hip advances toward neutral. These changing postures prepare the limb for swing.

The onset of TA and toe extensor muscle action at the end of pre-swing decelerates ankle PF. This also prepares the dorsiflexor muscles to rapidly lift the foot during initial swing.

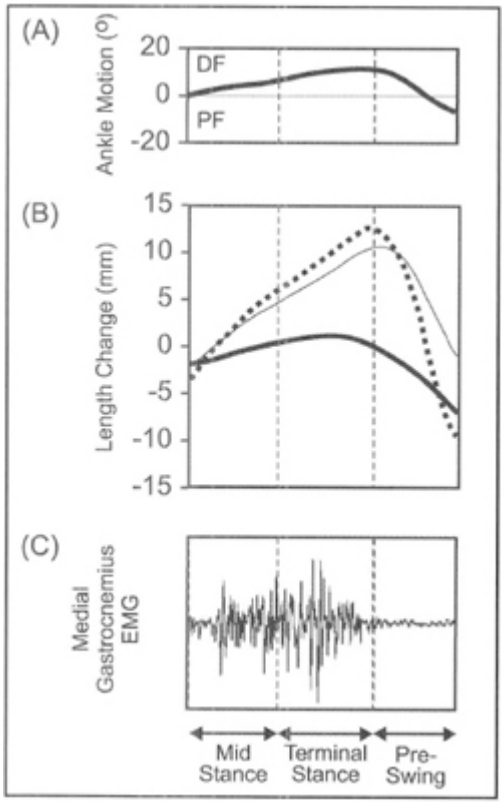

Figure 4-23. Late stance muscle-tendon divergence. (A) During mid and terminal stance, the ankle dorsiflexes. (B) Ultrasound reveals that the source of medial gastrocnemius musculotendinous lengthening (dashed line) is elongation of the tendon (thin line), as muscle fiber length (bold line) does not change substantially. (C) EMG confirms that the medial gastrocnemius muscle is active during the single limb support period. During pre-swing, rapid ankle plantar flexion arises primarily from recoil (shortening) of the stretched tendon as the limb abruptly unloads. Thus, the “push-off” force arises primarily from passive recoil of the stretched tendon and is not a function of dynamic muscle activity. (Adapted from Fukunaga T, Kubo K, Kawakami Y, Fukashiro S, Kanehisa H, Maganaris C. In vivo behavior of human muscle tendon during walking. *Proc R Soc Lond B*. 2001;268:229-233.)

Figure 4-24. Pre-swing pattern of motion, muscular control, and vector alignment at the ankle. Stored energy within the gastrocsoleus plantar flexes the ankle, rolls the limb forward over the toe rocker, and flexes the knee.

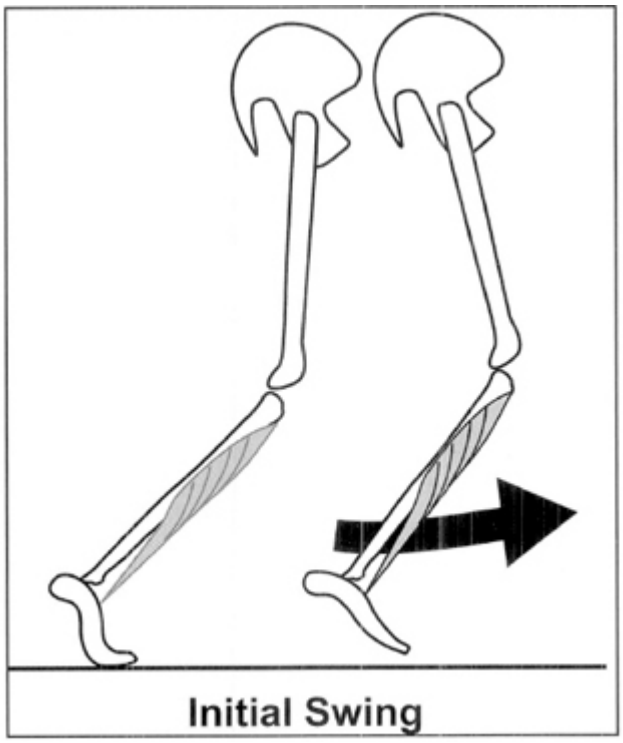

Figure 4-25. Initial swing pattern of motion, muscle control, and vector alignment at the ankle. A rapid increase in pretibial muscle action lifts the foot and toes for floor clearance.

INITIAL SWING (62% TO 75% GC)

Motion: The second arc of DF

Function: Floor clearance for limb advancement

The actions occurring during initial swing are designed to facilitate floor clearance and limb progression. DF of the ankle and toes lifts the foot to aid limb advancement (Figure 4-25). At the instant of "toe-off," the ankle is in 15° PF and the tibia is behind the body. While the limb's trailing position does not immediately obstruct advancement of the limb, it is necessary to promptly reverse ankle motion into DF for subsequent floor clearance as the tibia becomes more vertical. The pretibial muscles quickly increase their intensity, reaching 25% MMT within the first 5% GC interval of initial swing. This almost lifts the foot to neutral (5° PF) by the time the swing foot is opposite the stance limb. As the tibia becomes progressively more vertical by the end of initial swing, the toe extensors respond by increasing their activity to a peak (EDL, 32% MMT; EHL, 40% MMT). Visible DF of the toes is apparent. The anterior tibialis experiences a peak in swing phase activity (34% MMT).

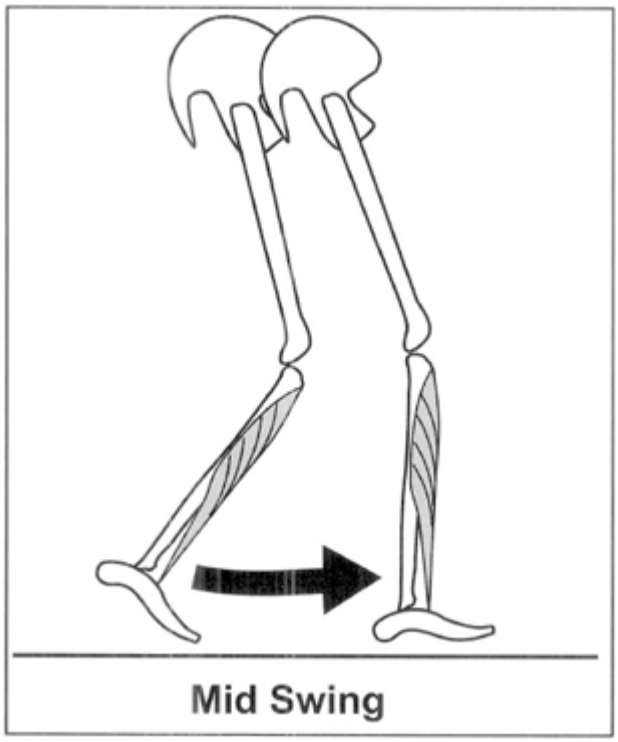

Figure 4-26. Mid swing muscle activity is needed to support the weight of the forefoot.

MID SWING (75% TO 87% GC)

Motion: Continued ankle DF
Function: Floor clearance

Moderate-level activity of the pretibials continues through the first half of mid swing to ensure foot clearance. Ankle DF to neutral or a couple of degrees above is accomplished but not totally maintained (Figure 4-26). The greater relative level of EHL activity may relate to the medial side of the foot being the heavier mass. The notable decline in pretibial activity that occurs during the latter half of mid swing implies that supporting the foot at neutral (an isometric action) is less demanding than the rapid concentric muscle action previously required to lift the foot.

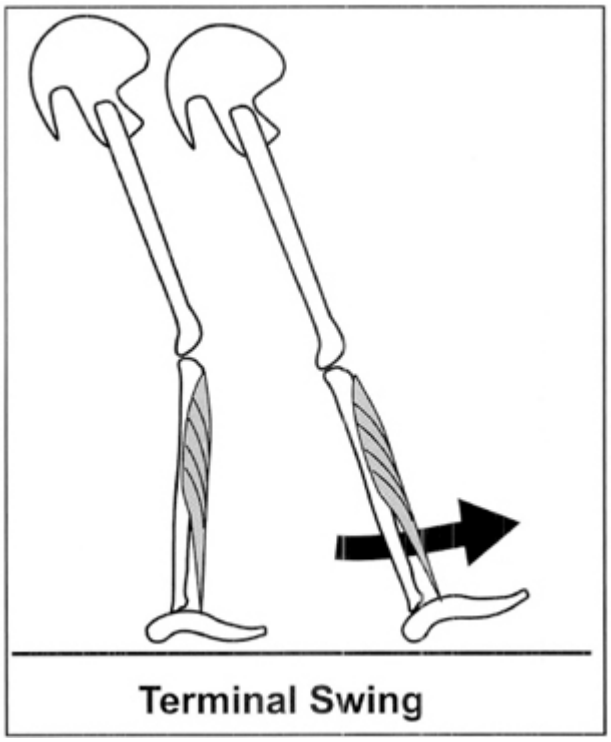

Figure 4-27. Terminal swing. Activity of the pretibial muscles increases in preparation for IC.

TERMINAL SWING (87% TO 100% GC)

Motion: Support of the ankle at neutral
Function: Prepare for IC

Increased pretibial muscle action during terminal swing ensures that the ankle will be at neutral for optimum heel contact in stance (Figure 4-27). Commonly, however, there is a 3° to 5° drop into PF, suggesting the response is not precise. Terminal swing muscle activity prepares the pretibial muscles for the higher demand they will experience during limb loading.

CONCLUSION

Each arc of ankle motion has a unique purpose. The brief arc of ankle PF at the beginning of loading response reduces the impact of heel contact. The prolonged arc of DF from late loading response to

the end of terminal stance facilitates advancement of body weight across the foot. In combination with heel rise, this serves as a major source of forward progression during stance. The final arc of PF prepares the limb for swing by transforming the extended knee of stance into a freely flexing joint. During swing, another arc of DF ensures foot clearance as the limb advances.

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Chapter 5

Knee

The knee is the junction of the 2 long bones (femur and tibia) that constitute the major segments of the lower limb. Small arcs of motion result in significant changes in either foot or body location. Consequently, knee mobility and stability are major factors in the normal pattern of walking. During stance, the knee is the basic determinant of limb stability. In swing, knee flexibility is the primary factor in the limb's freedom to advance. The number of joint muscles involved in knee control also indicates close functional coordination with the hip and the ankle.

KNEE GAIT DYNAMICS

MOTION

The knee is a very complex joint characterized by a large ROM in the sagittal plane and small arcs of coronal and transverse mobility (Figure 5-1). Sagittal motion (flexion and extension) is used for progression in stance, and limb clearance and advancement in swing. Motion in the coronal plane facilitates vertical balance over the limb, particularly during single support. Transverse rotation accommodates the changes in alignment as the body swings from behind to ahead of the supporting limb. Unless joint mobility is exaggerated by pathology, visual analysis identifies only the sagittal motion. Instrumented measurement systems are needed to discern the other events.

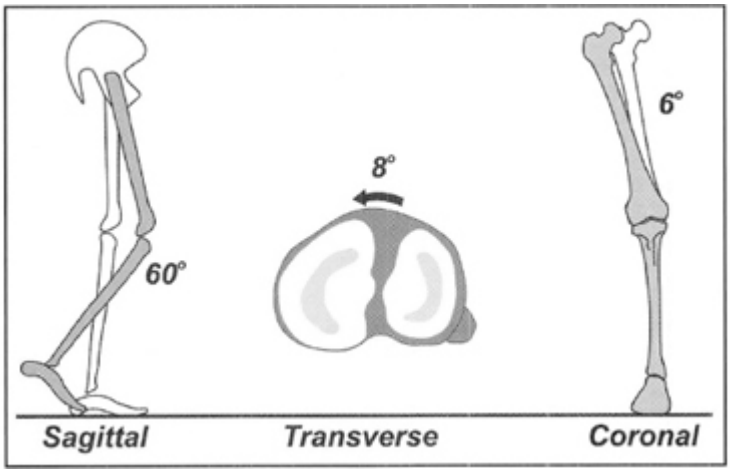

Figure 5-1. Three-dimensional knee motion and arcs used in free walking: sagittal plane flexion (60°); transverse plane rotation (4° to 8°); coronal plane motion (4° abduction, 2° adduction).

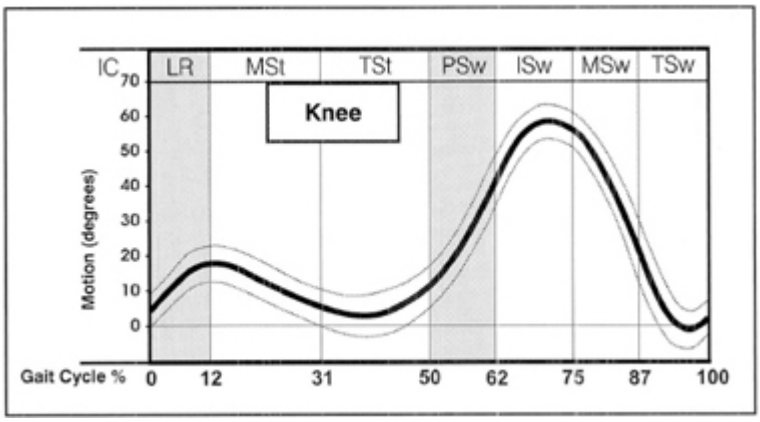

Figure 5-2. Sagittal plane knee motion. Normal range during a GC for free walking. Black line = the mean, dotted lines = one standard deviation. Vertical bars designate the gait phase divisions.

Sagittal Motion

Normal knee motion during walking represents greater and lesser degrees of flexion within the full range of 0° to 60°. During each stride, the knee experiences 2 waves of flexion (Figure 5-2).[3,6-8,10,14] The smaller first wave peaks (20° flexion) at the transition between loading response and mid stance and contributes to controlled shock absorption at the knee. The latter, larger wave crests (60° flexion) during initial swing and assists with foot clearance. The exact limits

of the flexion and extension arcs vary across studies due to differences in walking speed, subject individuality, and the landmarks selected to designate limb segment alignments. As not all studies identified the velocity of walking at the time knee motion was recorded, an exact relationship between these 2 factors cannot be calculated.

At the time of heel contact, the knee is relatively extended with a mean posture of 5° flexion. The normal starting posture, however, may vary from full extension (0°) to 10° flexion.[8] Faster walking speeds are associated with greater knee flexion at IC compared to slower velocities.[15]

Following the onset of loading response, the knee rapidly flexes throughout WA. The rate of flexion at this time (300°/sec) almost equals that occurring in swing. Forefoot contact at 12% GC terminates the heel rocker and, thus, the stimulus for knee flexion ceases. At this time, stance knee flexion is at 20° and the joint is under maximum weight-bearing load.

Loading response knee flexion is significantly influenced by walking speed. Slowing the pace leads to a greater change than going faster. Compared to the motion at 90 m/min, walking at 60 m/min reduced knee flexion by 67%, while increasing the gait speed to 120 m/min led to 38% more knee flexion in loading.[8]

With the onset of mid stance, the knee immediately begins to extend. The rate of motion, however, is half that of the preceding arc of flexion.

During the first half of terminal stance, motion continues toward extension. Minimum stance phase flexion (averaging 5°) is reached about midway in terminal stance (39% GC) and persists for only a short time before the knee slowly begins to flex again. The knee is flexed 10° when floor contact by the other foot ends terminal stance.

Knee flexion rapidly increases following the onset of double limb support in pre-swing. A position of 40° flexion is attained by the end of the phase (62% GC). This primarily passive event occurs as the trailing limb rolls over the anterior edge of the forefoot (toe rocker).

Knee flexion continues at the same fast rate throughout initial swing until the swing foot is opposite the stance foot. At this point, peak knee flexion averages 60°, the maximum knee angle occurring

during the GC.[9] Murray et al reported a peak of 70° flexion based on a strobe system that provided two-dimensional data only.[14] To reach this position in the time available (pre-swing and initial swing phases), the knee flexes at 350°/sec.

During mid swing, as the swing foot advances ahead of the stance limb, less knee flexion is required for foot clearance. Then, following a momentary pause, the knee begins to extend as rapidly as it flexed in the preceding phases.[2,17] Half of the recovery toward maximum extension occurs during mid swing. As mid swing ends, the foot is parallel with the floor and the tibia is vertical.

Knee extension continues at the same rapid rate until full extension (0°) is attained slightly before the end of the swing phase (95% GC). Then the knee tends to drop into a minor degree of flexion. The final knee posture at the end of terminal swing averages 5° flexion.

Transverse Rotation

The basic motion patterns of the tibia, femur, and pelvis, defined by pins in each bone, were found to be of similar direction but of different magnitudes.[11] From a position of maximum external rotation at the end of stance, the entire limb (pelvis, femur, tibia) begins internal rotation at toe-off and continues through swing and loading response. These body segments externally rotate during the rest of stance.

The magnitude as well as direction of rotation at the knee changes across the gait phases. At IC, the tibia is in slight external rotation relative to the femur (ie, the knee is locked). During the loading response, internal rotation of the tibia is markedly accelerated and the femur follows but at a slightly slower rate. As a result, by the end of loading response, the tibia has moved through a 4° to 8° arc of internal rotation relative to the femur.[11] Transfer of the ST eversion to the tibia has unlocked the knee at a time when the knee is functionally required to flex for shock absorption. By the end of loading response (ie, initial double support), bone pin data indicate

that both the knee joint and the total limb have reached their peak of inward rotation.

As the knee begins to extend in SLS, the pelvis, femur, and tibia begin to rotate externally. During mid stance and the first half of terminal stance, the tibia externally rotates at a faster rate than the femur. This relative tibial external rotation locks the knee at the time in SLS when knee extension is required for stability.

During the latter half of terminal stance and the beginning of pre-swing, the tibia internally rotates approximately 1° relative to the femur. This coincides with the period when knee flexion is initiated in preparation for toe clearance during swing.

With toe-off (initial swing), the tibia, femur, and pelvis each initiate an arc of internal rotation. This motion will terminate at the end of loading response.

Consistent with descriptions of the "screw-home mechanism,"[13] terminal extension of the knee during the GC is generally accompanied by external rotation of the tibia relative to the femur. Correspondingly, the initiation of knee flexion is accompanied by internal rotation of the tibia relative to the femur.

Coronal Plane Motion

The knee moves into both abduction and adduction within each GC.[5] The knee is abducted during stance, with the greatest angle (4°) occurring during WA. With the onset of swing, the knee transitions to adduction, with the peak posture (2°) occurring in mid swing. Terminal swing marks the transition to knee adduction.

Muscle Control

Fourteen muscles contribute to knee control during each GC. Their purpose is to provide the stability and mobility needed for walking, but they also are quiescent whenever possible to conserve energy.

During stance, the extensors act to decelerate knee flexion. In swing, both the flexors and extensors contribute to limb progression.

Of the multiple muscles acting on the knee, only 6 have no responsibility at another joint. These are the 4 vastii heads of the quadriceps, which resist knee flexion in stance and assist with extension at the end of swing, and the popliteus and biceps femoris short head (BFSH), which flex the knee. The primary role of one knee flexor, the gastrocnemius, is ankle PF. All of the other muscles also control hip motion (either flexion or extension).

Knee Extension

The quadriceps is the dominant muscle group at the knee. Four heads cross only the knee joint (vastus intermedius, vastus lateralis, vastus medialis oblique, vastus medialis longus). The fifth head (rectus femoris [RF]) includes both the knee and hip.

Activity of the vastii muscles begins in terminal swing (89% to 95% GC) (Figure 5-3). Muscle intensity rapidly increases to a peak (21% to 38% MMT) early in loading response (approximately 6% GC). This level of effort gradually diminishes throughout the remainder of loading response. With the onset of mid stance, the quadriceps rapidly reduces its effort and ceases by the 20% GC point.

The timing and intensity of RF action is much different from that of the vastii (see Figure 5-3). Activity of the RF, demonstrated with the selectivity of wire electrodes, has a short period of action between late pre-swing (57% GC) and early initial swing (65% GC). The intensity during this interval is under 20% MMT. Seldom does the RF accompany the vastii in loading response (unless surface electrodes have been used and cross talk from the vastii is recorded).[16]

One hip extensor muscle also contributes to knee extension stability in early stance. The upper gluteus maximus provides a knee extensor force through its iliotibial (IT) band insertion on the anterior-lateral rim of the tibia (Figure 5-4). Tension of the IT band, which crosses anterior to the knee joint, resists flexion. Activity of the upper gluteus maximus begins in late terminal swing (95% GC) and terminates by the middle of mid stance (24% GC). The upper gluteus maximus registers a significant effort level (25% and then 20% MMT) throughout most of this time period.

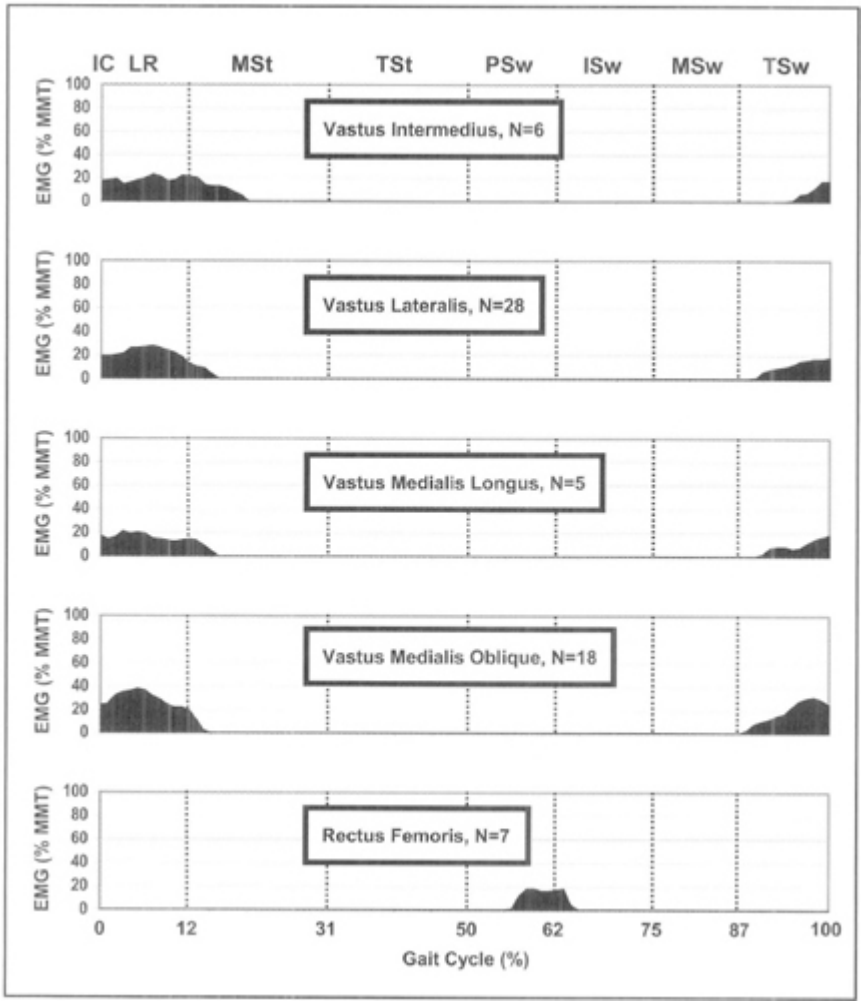

Figure 5-3. Knee extensor muscles: Normal mean intensity and timing during free walking (quantified EMG). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. Vertical bars designate the gait phase divisions. N = samples included in data.

Knee Flexion

Two single joint muscles, popliteus and the BFSH, provide direct knee flexion (Figure 5-5). The BFSH is primarily active in initial and mid swing (65% to 82% GC). Less frequently, there may be activity in terminal stance (33% to 45% GC). EMG recordings of the popliteus show no consistent pattern. While individuals are

consistent in their use of the popliteus, its action may occur during any of the phases of the GC except initial swing. The muscle's greatest intensity generally occurs at the initiation of pre-swing (50% GC; 20% MMT). Another period of moderate effort (17% MMT) begins in terminal swing and continues through loading response.

The 3 hamstring muscles (semimembranosus, biceps femoris long head (BFLH), and semitendinosus) are primarily hip extensors, but these muscles are better known for their flexor role at the knee (Figure 5-6). Semimembranosus and BFLH initiate their activity during mid swing and are joined in terminal swing by semitendinosus. All 3 muscles have their most intense action in terminal swing and then show a progressive decline until their cessation in either loading response (BFLH) or mid stance (semimembranosus and semitendinosus).

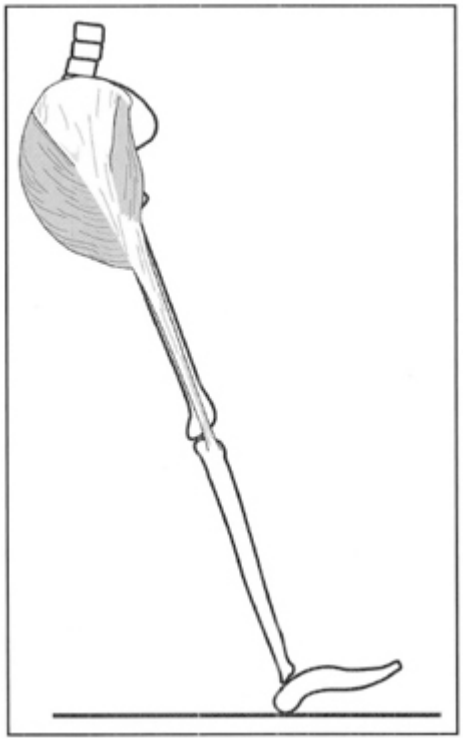

Figure 5-4. Upper gluteus maximus as a knee extensor through its insertion on the IT band.

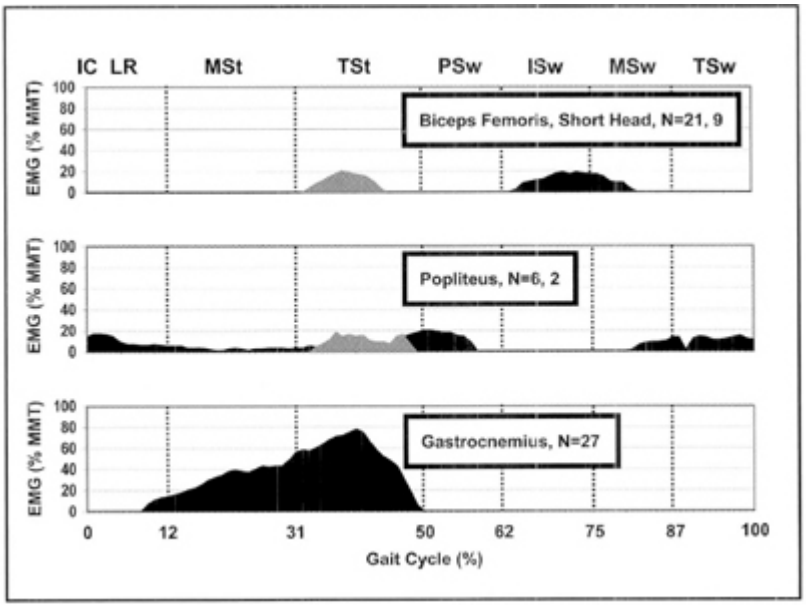

Figure 5-5. Knee flexor muscles (distal). Normal mean intensity and timing during free walking (quantified EMG). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of the shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in data.

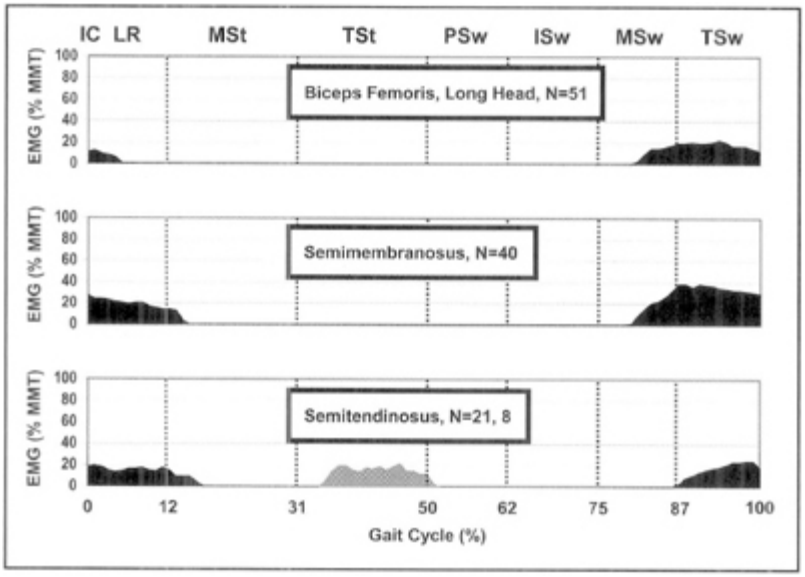

Figure 5-6. Hamstring muscles as knee flexors. Normal mean intensity and timing during free walking (quantified EMG). Intensity as a percent of maximum manual muscle test value (% MMT)

indicated by height of the shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in data.

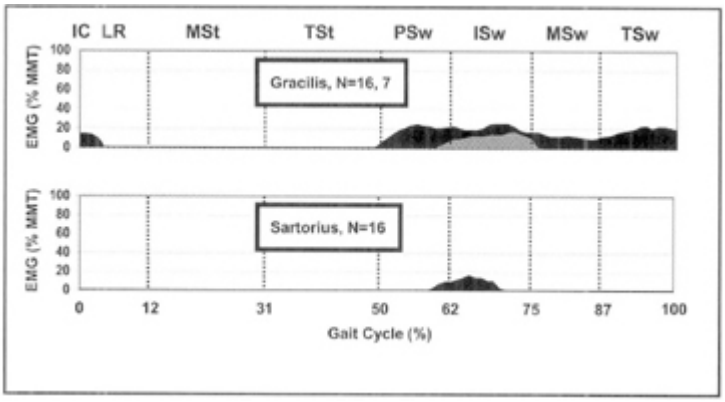

Figure 5-7. Combined knee and hip flexor muscles. Normal mean intensity and timing during free walking (quantified EMG). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in data.

The gastrocnemius progressively increases its intensity from the time of onset (9% GC) until the middle of terminal stance (78% MMT at 40% GC) (see Figure 5-5). Then there is a rapid decline of action until it ceases with the onset of pre-swing.

Two hip flexor muscles also contribute to swing phase knee flexion. These are the gracilis and sartorius (Figure 5-7). Both muscles become active in pre-swing (50% and 60% GC, respectively). The duration of activity is much longer for the gracilis (ceases in loading response) than the sartorius (activity ends in initial swing).

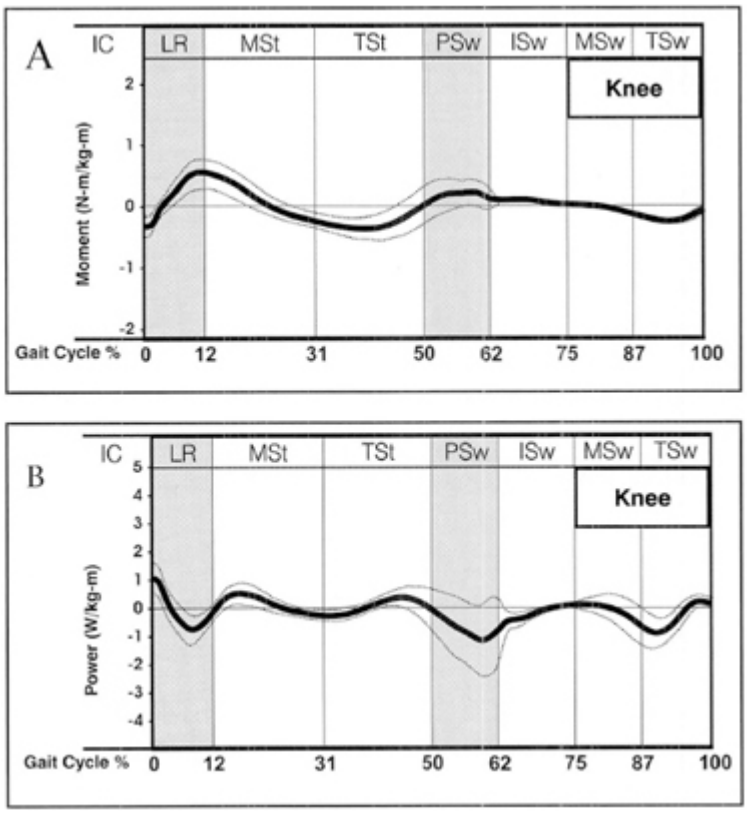

Figure 5-8. Knee joint forces. (A) Knee joint moments: Normal weight-bearing pattern generated by the sagittal vector during walking. There are 5 peaks, 3 flexor moments (-) with extensor moments (+) interspersed between. (B) Knee joint power demonstrating periods of power absorption (-) and generation (+).

FORCES

The abrupt impact of initial floor contact creates a vertically oriented vector that is aligned anterior to the knee joint. A brief, low amplitude flexor moment (0.35 N•m/kg) prevents knee hyperextension as energy is generated (1.0 W/kg•m at 1% GC; Figure 5-8). As the knee rapidly flexes during loading response, an extensor moment ensures stability (0.52 N•m/kg) across the joint, and power is absorbed (peak 0.8 W/kg•m at 8% GC) due to the eccentric activity of the vastii. A small burst of power generation in early mid stance (peak 0.5 W/kg•m at 16% GC) augments knee extension. Then, the extensor moment rapidly diminishes and by the end of mid stance, a small flexor moment is present that persists

through terminal stance (peak 0.36 N•m/kg at 38% GC). During pre-swing and initial swing, a low amplitude extensor moment (peak 0.21 N•m/kg at 58% GC) modulates the rate of rapid knee flexion. Peak power absorption at the knee occurs during this period (1.2 W/kg•m at 59% GC). As the knee extends in late swing, the flexor moment again increases (peak 0.26 N•m/kg at 93% GC) and power is absorbed (peak 0.9 W/kg•m at 90% GC) as the hamstrings eccentrically control the rate of knee extension.

FUNCTIONAL INTERPRETATION OF THE KNEE

The knee has 4 functional obligations during walking. Two occur during stance as the limb is loaded: shock absorption and extensor stability for secure weight bearing. In swing, the knee must rapidly flex for foot clearance and then extend to ensure optimal limb advancement. The relationships among motion, muscle action, and force relate to these demands.

Figure 5-9. IC knee control: Anterior and posterior stabilization by the quadriceps and hamstrings. The anterior vector presents a flexor moment.

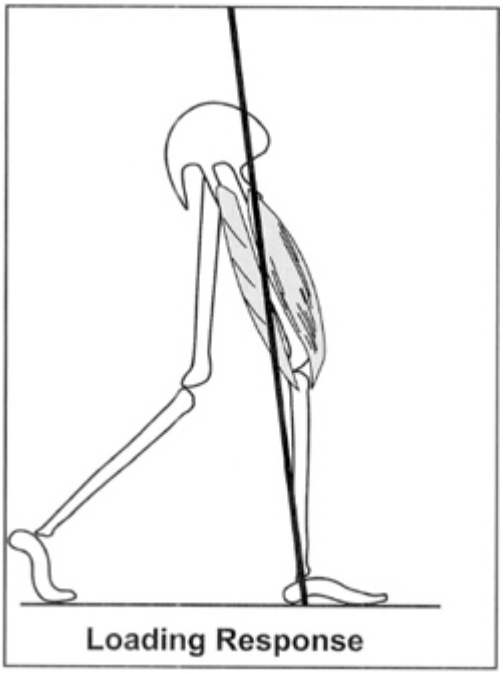

Figure 5-10. Loading response knee control: Quadriceps extension opposes the posterior vector. Hamstring activity is waning.

INITIAL CONTACT (0% TO 2% GC)

Posture: Knee appears fully extended
Function: Stable weight bearing

At the instant of initial heel contact with the floor, the knee observationally appears extended (5° flexion) and 2 extensor mechanisms are present (Figure 5-9). First is the alignment of the body vector anterior to the knee axis. Second is active muscular control by the vastii and a tense IT band by upper gluteus maximus activity. Continued low-level action (approximately 10% to 20% MMT) by the hamstring provides a protective flexor moment to prevent knee hyperextension.

LOADING RESPONSE (2% TO 12% GC)

Motion: Knee flexion (20°)
Function: Shock absorption
Stability maintenance

The rapid rate of body weight transfer onto the limb disrupts the knee's stable extended posture and initiates knee flexion (Figure 5-10). The heel rocker rolls the tibia forward faster than the femur can advance. This drives the knee joint anterior to the body vector, and an extensor moment is required to stabilize the knee. The prompt response by the vastii muscles limits knee flexion to approximately 20°. The vastii function eccentrically to restrain (decelerate) but not totally prevent knee flexion. This action serves as a shock absorber while also maintaining stability so that the knee is not allowed to collapse. The joint is protected from the deleterious force of full floor impact by having the heel rocker partially redirect the force into the contracting, yet yielding quadriceps musculotendinous unit. As a result, the peak vertical force is only 110% to 120% of body weight (see Figure 3-29).

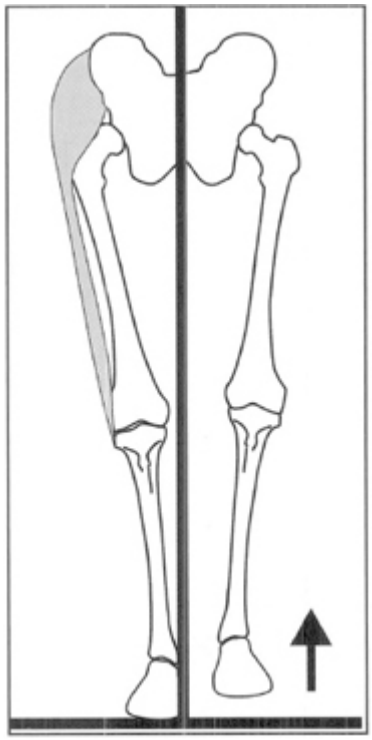

Figure 5-11. The IT band provides lateral restraint in the presence of an abductor moment.

During this phase, BFLH activity ceases (5% GC) while the medial hamstrings slowly decline. The continuous decline in hamstring activity during loading response implies the role of these muscles is primarily protection from potential hyperextension during IC. Thus, the loading response phase challenges the knee's weight-bearing posture in order to provide shock absorption. The strength of the vastii's response is critical to establishing a stable limb.

A second shock-absorbing mechanism also challenges knee stability. The rear foot eversion that occurs in early stance initiates internal rotation of the tibia relative to the femur. Excessive rotation within the joint may be opposed by the external rotational pull of the tensor fascia lata and biceps femoris during the heel support period of loading response.

In the coronal plane, an abductor moment develops at the knee in response to the rapid unloading and dropping of the opposite side of the body (Figure 5-11). Tensing of the IT band provides a lateral counter force to stabilize the knee.

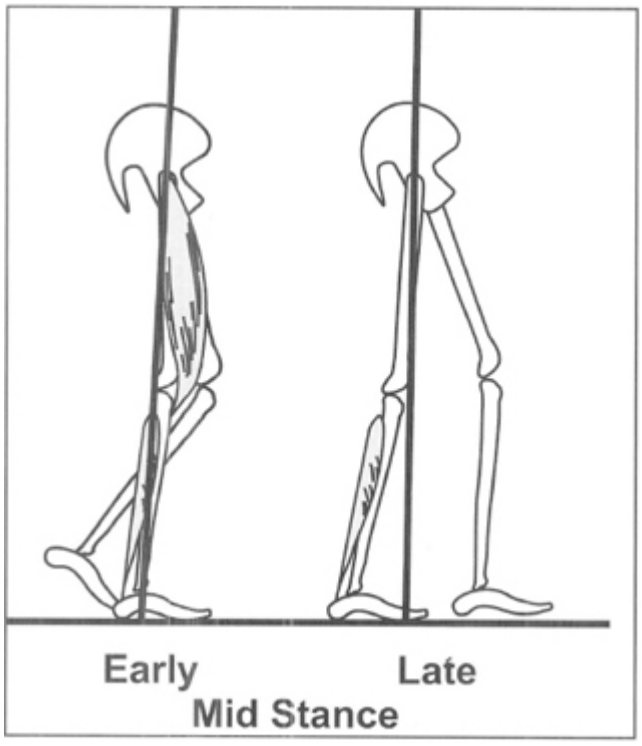

Figure 5-12. Mid stance knee control. Early: Concentric vastii activity. Late: Anterior force vector provides passive knee extension over a tibia stabilized by the soleus.

MID STANCE (12% TO 31% GC)

Motion: Knee extension
Function: Stable weight bearing

Stance stability is optimum when the knee is extended. Attaining this posture, however, involves several steps. At the initiation of mid stance, the vastii generate a small burst of power to advance the femur over the stable tibia. Knee flexion decreases to 15°. Quadriceps activity ceases by the middle of mid stance (20% GC). Momentum from the contralateral swinging limb provides a passive force to continue the reduction in knee flexion. With the ankle now positioned in DF, slowing of tibial advancement by the soleus enables the femur to advance at a faster rate than the tibia (Figure 5-12). The body mass moves slightly anterior as the knee extends and the ankle dorsiflexes, bringing its vector closer to the knee joint axis. By the middle of the mid stance phase (23% GC), the vector is in line with the knee axis. It then moves slightly anterior to the knee joint center, replacing the need for muscle action by the stability created by the body's alignment. There is no further action by the vastii and the accelerative phase of swing limb advancement is terminating. The posterior capsule and tendinous structures prevent knee hyperextension.

The coronal plane abductor moment persists through mid stance at a reduced level. The body's COG shifts laterally 2 cm while the feet follow a path 4 cm from the midline. Hence, body weight never moves a sufficient distance to have it lie directly over the supporting foot. As a result, the body weight vector remains on the medial edge of the knee (2.5 cm from its center). This increases the force on the medial portion of the knee and also necessitates extrinsic lateral support as the collateral ligament is small. Despite this apparent asymmetry in forces, none of the lateral musculature that directly affects the knee remains active during mid stance. An available

source of lateral stability, however, is IT band tension through the activity of the hip abductors.

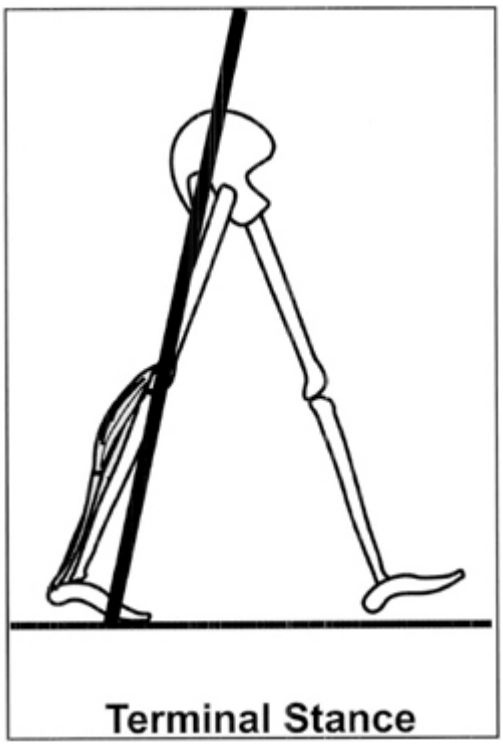

Figure 5-13. Terminal stance knee control: Passive extension by an anterior vector with the tibia stabilized by the soleus.

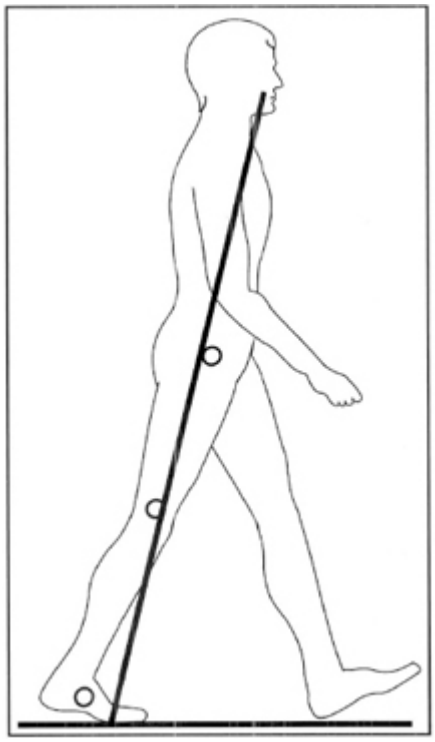

Figure 5-14. Relationship between the COP and the anatomical forefoot rocker during terminal stance.

TERMINAL STANCE (31% TO 50% GC)

Motion: Maximum extension
Function: Stable weight bearing
Maximum step length

As the femur continues to advance over the stable tibia, knee flexion is reduced to the minimum stance posture by the middle of terminal stance (5° at 39% GC; Figure 5-13). The energy source appears to be passive momentum from the advancing body mass. At the same time, the forefoot rocker facilitates forward fall of the body vector across the metatarsal heads (Figure 5-14). These extensor mechanisms have the potential to create knee hyperextension, but protection is available by posterior muscle action. The popliteus starts increasing its activity (peak of 14% MMT during terminal stance) and the gastrocnemius already is contracting vigorously (78% MMT) in its role as an ankle stabilizer. While the intensity of the gastrocnemius is much greater, the deep popliteus has the

advantage of lying on the joint capsule. The BFSH is active during terminal stance (21% MMT) in a small percentage (approximately one third) of subjects and may provide further protection from knee hyperextension.

The attainment of maximum stance knee extension is almost immediately reversed toward knee flexion. Several factors contribute. Tibial stability is lost as the body vector moves across the MTP joints and the knee joint advances ahead of the vector. The posterior muscles, which initially prevent hyperextension (popliteus, gastrocnemius, BFSH), can also initiate knee flexion as heel rise advances the knee joint toward the body vector. Additionally, during the latter half of terminal stance, the tibia internally rotates approximately 1° relative to the femur, which may further free the knee to unlock. By the end of terminal stance, the knee is flexed 10°.

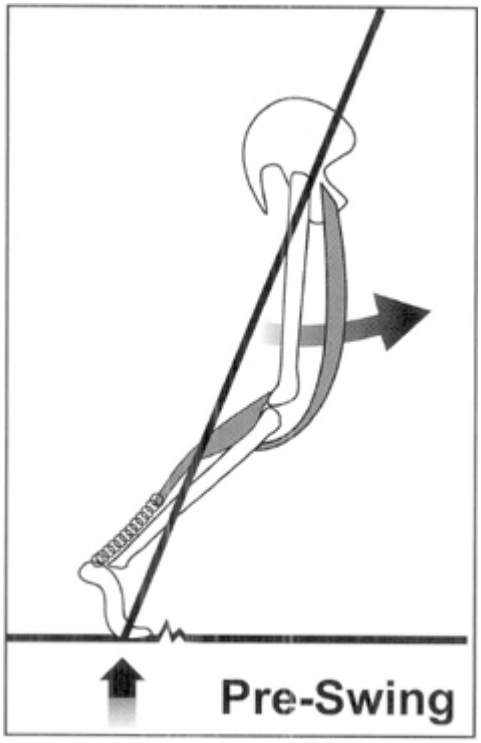

Figure 5-15. Pre-swing knee control: Residual tension in the plantar flexors advances the foot over the toe rocker. Excessive flexion (arrow) restrained by RF (occasionally a vastus responds).

PRE-SWING (50% TO 62% GC)

Motion: Passive knee flexion

Function: Prepare for swing

The rapid rate of body weight transfer that follows contralateral foot contact simultaneously reduces the load on the trailing toe rocker (Figure 5-15). Advancement of the COP (base of the vector) to the distal side of the MTP joints removes the force that previously held the midfoot close to the floor. With foot stability lost, the tibia is free to roll forward. Residual tension in the previously strongly active plantar flexor muscles accelerates heel rise and tibial advancement. As a result, the knee is free to passively fall into flexion as the elastic recoil of the Achilles tendon rolls the tibia forward. There is also direct knee flexor muscle action by the popliteus, gracilis, and sartorius (10% to 20% MMT). The result is 40° of knee flexion, which prepares the limb for easy toe clearance in swing.[1] When knee flexion proceeds too fast, the RF responds. This muscle serves to simultaneously decelerate excessive knee flexion and to initiate hip flexion. As the average intensity of RF activity is only 13% MMT, the demand obviously is mild. Occasionally, the vastus intermedius also responds briefly.[4]

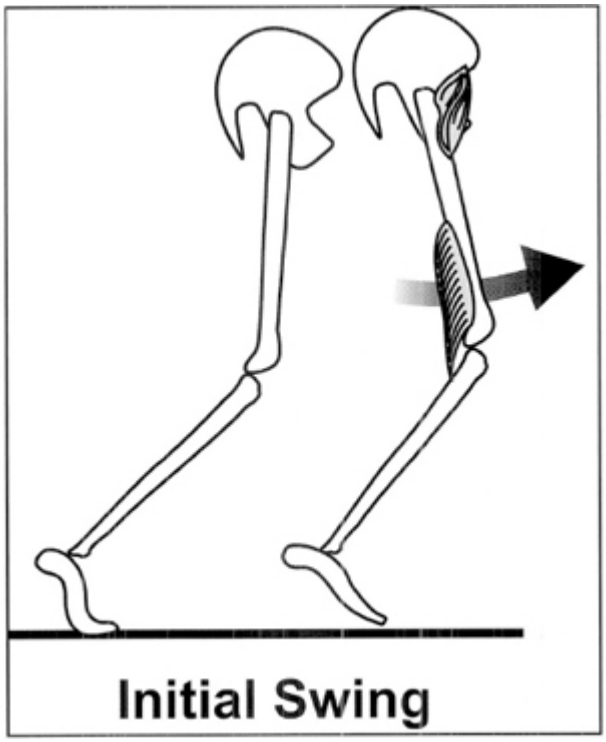

Figure 5-16. Initial swing knee control: Flexion augmented by forward thigh momentum (arrow) and flexor muscle action (BFSH, gracilis, sartorius).

INITIAL SWING (62% TO 75% GC)

Motion: Knee flexion

Function: Foot clearance for limb advancement

Knee flexion is the essential motion to lift the foot for limb advancement. The trailing posture of the limb at the end of pre-swing combined with knee flexion places the toe in an equinus position (ie, toe down). This adds foot length to the distance between the hip and toe. Consequently, the trailing limb is functionally longer than the other limb's standing distance between the hip and floor. Mere ankle DF is insufficient to lift the toe for unobstructed limb advancement. The necessary additional lift must be supplied by knee flexion of 60° (Figure 5-16). This is the critical action that ensures foot clearance of the floor as the limb swings forward from its trailing posture. Both timing and magnitude of motion are critical. Pre-swing knee flexion (40°) is essential. Direct flexor action is available from the BFSH, the sartorius, and gracilis muscles, which simultaneously flex the knee and the hip. All 3 muscles reach their peak in activity (approximately 20% MMT) during initial swing. Additionally, momentum is initiated by rapid hip flexion. This quickly advances the femur while tibial inertia leads to knee flexion.[12]

This complex mode of lifting the foot presents a functional paradox. The objective is toe clearance of the floor, yet knee flexion rather than ankle DF is the essential action.

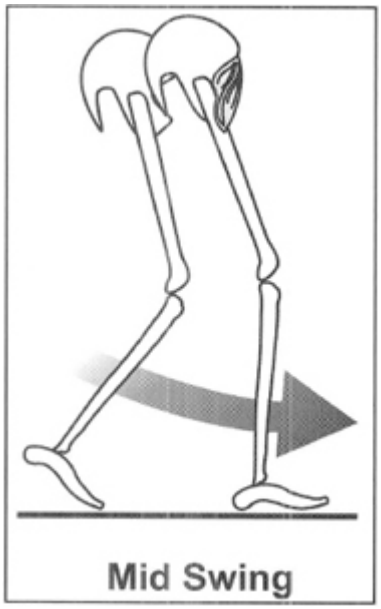

Figure 5-17. Mid swing knee control: Passive extension as the flexors relax and the thigh advances (arrow).

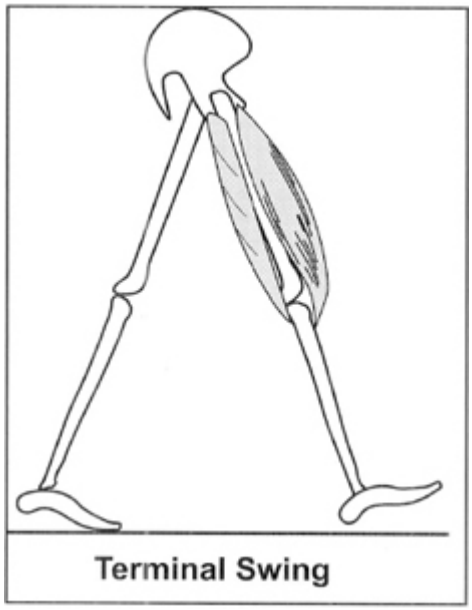

Figure 5-18. Terminal swing knee control: Hamstring activity eccentrically modulates the rate of knee extension while quadriceps activity ensures full extension and prepares for WA.

MID SWING (75% TO 87% GC)

Motion: Passive knee extension

Function: Limb advancement

Once the foot has moved forward of the hip joint, knee position does not contribute to the threat of the foot dragging on the floor. The need for acute flexion has subsided, and the knee is now free to extend (Figure 5-17).

No knee extensor muscle action is needed as the trailing posture of the shank makes gravity an available force once the knee flexor muscles relax. Momentum generated by the continuing hip flexion supplements the pull of gravity on the tibia. These forces reach a balance once the tibia becomes vertical. At the end of mid swing, BFLH and semimembranosus activity are initiated in preparation for their role in controlling the rate of knee extension in terminal swing.

Terminal Swing (87% to 100% GC)

Motion: Knee extension
Functions: Limb advancement
Prepare for stance

To prepare the limb for stance, the knee flexion used in early swing must be reversed to extension. A flexor moment at the knee controls the rate of knee extension. Activity of the hamstrings (semimembranosus, semitendinosus, and BFLH) peaks to eccentrically modulate the rate of knee extension (and hip flexion). The effect of the hamstring muscle force at the knee will be no more than half that at the hip because of the shorter functional lever arm available. Onset of all 4 vastii occurs during this phase to ensure full knee extension as well as to prepare the knee for the high demands associated with the abrupt drop of body weight during WA (Figure 5-18). Dynamic EMG studies confirm participation by the quadriceps (vastii).[16] The rectus is not used because further hip flexion is undesirable.

CONCLUSION

The knee is the key to stance stability, and the quadriceps is the most direct source of extensor control. During loading response, however, the quadriceps is only used to restrain the shock-absorbing flexion. With the transition to SLS, knee extension stability is provided by an anterior vector and calf stabilization of the tibia. In swing, the knee uses a larger arc of motion than any other joint. Sixty degrees of flexion are needed to ensure toe clearance of the floor, with 40° being attained during pre-swing. Consequently, knee function influences the entire limb in both stance and swing.

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Chapter 6

Hip

Function at the hip differs from the other joints in several respects. The hip represents the junction between the passenger and locomotor units. As a result, it is designed to more overtly provide three-dimensional motion with specific muscle control for each direction of activity. Sagittal plane motion (progression) involves the largest arc, while muscular requirements are brief. In the coronal plane, motion is limited but the muscular demands are substantial. Transverse rotation remains a subtle event.

The functional focus of the hip musculature also varies with the period in the GC. During stance, the primary role of the hip muscles is stabilization of the superimposed trunk. In swing, limb advancement and foot clearance are primary objectives.

HIP GAIT DYNAMICS

Motion

Clinically, it is more appropriate to define the joint's motion by the path of thigh displacement from the vertical. Customary ways of describing the arc of hip motion are influenced by displacement of both the femur and pelvis. Hence an arc of pelvic tilt may either add to or subtract from the arc of hip motion created by thigh displacement. In considering the mechanics of walking, the thigh and pelvis motions should be evaluated separately in the clinical setting, although instrumented motion analysis generally measures total pelvic-femur angle. During normal function, the ranges of pelvic

motion are quite small, but they can be quite significant with disability.

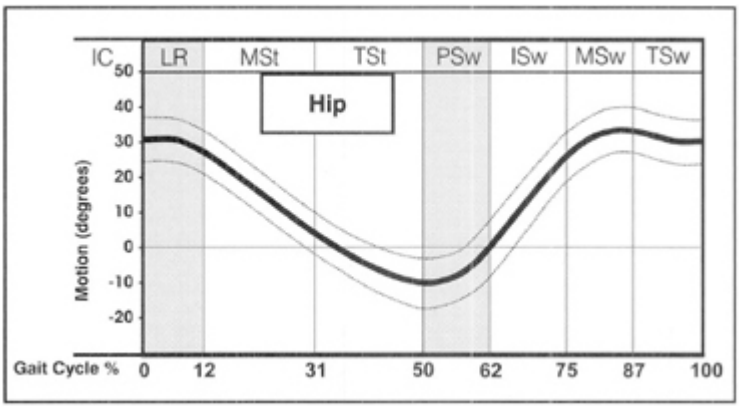

Figure 6-1. Sagittal plane hip motion (thigh relative to pelvis). Normal range during free walking is 40°. The starting position for the pelvis is with a 10° anterior tilt, resulting in an arc of motion of 30° flexion to 10° extension. Black line = the mean, dotted lines = one standard deviation. Vertical bars designate the gait phase divisions.

The coronal and transverse plane motions recorded during stance tend to be identified as pelvic motion, yet the hip joint is the site of this action. In this text, such events are described under both the hip and pelvis sections of the book.

Sagittal Plane Motion

The hip moves through only 2 arcs of motion during a normal stride: extension during stance and flexion in swing (Figure 6-1). The exchange of motion from one direction to the other is gradual. The normal arc of hip motion reported in the literature ranges from 40° to 48°,[7,8,10,11,15,18] with the precise definition varying based on the recording technique used. Some investigators consider maximum hip extension as zero (0°) and note maximum hip flexion as 40°.[7,10]

A technique that is more consistent with clinical practice is to consider the vertical thigh in quiet standing to be the zero position.[11,15,18] By expressing the thigh position relative to vertical (0°), the displacement of the limb in space is defined independent of pelvic motion. The pelvis is treated as a separate segment, riding on

top of a thigh. For normal walking, this excludes the mean 10° anterior pelvic tilt from the measurement of the hip. Using the vertical thigh reference system, the peaks in thigh motion are 20° extension during terminal stance and 25° flexion in mid swing (Figure 6-2). Hip motion would be determined by combining displacement of the pelvis and thigh.

Clinically, it is important to separate motion of the thigh from that of the pelvis as they respond differently to the various types of pathology that modify the patient's ability to walk. Also, independent analysis of the pattern of normal thigh extension and flexion relative to the vertical plane more clearly identifies the contribution of the thigh to stride length.[14]

The thigh at IC is flexed 20° (see Figure 6-2) from the vertical. During the loading response, thigh position is relatively stable, perhaps losing 2° or 3° of flexion. The hip progressively extends with the onset of mid stance, reaching neutral at 27% GC. In terminal stance, the thigh continues to extend at the same rate until it reaches a peak of 20° of apparent hyperextension as the other foot contacts the ground (50% GC). The term *apparent hyperextension* is used as the hip joint normally allows no more than half of this range. Instead, the posture arises from 3 anatomical interactions that occur at the end of terminal stance: full hip extension, an increase in anterior pelvic tilt (3° to 7° degrees),[15,16] and 5° backward (external) pelvic rotation.[12]

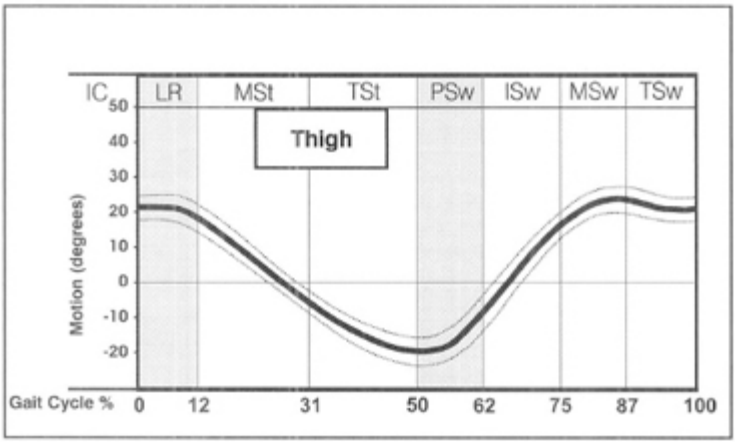

Figure 6-2. Sagittal plane thigh motion (thigh relative to vertical). Normal range during free walking. Black line = the mean, dotted lines

= one standard deviation. Vertical bars designate the gait phase divisions.

During pre-swing, the hip begins flexing, and apparent hyperextension of the thigh is reduced to 10° by the end of the phase. The motion toward flexion continues through the first 2 phases of swing. During initial swing, the hip attains a large portion of its flexion range, with the thigh reaching 15°. By the end of mid swing, an additional 10° of flexion occurs for a peak of 25° flexion. A subtle past retract (backward movement of the flexed thigh) during terminal swing contributes to a final thigh position of 20° flexion just prior to IC.

Coronal Plane Motion

The hip moves through a small arc of adduction and abduction as the unloaded side of the pelvis follows the swinging limb. This action begins with the onset of stance. At IC, the hip (thigh relative to pelvis) is approximately neutral (0°) in the coronal plane due to the anatomical angle between the femur and tibia. As body weight loads onto the limb, adduction increases to 10° by the end of loading response. This arises due to a combination of contralateral pelvic drop and displacement of the femur. During SLS, this motion gradually diminishes and by the mid point of pre-swing (56% GC) the thigh regains its neutral position in the coronal plane. During the remainder of pre-swing and initial swing, the thigh abducts, achieving a peak of 5° shortly after toe-off (65% GC). Then, during mid and terminal swing, the thigh assumes a neutral posture. Relatively small arcs of motion occur in both men and women.[15]

Transverse Plane Motion

During each stride, the limb moves through an arc of internal rotation followed by a similar arc of external rotation. Pelvis and thigh skeletal pins demonstrated that the limb is neutral at IC. Peak internal rotation of the thigh occurs at the end of the loading

response and maximum external rotation is found at the beginning of initial swing.[12] The total arc of transverse thigh motion averages 8°. When this arc is added to the pelvic motion (7.7°), total hip rotation averages 15°. Surface markers show a similar arc of hip motion, although there is great variability in actual values among the different gait laboratories.[2] The tracking of relatively small arcs of motion when combined with the potential for substantial artifact due to soft tissue movement may account for differences across studies.

Muscle Control

During stance, the primary muscles controlling the hip are the extensors and abductors. In swing, they are the flexors. The adductors tend to participate during the intervals of exchange between swing and stance. Inability to isolate the deep external rotators has prevented definition of their timing. Internal rotation is provided secondarily as the muscles also perform their primary function.

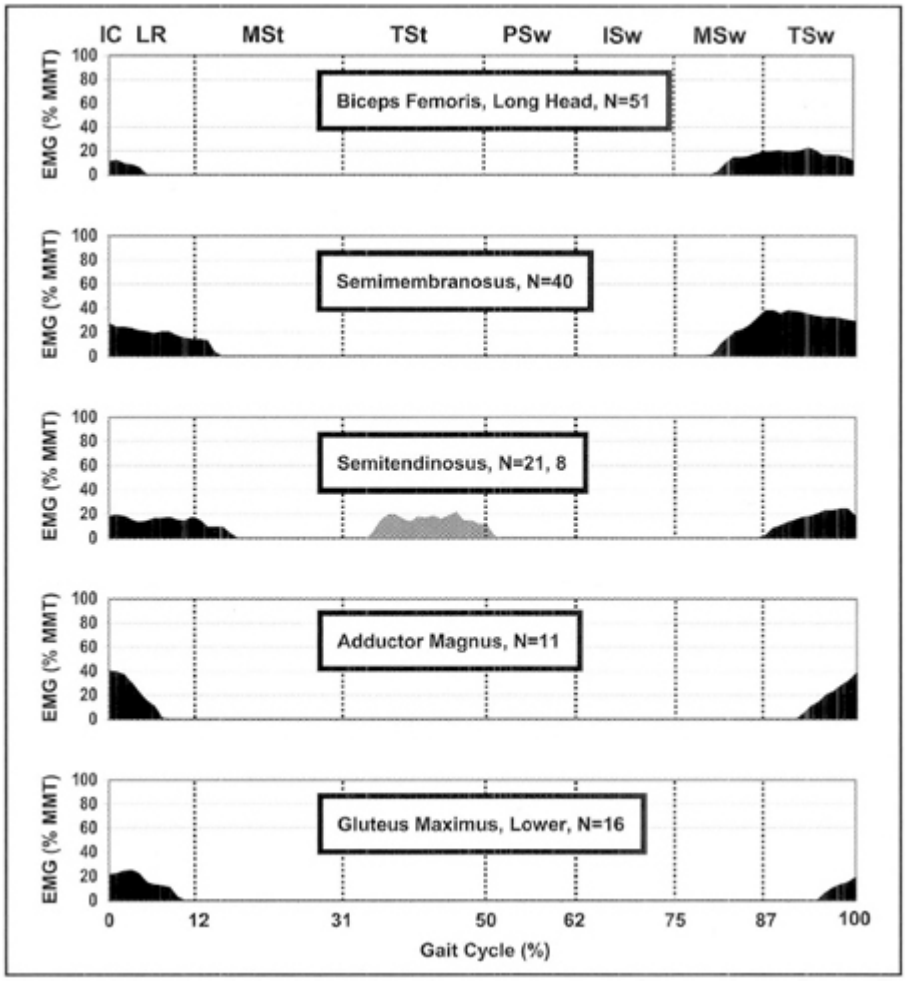

Figure 6-3. Hip extensor muscles. Normal mean intensity and timing during free walking (quantified electromyogram). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in data.

Extensor Muscles

Action of the hip extensor muscles occurs from late mid swing through the loading response. The 5 muscles involved are selective in their timing (Figure 6-3).[9,13]

Hamstrings

The semimembranosus (81% GC) and long head of the biceps femoris (82% GC) begin contracting in late mid swing, while the semitendinosus waits until the onset of terminal swing (88% GC). All 3 muscles rapidly increase the intensity of their action, reaching peak effort early in terminal swing (semimembranosus 38% MMT, semitendinosus 24% MMT, and BFLH 22% MMT). The biceps femoris then decreases its intensity and ceases action in early loading response (5% GC), while the semimembranosus and semitendinosus continue into early mid stance (15% GC and 17% GC, respectively).

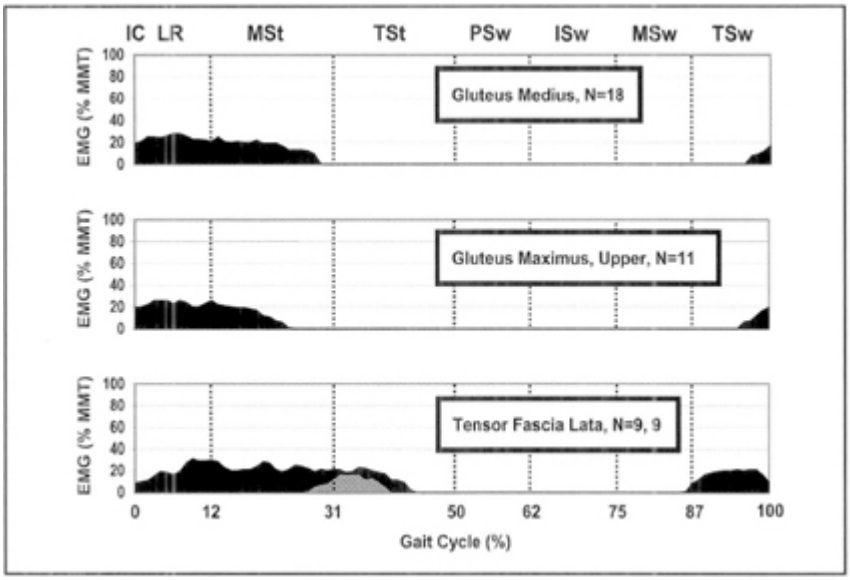

Figure 6-4. Hip abductor muscles. Normal mean intensity and timing during free walking (quantified electromyogram). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in data.

Adductor Magnus

The adductor magnus begins to contract near the end of terminal swing (92% GC) and progressively increases its intensity throughout the phase. There is a further increase to 40% MMT with IC. During loading response, the adductor magnus remains moderately active

for 7% of the GC and then relaxes. No further activity occurs throughout the stride.

Gluteus Maximus

Functionally, the muscle divides into 2 halves. The upper half acts as an abductor while the lower half serves as a hip extensor.[13] Lower gluteus maximus action begins with a low intensity (10% MMT) near the end of terminal swing (95% GC). With IC, the muscle quickly increases in intensity to reach a 25% effort level early in the loading response. Following this peak, the lower gluteus maximus rapidly decreases its activity and ceases by the end of loading response (10% GC).

Abductor Muscles

The hip abductors are the other major muscle group functioning during the initial half of stance. Three muscles are involved: gluteus medius, upper gluteus maximus, and tensor fascia lata (Figure 6-4).

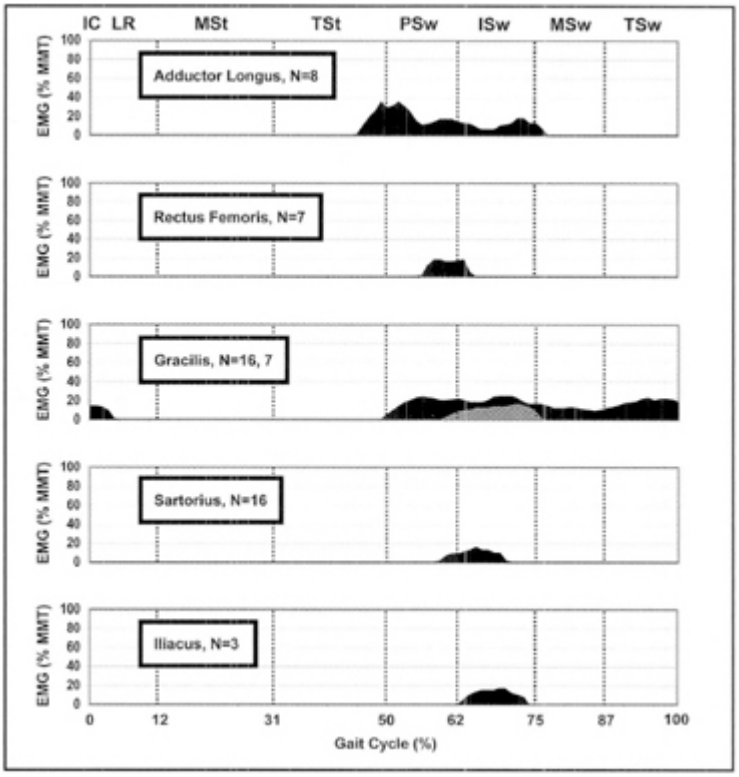

Figure 6-5. Hip flexor muscles. Normal mean intensity and timing during free walking (quantified electromyogram). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in data.

The pattern of gluteus minimus activity during walking has been identified as being similar to that of the medius.[1] Detailed analysis of the medius/minimus muscle complex, however, has been limited to the medius. Gluteus medius activity begins at the end of terminal swing (96% GC). Following IC, the intensity of this abductor muscle quickly increases, reaching peak intensity (28% MMT) by 6% GC. Muscle activity gradually subsides and ceases in mid stance (29% GC).

The upper gluteus maximus follows a similar pattern. Beginning in terminal swing (95% GC) the intensity rapidly rises to a peak during early loading response (26% MMT at 3% GC) and then slowly decreases until termination in mid stance (24% GC).

Tensor fascia lata muscle activity varies between its posterior and anterior portion. Posterior fiber action of moderate intensity (25% MMT) occurs at the onset of the loading response.[17] In contrast, the anterior fibers do not become active until terminal stance and the level of effort is lower (10% MMT).[9,13] Approximately half of the subjects tested in our laboratory (9 of 20) began their activity at the end of mid swing (87% GC) and continued into terminal stance (43% GC). Nine other subjects only had activity during SLS (28% to 40% GC). The remaining 2 subjects had no significant EMG.

Flexor Muscles

Normal persons walking at their preferred speed may display no significant flexor muscle action after initiating the first step (ie, less than 5% MMT). This was true for approximately half of the subjects studied. A change in velocity to either a faster or slower pace

introduced a consistent pattern of muscular effort, however. This is the model used to define the role of the hip flexor muscles in walking (Figure 6-5). The primary pattern of flexor muscle action begins in pre-swing and continues until early mid swing.

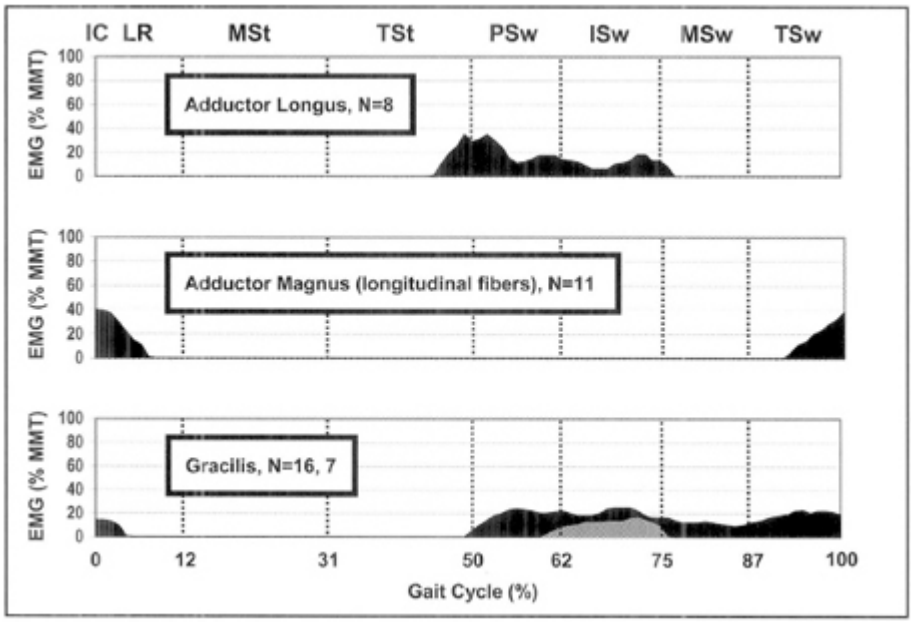

Figure 6-6. Hip adductor muscles. Normal mean intensity and timing during free walking (quantified electromyogram). Intensity as a percent of maximum manual muscle test value (% MMT) indicated by height of shaded area. The dark shading indicates the activity pattern of the majority of subjects. The light gray area indicates less frequent activity patterns. Vertical bars designate the gait phase divisions. N = samples included in data.

The adductor longus, with its onset in late terminal stance (46% GC), is the first hip flexor to become active. The muscle's activity peaks at the transition to pre-swing (35% MMT, 50% GC) and then remains active until initial swing (77% GC). Adductor brevis function is likely similar to that of adductor longus, but inability to confirm EMG isolation prevents verification of this assumption.

The gracilis, the second muscle to become active, is generally more persistent. Activity begins with the onset of pre-swing (50% GC), peaks during initial swing (25% MMT, 69% GC), and remains active until the beginning of loading response (4% GC). Approximately one-quarter (7 of 33) of the subjects tested in our

laboratory demonstrated only swing phase gracilis activity (63% to 78% GC).

RF activity is brief, inconsistent, and of low amplitude. The onset of action occurs in pre-swing (57% GC) and cessation follows shortly after in early initial swing (65% GC). While walking at a self-selected speed, only one-third of subjects (7/20) activated the muscle, and the activity was of low magnitude (peak of 18% MMT).

Similar periods of action are apparent for the sartorius (60% to 71% GC) and iliacus (63% to 74% GC). While the iliacus muscle has the largest cross-sectional area, its action is limited to initial swing. EMG studies of the psoas indicate its action during gait is similar to that of the iliacus.[6]

Adductor Muscles

Among the major adductor muscles, only the actions of the adductors longus, adductor magnus, and the gracilis can be defined by dynamic EMG (Figure 6-6). The actions of these muscles already have been described under their roles as hip flexors (adductor longus and gracilis) and extensor (adductor magnus).

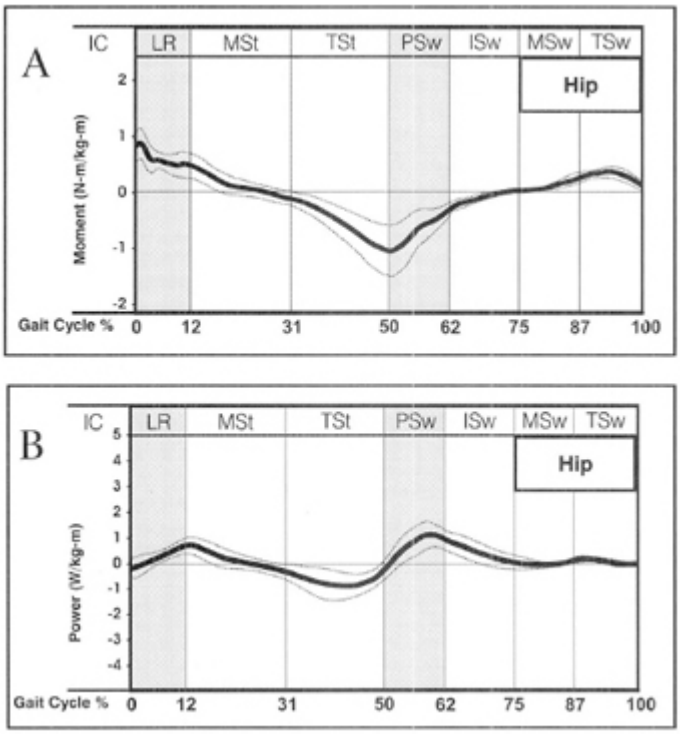

Figure 6-7. Hip joint forces. (A) Hip joint moments: Normal weight-bearing pattern generated by the sagittal vector during walking. The early high extensor moment (+) at the onset of stance quickly decreases, crossing the 0 line during mid stance, and becoming a flexor moment (-) during the remainder of stance and initial swing. During mid swing and the first half of terminal swing, the extensor moment gradually builds before diminishing. (B) Hip joint power demonstrating 2 periods of power generation (+), which contribute to forward progression.

FORCES

Sagittal Plane

As the trunk advances over the supporting foot, the weight line vector changes its relationship to the hip joint in both the sagittal and coronal planes. The resulting moments of both patterns are functionally significant (Figure 6-7).

With the hip flexed 20° at the time of IC, the body weight vector is significantly anterior to the hip joint center (Figure 6-8A). The impact that follows the abrupt drop of body weight on the foot necessitates an immediate peak in the extensor moment (0.84 N•m/kg at 2% GC, 6.9 BW/LL units,[19] or 35 Nm^3). As the initial inertia is replaced with the developing shear forces, the vector rapidly realigns itself toward the body's COM and moves backwards toward the hip (Figure 6-8B). Although the moment arm's length decreases throughout the remainder of loading response, a rapid rise in the magnitude of the GRF preserves the need for an extensor moment throughout WA. By the end of loading response, the extensor moment is half (0.44 N•m/kg) of its earlier peak. During the transition from loading response to mid stance, the first of two spikes in power generation occurs (0.72 W/kg•m at 12% GC), contributing to hip extension.

As the thigh progressively extends in mid stance (25% GC), the hip joint center moves in front of the body weight vector, contributing to a flexor moment (see Figure 6-7). During mid and terminal stance,

passive resistance from the Y ligament is the primary source of the resistance provided by the flexor moment. The flexor moment rises through terminal stance and peaks at the beginning of pre-swing (1.06 N•m/kg at 51% GC). As body weight shifts to the opposite limb, the flexor moment rapidly declines. While the flexor moment is declining, a second short burst of power is generated (peak of 1.14 W/kg•m at 60% GC) as the hip rapidly flexes due to low-level muscle activity (adductor longus, gracilis, sartorius, and RF). In the latter half of mid swing and terminal swing, a low-level extensor moment controls the rate and magnitude of thigh extension. This moment arises with the onset of hamstring muscle activity.

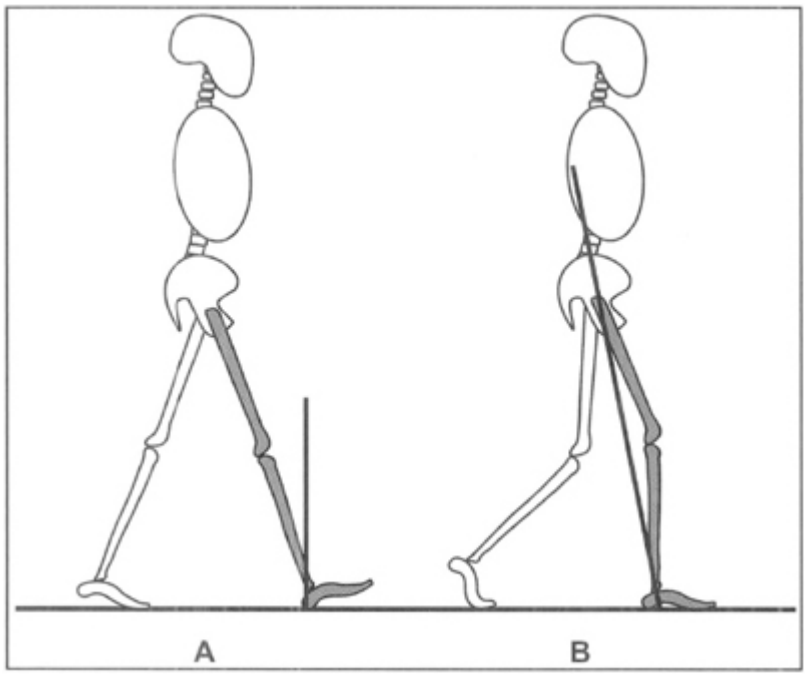

Figure 6-8. Hip vector alignment in early stance (vertical line). (A) IC. The anterior vector alignment signifies an extensor moment. Short line = low force. Distance from hip = long lever arm. (B) Loading response. Vector close to hip joint, extensor moment small.

Coronal Plane

With the impact of IC, the vector abruptly moves medial to the hip joint center, and an abductor moment (and abductor muscle action) is required to stabilize the hip. The stimulus is the abrupt unloading

of the trailing limb and drop of the inadequately supported body mass. Basically, the alignment of the vector is between the center of the foot and the midpoint of the pelvis. The magnitude of the abductor moment primarily reflects the amplitude of the ground reaction force vector during stance as there is little change in the moment lever length. Hence, its contour is similar to the sagittal force pattern (see Figure 3-29).

During loading response, the abrupt drop of the opposite side pelvis is accompanied by a period of power absorption that peaks at the end of loading response (peak of 0.75 W/kg•m at 9% GC).

FUNCTIONAL INTERPRETATION OF THE HIP

The three-dimensional demands imposed on the hip during stance as the trunk remains erect and the limb progresses over the foot are very dependent on its ball and socket contour. A high demand is placed on the hip muscles for control in the sagittal, frontal, and transverse planes during WA. This is followed by substituting passive forces for direct muscular effort as the limb moves into SLS.

The demand on hip musculature during limb advancement is less intense. Only the mass of the limb must be controlled. Again momentum is available to lessen direct muscular requirements. Consequently, the requirements of the hip are met by varying the interplay between motion and muscular control throughout the GC.

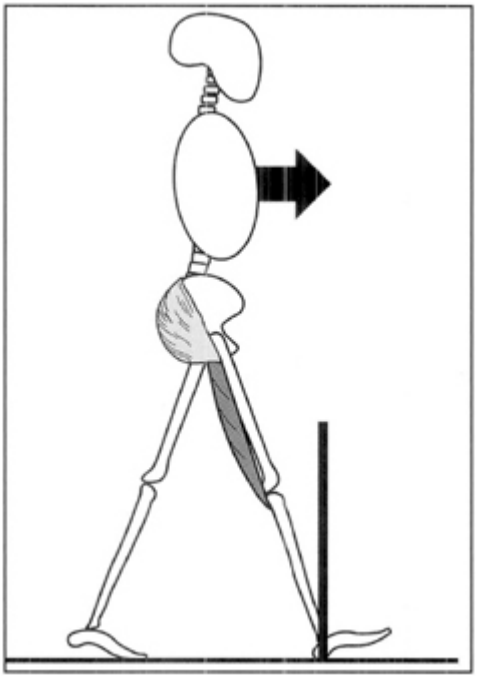

Figure 6-9. IC hip extensor muscle action restrains flexor momentum (arrow and vector line). The hamstring muscles and gluteus maximus are active.

INITIAL CONTACT (0% TO 2% GC)

Posture: Thigh maintained in 20° flexion (thigh forward)

Function: Thigh positioned optimally for forward progression and stability

The 20° flexed position of the thigh at the moment of initial floor contact represents an optimum compromise between the benefits of a long step length and the need to ensure stability and prevent the foot from slipping. Body weight is directed toward the ground while a significant element of progression is preserved. On a theoretical basis, having the limb in a 20° diagonal posture makes the potential vertical force greater than twice the shear component. Hence, the opportunity for weight-bearing stability on a nonslippery surface exceeds the probability of the limb sliding forward. Longer steps would increase the potential for slipping due to greater shear[5] and the demand on the hip extensors to maintain stability, while shorter steps would limit forward progression.[4]

At the moment of floor contact, however, the hip is in an unstable situation. Floor contact interrupts advancement of the limb, yet the trunk continues to move forward. This places the body vector well anterior of the hip joint at the instant of floor contact (Figure 6-9).

LOADING RESPONSE (2% TO 12% GC)

Posture: Thigh maintained in 20° flexion (thigh forward)
Function: Hip stability maintained and trunk flexion avoided

Weight-bearing stability during limb loading is maintained by the action of all 5 hip extensors, although they contribute at different intensities because of their varying effects at the knee (Figure 6-10). The lower gluteus maximus and adductor magnus provide the most direct response as they act only at the hip joint (ie, single joint muscles). Activity of the hamstrings is reduced because of the flexor effect at the knee. The more prolonged action of the medial hamstrings (semimembranosus, semitendinosus) suggests an internal rotation role to assist in advancing the contralateral pelvis. As a result of the interplay between hip and knee demands, activity increases in the gluteus maximus (25% MMT) and adductor magnus (40% MMT) during early loading response, while the hamstrings reduce their intensity.

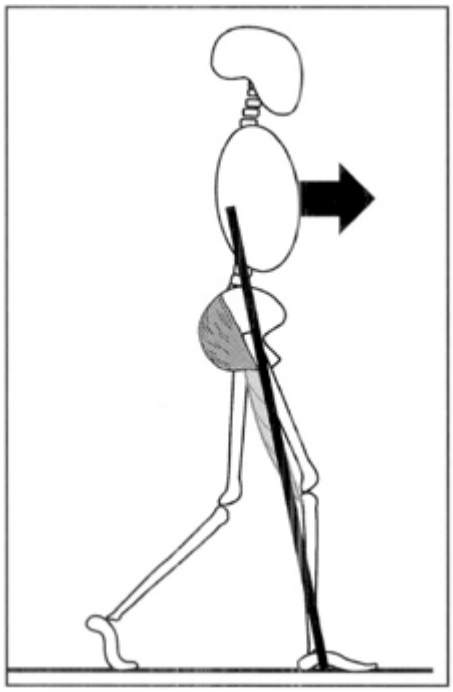

Figure 6-10. Loading response hip extensor action. Vector close to hip and posterior to the knee. When the hamstring action is reduced, the gluteus maximus activity increased.

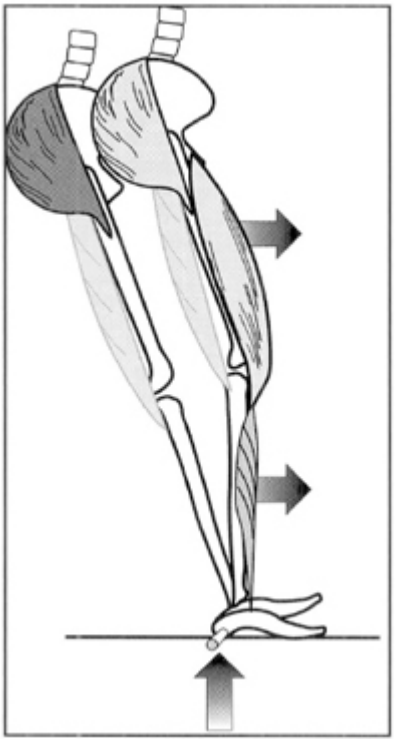

Figure 6-11. Loading response. Peak activity of the single joint hip extensors combines with low-level hamstring activity to provide the

necessary hip stability. The heel rocker advances the tibia and the quadriceps advances the thigh, extending the hip against inertia of the pelvis and trunk.

By the end of loading response, the single joint hip extensor muscles cease their activity. The vector is aligned closer to the hip due to flexion of the knee, resulting in a decreased hip extensor moment. Continued low amplitude (~15% MMT) activity of the medial hamstrings until the beginning of mid stance helps to preserve hip stability.

Passive hip extension follows the propagation of limb progression. The tibia is drawn forward by the heel rocker, and the femur is pulled forward by the quadriceps (Figure 6-11). As the vastii act to restrain knee flexion, their origin also pulls the femur forward with the advancing tibia. Inertia delays the advancement of the pelvis, further contributing to relative hip extension.

In the coronal plane, rapid transfer of body weight onto the loading limb demands active lateral stabilization of the pelvis over the hip. The base of the body vector shifts to the supporting foot while the controlling CG is in the midline of the body. Weight transfer also reduces the support for the contralateral side of the pelvis. The effect is a large medial (ie, abductor) moment at the hip that is necessary to control drop of the unsupported contralateral side of the pelvis (Figure 6-12). To stabilize the pelvis (and secondarily the trunk), the abductor muscles (gluteus medius, upper gluteus maximus, and posterior tensor fascia lata) react promptly and with considerable intensity (26% to 31% MMT). This is the interval of greatest abductor muscle action. The intensity of the response is stimulated by 2 situations: 1) speed with which the muscles must act and 2) a high mechanical demand (muscle moment arm is only 67% of body weight vector lever). The need for eccentric abductor muscle control is reflected by the burst of power absorption that occurs (peak of 0.75 W/kg•m at 9% GC).

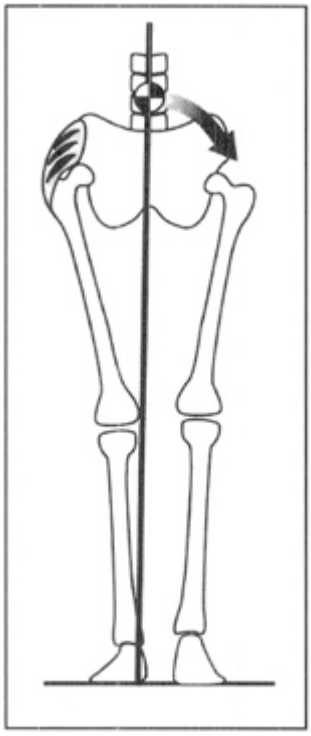

Figure 6-12. Coronal plane muscle control of the hip during loading response. Strong response by the hip abductor muscles to reduced support beneath the contralateral limb is associated with an ipsilateral abductor moment.

Internal rotation in the transverse plane is a third action being initiated during the loading response. There are 3 causes of rotation, two of which are due to the ipsilateral limb and one resulting from actions of the contralateral leg. Subtalar eversion allows the tibia to rotate internally while contralateral elastic recoil of the plantar flexors advances the opposite pelvis. Prolonged action of the ipsilateral medial hamstrings prolongs internal rotation. Hip rotation is decelerated by the external rotation force of the gluteus maximus muscle.

MID STANCE (12% TO 31% GC)

Motion: Thigh extension to neutral (0°)
Function: Forward progression of the HAT

As the limb rolls forward over the supporting foot during mid stance, the hip moves from its 20° flexed posture toward extension in the sagittal plane. This results from an interplay between the vastii and the 2-joint medial hamstrings (semimembranosus and semitendinosus). Continued activity of the vastii in early mid stance causes the knee to extend. As knee extension is initiated, the resulting tension in the hamstring muscles (approximately 15% MMT) contributes to the hip extending at the same time. A low amplitude power burst of the medial hamstrings at the initiation of mid stance (peak of 0.72 W/kg•m at 12% GC) augments hip extension. The extending hip advances in front of the sagittal plane vector by the end of mid stance and removes the need for further extensor muscle action (Figure 6-13).

Figure 6-13. Early mid stance. Activity of the vastii and medial hamstrings contributes to extension of the hip.

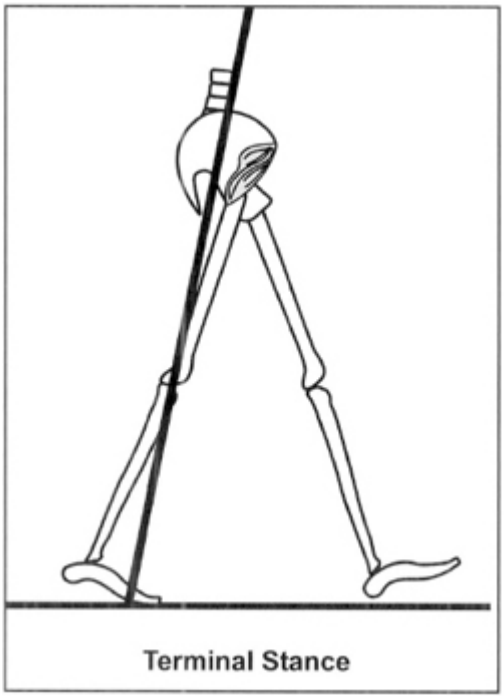

Figure 6-14. Terminal stance hip control does not require activity of the hip extensors. The tensor fascia lata stabilizes the pelvis in the frontal plane.

In the latter half of mid stance, the major abductor muscles have returned the pelvis to neutral alignment in the coronal plane. Little additional force is needed, thus, the gluteus medius and upper fibers of the gluteus maximus relax and the tensor fascia lata muscle becomes the dominant force (see Figure 6-4).

TERMINAL STANCE (31% TO 50% GC)

Motion:	Thigh extension to a trailing limb posture (20° apparent hyperextension)
Function:	Forward progression for maximum step length

As body weight rolls forward over the forefoot rocker, it pulls the limb into a trailing posture. With the pelvis and trunk remaining erect, the hip joint center moves farther anterior to the body vector and the thigh is pulled into apparent hyperextension (Figure 6-14). The anterior portion of the tensor fascia lata responds for 2 purposes. Its flexor role restrains the rate and extent of passive hip extension.

Also, this muscle action provides the low level of abduction force that continues to be needed. At the end of terminal stance, adductor longus activity is initiated and peaks. The flexor component of this muscle also restricts hip hyperextension. Additionally, this muscle restrains the lateral fall of body weight toward the contralateral limb.

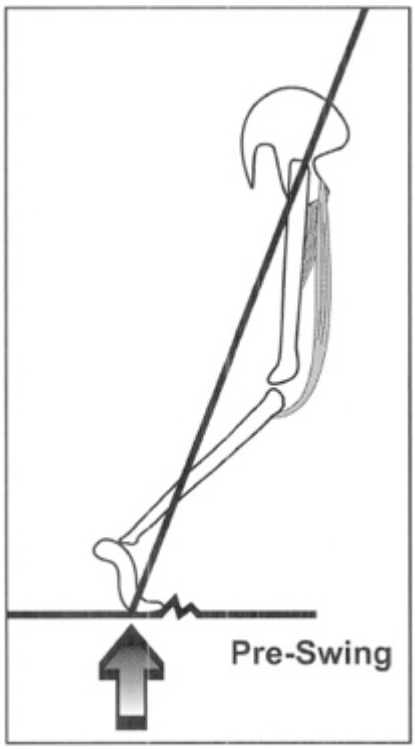

Figure 6-15. Pre-swing hip flexion being initiated by the adductor longus, gracilis, and RF (if active).

The low level of abductor muscle activity presents a bit of a paradox as the medial vector continues through the entire single support period. The answer probably lies in the body's lateral shift pattern. By the middle of mid stance (25% GC), the body's COG is at its most lateral point. Then it begins to retreat back toward the midline. This introduces passive abduction and correspondingly reduces the need for direct muscle action. Energy is conserved by the relaxation of the gluteus medius and upper gluteus maximus. The smaller tensor fascia lata is sufficient for continued support.

PRE-SWING (50% TO 62% GC)

Motion: Thigh advances to 10° apparent hyperextension

Function: Limb advancement

Hip flexion in pre-swing is a reaction to multiple events. First, the limb is advanced by the plantar flexor tendon recoil mechanics. While primarily inducing knee flexion, the toe rocker also carries the thigh forward. While the RF restrains knee motion, it contributes to hip flexion (Figure 6-15). A third thigh-advancing maneuver is a consequence of the flexor component of adductor longus and gracilis. The primary objective of these muscles is to decelerate the abduction caused by weight transfer to the other foot. Just prior to toe-off, activation of the sartorius, which has an external rotation and abduction component, balances the adduction and internal rotation forces of these adductors. The hip regains a neutral alignment through these various mechanisms. Because hip motion is rather rapidly reversed from extension into flexion, the pre-swing phase also has been called the *interval of limb acceleration*. A burst of power generation occurs in the sagittal plane as the hip quickly flexes.

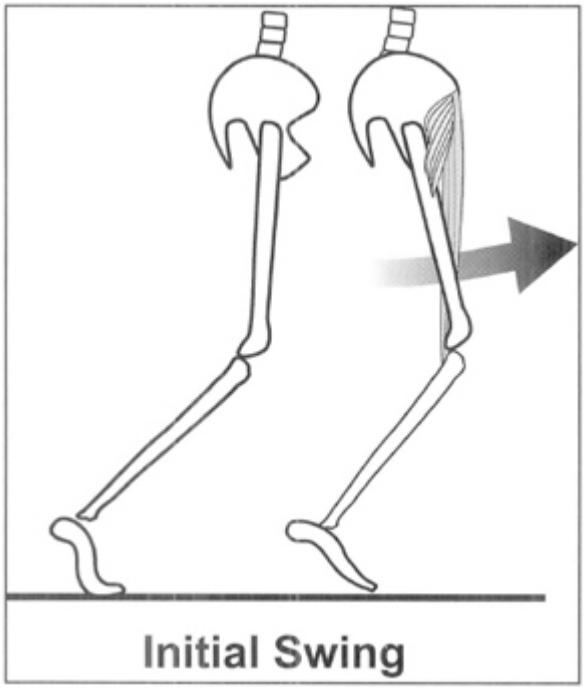

Figure 6-16. Initial swing hip control with flexion (arrow) being stimulated by the iliacus muscle and augmented by activity of the gracilis and sartorius.

Initial Swing (62% to 75% GC)

Motion: Thigh advances to 15° flexion
Function: Forward progression

Momentum generated in pre-swing continues into initial swing. At a normal free-walking speed, limb advancement may be passive as a result of the ankle's propulsive mechanics. This is supplemented by direct muscle action as needed. Being unloaded, the limb rapidly advances 20° within 0.1 sec (10% of the GC). Walking faster or slower stimulates dependence on the iliacus (Figure 6-16). Two muscles that commonly display peak activity in initial swing are the gracilis and sartorius. The gracilis provides adduction, internal rotation, and flexion. Simultaneous sartorius activity provides counter forces of abduction and external rotation as it assists in flexion. The final three-dimensional path of the limb in initial swing could represent the balance between these 2 muscles. The gracilis and sartorius also induce knee flexion as they act at the hip. Generally, this is a desirable synergy in initial swing. When tibial inertia causes excessive knee flexion, response of the RF preserves accelerated hip flexion while correcting the knee motion. Because of the varying needs for three-dimensional control of a dangling limb and the interactions of hip and knee motion, the pattern of hip flexor activity varies considerably among individuals.

Mid Swing (75% to 87% GC)

Motion: Thigh advancement peaks at 25° flexion
Function: Forward progression
Foot clearance

The limb advances an additional 10° by a continuation of the action occurring in initial swing. This is a period of virtually passive hip flexion. Recordable muscle action may be absent or minimal and occur among any of the flexors (see Figures 5-17 and 6-5). Momentum persisting from the initial flexor effort is the prime mover.

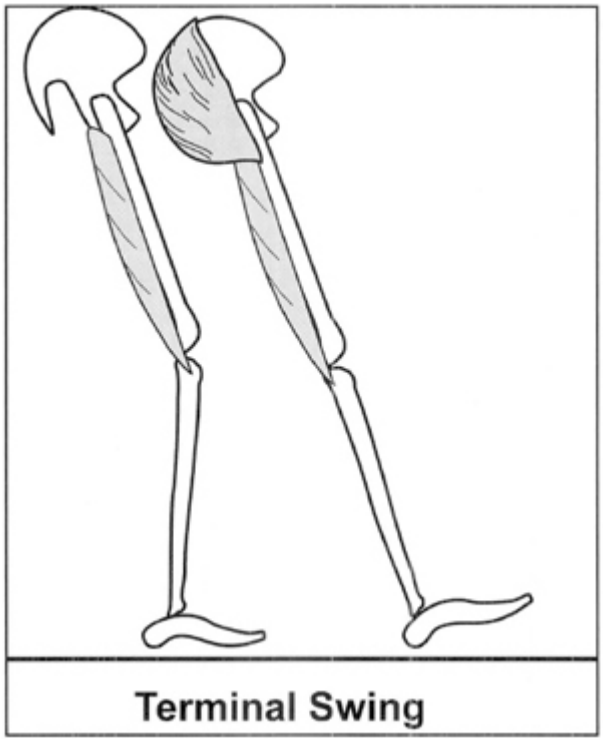

Figure 6-17. Terminal swing cessation of hip flexion by peak hamstring muscle activity. Onset of gluteus maximus and adductor magnus at the end of the phase prepares for WA demands.

TERMINAL SWING (87% TO 100% GC)

Motion: Subtle past retract to 20° flexion

Function: Limb positioned for stable heel first IC

Terminal swing is the transitional phase between swing and stance. Hip muscle action at this time prepares the limb for stance by stopping further flexion. Strong action by the hamstring muscles is the controlling force. Imbalance in the size of the muscles probably is responsible for the mild internal rotation that occurs. All 3 hamstrings participate at a moderate intensity (Figure 6-17). The medial muscle mass (semimembranosus and semitendinosus) is approximately 50% larger than the long head of the biceps.[20] Preferential use of the hamstring muscles rather than the single joint hip extensors (gluteus maximus and adductor magnus) indicates the advantage of simultaneous deceleration at the knee. Otherwise, the thigh would be restrained while the shank was free to respond to the existing

momentum and quadriceps pull. The reduction of hamstring muscle activity at the end of terminal swing and accompanying onset of the gluteus maximus and adductor magnus indicates a need for hip extension control with less impact on the knee.

The onset of gluteus medius action in terminal swing counteracts the earlier adducting influences of the hip flexors. As a result of these many actions, the limb is optimally positioned for IC and the beginning of another weight-bearing interval.

CONCLUSION

Hip motion during the stance period allows the pelvis and trunk to remain erect while the limb rolls forward over the supporting foot. The hip extensor muscles have 2 functions. First, they decelerate the limb's momentum in terminal swing to prepare for stance. Secondly, they act to restrain the forward momentum in the pelvis and trunk as the limb is loaded. The abductor muscles counteract contralateral pelvic drop induced by the medial alignment of body weight. During swing, the hip flexors advance the limb, but the demand is low.

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Chapter 7

Head, Trunk, and Pelvis

The axial core of the body consists of 3 rigid structures (head, thorax, and pelvis) separated by 2 mobile areas (cervical and thoracolumbar spines). Functionally, the head and neck (cervical spine) are considered one unit resting on top of the trunk. The definition of trunk is inconsistent. The word may refer to all body segments between the base of the neck and the hip joints (except the arms) or represent just the lumbar and thoracic segments. This latter interpretation is more useful for gait analysis because the thoracolumbar trunk and the pelvis have different functional obligations, leading to dissimilar motion patterns. The lumbosacral joint separates the lumbar spine from the sacrum and pelvis.

HEAD, TRUNK, AND PELVIS GAIT DYNAMICS

Motion

While the neck allows the head to move independently to expand one's field of vision, the head and trunk travel as a unit during normal gait. Neither segment displays any observable change in position except that both the head and trunk move up and down as the body's COG follows the mechanics of the limbs. Instrumented analysis, however, has registered small arcs of displacement in both the sagittal and coronal planes. Minor arcs of motion also occur at the pelvis.

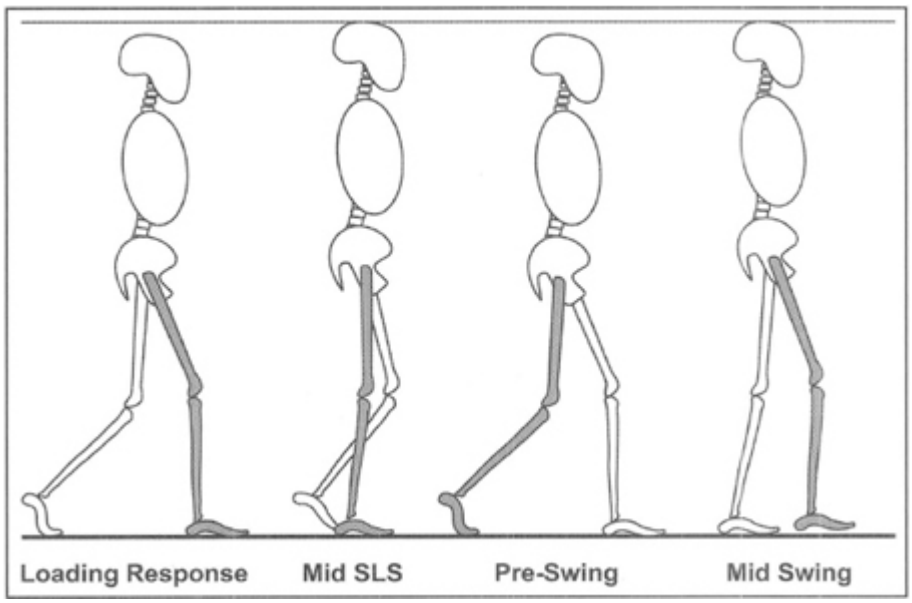

Figure 7-1. Vertical displacement of the trunk during stride indicated by head height. It is lowest in double support (loading response and pre-swing) and highest in mid SLS and mid swing.

Total Axial Displacement

Throughout the GC, the HAT deviates from the mean line of progression in all 3 planes (vertical, lateral, and progressional). Each displacement pattern is a sinusoidal curve, but the individual characteristics differ for the 3 directions of motion.

Vertical displacement of the sacrum, trunk, and head is equal for each segment and follows a double sinusoidal path. The average amount of vertical change is approximately 4.2 cm.[10,13] There are 2 cycles of downward and upward displacement in each stride (Figure 7-1). These reflect the mechanics of the right and left steps. Peak downward deviation occurs in loading response (10% GC) and in pre-swing (60% GC) while walking on a treadmill (73 m/min). Both of these phases are periods of double limb stance. Each descent is followed by a progressive rise above the mean level. These occur in the 2 SLS intervals, the transition from mid to terminal stance (34% GC), and late mid swing (84% GC). The amount of displacement varies with the subject's walking speed, with greater amplitudes occurring at faster velocities. Thorstensson et al reported that the

amount of vertical trunk excursion varied from 2.7 cm at 90 m/min to 6.8 cm at 150 m/min.[12]

The rate of vertical displacement also varies. While walking on level surfaces at a comfortable speed (66 m/min), rapid upward acceleration peaks (0.36 gravity) during each period of double limb stance (5% and 55% GC) and then quickly dissipates. A minor and brief second wave follows (10% and 60% GC). Subsequently, there is a relatively consistent downward acceleration that is maximal (0.28 gravity) during periods of SLS for each limb (35% and 85% GC).

Lateral displacement also is the same for all of the axial segments, averaging 4.5 cm for the total arc between maximum right and left deviations. In the lateral direction, however, the path is a single sinusoid for each GC (Figure 7-2).[13] From an initial neutral point at the onset of stance, the axial segments reach maximum displacement over the stance limb at the 31% point in the GC (ie, the onset of terminal stance). This is followed with a gradual return to neutral (50% GC). The head, trunk, and sacrum then deviate toward the other side. Maximum contralateral displacement occurs during mid swing (81% GC), correlating with contralateral terminal stance.

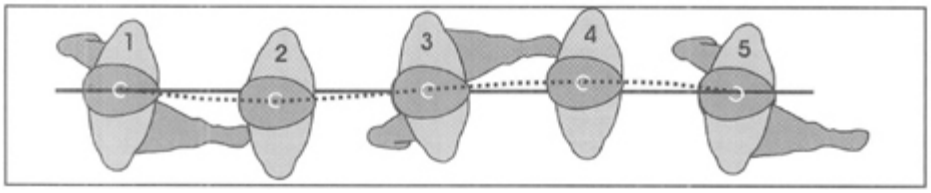

Figure 7-2. Lateral displacement of the trunk during stride indicated by head location. It is midline during double limb support (figures 1, 3, and 5), displacement to right with right single stance (figure 2), and displacement to left with left single stance (figure 4).

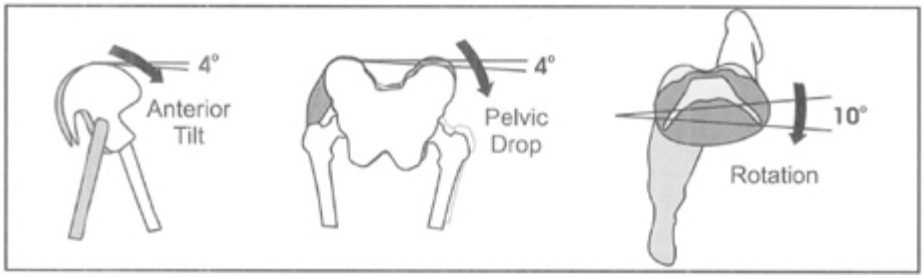

Figure 7-3. Motions of the pelvis during walking: Anterior tilt (4°), contralateral pelvic drop (4°), and transverse rotation (10°).

The overall amplitude of progressional displacement (anterior-posterior) of the axial segments (HAT) during each GC is greatest at the sacrum, intermediate at the thorax, and least at the head.[13] When compared to a fast speed (97 m/min), walking slowly (49 m/min) causes greater displacement of the head (0.9 versus 0.2 cm), thorax (2.4 versus 1.5 cm), and sacrum (3.5 versus 2.3 cm) in the plane of progression.[13]

Progressional displacement shows a relationship to gait velocity that follows a double sinusoidal curve during treadmill walking. During the first third of each step cycle, the axial segments advance faster than the mean gait velocity. The average maximum increase in forward velocity is greatest for the sacrum (23 cm/sec), moderate for the thorax (T10 = 14 cm/sec), and least at the head (3 cm/sec). The difference between HAT advancement and average progression is maximal at the 15% and 55% points in the stride. In contrast, the axial segments advance more slowly than the mean walking velocity at the end of each limb's SLS period (maximum slowing occurs at the 45% and 95% GC points). The average peak decrease in forward segment velocity is 15 cm/sec at the sacrum, 8 cm/sec at the thorax, and 2 cm/sec for the head.

Progressional accelerations are attenuated across the spinal segments, with the peak accelerations experienced by the head being approximately one-tenth of those experienced by the hip.[9] Minimization of HAT progressional accelerations is accomplished, in part, by activation of the paraspinal muscles in a superior to inferior pattern between the cervical and lumbar regions.[9] The attenuation of accelerations at the head aids in providing a stable visual frame of reference during walking.[6,9]

Pelvis

The pelvis moves asynchronously in all 3 directions during each stride. The site of action is the supporting hip joint. All of the motion

arcs are small, representing a continuum of postural change (Figure 7-3).

In the sagittal plane, the pelvis has an anatomical 10° anterior pelvic tilt due to the anterior superior iliac spine being lower than the posterior superior iliac spine.[8] Observationally, the pelvis appears neutral. During walking, an additional 4° pelvic tilt occurs,[5,7] which is hard to observe, yet one can sense the mobility during gait. A relative tipping of the pelvis in the posterior direction (pubic symphysis up) occurs early in SLS as the trunk assumes an erect posture over the supporting limb, and again during initial swing as the contralateral limb enters its early SLS period. Conversely, the pelvis tilts anteriorly (pubic symphysis down) during terminal swing as the trunk leans forward toward the support surface and again in terminal stance as the limb achieves maximum trailing limb posture.

During WA, the contralateral side of the pelvis drops an average of 4° in the coronal plane when the demands on the loading limb exceed the forces generated by the abductor muscles (see Figure 7-3).[10] In pre-swing, the ipsilateral pelvis drops 4° as the contralateral limb's abductors yield under the high demands of loading response.[10]

In the transverse plane, the pelvis rotates through a total arc of 10° (5° forward and 5° backward; see Figure 7-3).[7,10] Maximum forward rotation (5°) occurs during terminal swing and IC and contributes to step length of the lead limb. Maximum backward rotation (5°) happens during terminal stance, contributing to a trailing limb posture. Mid stance and mid swing are the phases of transition when the pelvis rotates through neutral (0°).

Motion at the junctions of the pelvic bones and sacrum (sacroiliac joint) has not been noted during walking. The larger effort of sitting up from a reclining position may involve 0.5 cm of sacroiliac motion.[15] This is an inconstant finding, however, as fusion of part or all of the sacroiliac joint is common. Mobility of the symphysis pubis has not been studied. It is assumed to allow minute degrees of rotation and translation.[15]

Forces

During quiet standing, alignment of the body vector passes approximately 1 cm anterior to the ear hole.[1] It can be assumed this also is true during free walking as the head only deviates 2% from the mean rate of progress. Further analysis of the body vector location relates to the pelvis.

Sagittal Plane

The precise relationships between the body vector and the center of the pelvis have not been defined. Gross analysis of mean visible vector records indicates the following pattern.

At the time of IC, the floor impact vector lies anterior to the pelvis. The center of the pelvis rapidly moves anterior toward the vector during loading response. Gradual anterior displacement continues through the rest of stance. The vector is in the area of the sacrum by pre-swing.

Coronal Plane

The body vector lies in the mid line of the pelvis throughout the GC. Two brief exceptions occur at the onset of loading response and pre-swing when there is a momentary deviation laterally toward the hip joint being loaded.[2]

Muscle Control

Stabilization of one vertebral segment on the other within the spine is provided by the musculature contained within the trunk. Gross alignment is accomplished by the long muscles arising from the pelvis. Basic stability of the HAT unit depends on muscular control of the pelvis at the hip.

Pelvis

Two muscle groups, the abductors and extensors of the hip, are the primary source of pelvic control. The low intensity of trunk muscle action does not appear to affect the motion of the pelvis.

Action of the hip abductor muscles is the only type of pelvis control that has been specifically identified (see Figure 6-4). The upper gluteus maximus and gluteus medius have a similar pattern of action.[4] Onset is in late terminal swing (upper gluteus maximus at 95% GC; gluteus medius at 96% GC) and continues into the middle of mid stance (24% and 29% GC). The intensity of both muscles rapidly rises during early loading response (3% to 7% GC) and reaches peak intensities of 26% to 28% MMT. Activity of both muscles slowly subsides until they relax in mid stance.

Trunk

Stabilization of the normally aligned trunk requires minimal muscular action during quiet standing. Walking introduces phasic muscle activity. The lumbar and thoracic components of the erector spinae act synchronously.[11,14] Their prime activity occurs at the time of contralateral IC (ie, peak action is at 50% GC of the ipsilateral reference stride). The muscles begin their activity in late terminal stance (40% GC), peak at 50% GC, and continue through pre-swing (this timing is related to the contralateral limb's phases of terminal swing and loading response). Ipsilateral action is less intense. Beginning in terminal swing (90% GC), a low peak action (10% MMT) occurs during loading response (5% GC) or mid stance (15% GC).

The large lumbar intrinsic group (multifidus) is bilaterally active at the time of each heel strike (right and left). Ipsilateral peak activity tended to be greater than the contralateral effort (30% versus 20% MMT).[11] Action of rotatores and the quadratus lumborum is similar to that of the multifidi.[13] The activity of these localized muscles coincides with the loading phase of both the ipsilateral (90% to 12% GC) and contralateral limbs (45% to 62% GC).

The abdominal muscles have 2 patterns of action. Activity of the external oblique muscles is an intermittent, low intensity (5% MMT) pattern throughout stance. Peak action at a 10% MMT intensity occurs during late mid swing and early terminal swing (75% to 90% GC). The rectus abdominis has a low level of continuous action.

Study of the trunk muscles during treadmill walking showed a similar pattern of extensor muscle action. Abdominal muscle activity, however, was reversed, with the rectus abdominis being more phasic. This may represent a response to the extensor thrust of the moving platform.[14]

FUNCTIONAL INTERPRETATION OF THE HEAD, PELVIS, AND TRUNK

During walking, displacement and acceleration of the axial segments (head, trunk, and pelvis) reflect the action of the limbs in swing and stance. Consequently, the greatest amount of motion occurs at the pelvis. Two mechanisms are in effect, the impact of limb loading and the weight of the contralateral swinging limb. Motion in the plane of progression is stimulated by the change in momentum induced by foot floor contact and the height of the HAT COM. Pelvic motion is initiated by the base of the trunk mass (sacroiliac joint) being eccentric to the center of the supporting hip joints. Movement of the pelvis is restrained by the hip muscles, while the back and abdominal musculature control the alignment of the trunk over the pelvis. Activity of the erector spinae and intrinsic muscles during limb loading and later action of the abdominal muscles decelerates the passive forces reflected to the trunk.

The muscles serve 2 functions, shock absorption and preservation of upright trunk stability. With only 5 lumbar segments available to dissipate the sacral motion compared to 17 intervertebral joints in the thoracic and cervical segments to preserve head neutrality, it is

evident that the major dynamic effects of walking are experienced by the lumbar spine.

Initial Contact (0% to 2% GC)

At the onset of stance, the pelvis observationally appears level in both the sagittal and coronal planes. Transversely, the pelvis is rotated forward about 5°.[3]

Loading Response (2% to 12% GC)

Loading the limb introduces postural changes in all 3 directions. WA by the limb is accompanied by forward displacement of the sacrum (S2), which is twice as great as that occurring at the trunk (T10). The difference in acceleration is 73%. Further intervertebral deceleration removes virtually all progressional effects on the head.[13]

Simultaneous unloading of the opposite limb removes the support for that side of the pelvis, leading to a rapid contralateral pelvic drop. When the focus is on the ipsilateral limb, the motion is described as relative elevation of that side of the pelvis because unloading the opposite limb has allowed the mid point of the pelvis to drop. Lateral drop of the contralateral side of the pelvis during loading response is the motion of concern. The rapid drop of the pelvis is decelerated by the hip abductor muscles (gluteus medius and upper gluteus maximus).

The back muscles respond to both of these events. Bilateral intrinsic extensor (multifidus and rotatores) and quadratus lumborum action decelerates the forward displacement of the trunk. Activity of the erector spinae (large, extrinsic extensors) is greater on the contralateral side in response to the pelvic drop. The ipsilateral erector spinae action contributes to decelerating trunk progression. With sacral displacement exceeding that of the trunk, the result is slight lumbar extension associated with a minor forward lean.

Transfer of body weight to the stance limb frees the unloaded side of the pelvis to begin rotating forward in the transverse plane. Prolonged action of the stance limb's semimembranosus and semitendinosus enhances pelvic rotation. External rotation incidental to extensor stabilization of the hip by the gluteus maximus and biceps femoris provides an antagonistic force.

Mid Stance (12% to 31% GC)

Vertical and anterior displacement of the trunk return to neutral while lateral displacement continues. Transverse rotation and lateral tilt of the pelvis also return to neutral by the middle of mid stance. The trunk muscles are quiet. By the end of this phase, the HAT is maximally displaced toward the supporting limb. The pelvis begins to reverse its transverse alignment during the latter half of mid stance.

Terminal Stance (31% to 50% GC)

Progression of the sacrum and other axial segments now is slower than the mean walking speed. At the same time there is increasing forward acceleration of the supporting limb throughout this phase, which reaches its peak at the end of SLS. The accelerating mechanism is forward roll of the supporting limb over the forefoot rocker.

At the onset of terminal stance the supporting limb is vertical. This, combined with heel rise, results in peak elevation of the body axis in early terminal stance (34% GC). Further forward roll of the limb lowers the sacrum, trunk, and head. Inertia delays the response of the axial segments. This introduces a relative hyperextension effect and anterior tilt of the pelvis. The trunk is stabilized by the flexor action of the rectus abdominus. Reduced participation of the oblique abdominal muscles is consistent with their less effective flexor action.

Pre-Swing (50% to 62% GC)

Total axis motion is the same as that occurring in loading response except for the fact that it reflects the effects of the contralateral limb. In this second double support period, the head, trunk, and sacrum again descend to their lowest level. As the limb is unloaded, the pelvis on that side rapidly tilts below the body line, creating an ipsilateral drop of 4°. The pelvis is also released to rotate forward in the sagittal plane.

Initial Swing and Mid Swing (62% to 87% GC)

This is a quiet transition period comparable to contralateral mid stance. Transversely, the pelvis regains its neutrally aligned posture. Both posterior tilt (symphysis up) and forward rotation begin.

Terminal Swing (87% to 100% GC)

At the onset of this phase, the axial segments are at their highest level. This represents the terminal stance posture of the contralateral limb. A progressive drop from this level then follows. As the pelvis continues advancing the swinging limb, the ipsilateral drop persists and a 3° anterior tilt (symphysis down) develops. The pelvis also is maximally rotated forward (5°) in the transverse plane.

CONCLUSION

Movement of the head, neck, trunk, and pelvis is secondary to the function of the lower limbs. The significant events are the impact of loading, the changing alignment of the stance and swing limbs, and the loss of bilateral support for the pelvis. Action by the trunk and hip muscles decelerates the imposed forces. As a result, all motions are

small. Also, both the magnitude and acceleration of displacement are least at the head.

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Chapter 8

Arm

Reciprocal arm swing spontaneously occurs during walking. Elftman calculated the angular momentum of the arm swing in the 3 functional planes and found the pattern was opposite to that of the rest of the body.[2] He concluded that this allowed the lower legs to perform their necessary motion without imparting marked rotation to the body. The significance of this calculation is challenged by the results of energy cost analysis. Subjects walking with their arms free to swing and with them bound were tested and showed no differences in oxygen usage.[7] These 2 findings suggest arm swing may be useful but that it is not an essential component of walking.

GAIT MECHANICS

Motion

During each stride, the arms reciprocally flex and extend (Figure 8-1). Timing between the 2 arms is offset by 50% in the cycle, with peak extension of each arm occurring during ipsilateral heel contact and peak flexion happening with contralateral IC.[5] During the period from IC until the initiation of pre-swing (50% GC), the ipsilateral shoulder and elbow progressively flex. Then, with the onset of pre-swing, the shoulder and elbow begin to extend at a rate similar to that observed during the previous period of flexion.[5] Considerable variation exists among individuals in the amount of flexion and

extension used. Faster walking increases the total arc of motion used at both the shoulder and elbow.[5]

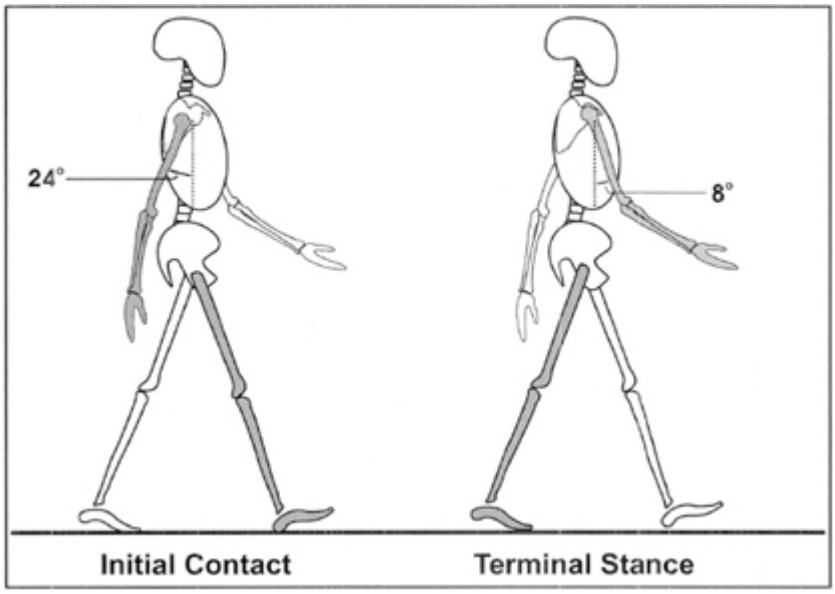

Figure 8-1. Arm swing during normal free walking. IC (maximum backward swing). Terminal stance (maximum forward swing).

Shoulder

The shoulder moves through an average arc of motion of 32° during moderate speed walking (92 m/min), although quite a bit of variability exists among individuals. At the onset of stance, the shoulder is positioned in maximum extension (24°) and then flexes to a position of 8° flexion by the end of terminal stance when the other limb is about to make contact with the ground (Figure 8-2).[5] After holding this position of peak flexion momentarily, the shoulder then extends throughout the swing phases.

Although the relative timing of peak shoulder motions remains consistent as walking speed increases, the total arc of motion is greater with faster velocities.[5] During walks at a rapid speed of 128 m/min, the average arc of motion is 39°. At this rapid velocity, maximum shoulder extension increases (peak of 31°) while shoulder flexion remains unchanged (peak of 8°) compared to the motion occurring while walking at 92 m/min.

Elbow

The pattern of elbow excursion during walking is similar to that observed for the shoulder, with a flexion arc in stance and an extension arc in swing.[5] During moderate and fast speed walking, the magnitudes of the arcs are comparable to those occurring in the shoulder (30° and 40° elbow flexion, respectively); however, the elbow remains in flexion throughout the GC. As a result, the maximum flexion position is greater at the time of contralateral floor contact (47° at 92 m/min; 55° at 128 m/min; see Figure 8-2).

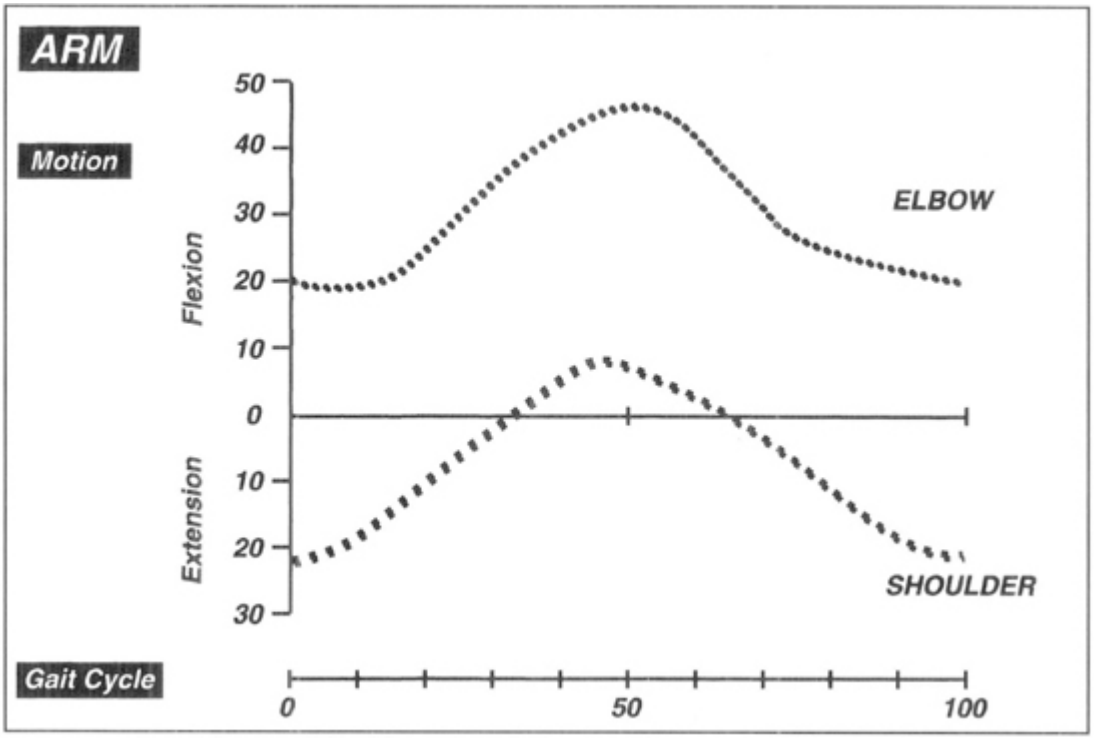

Figure 8-2. Arcs of elbow and shoulder motion during arm swing while walking. Horizontal scale indicates % GC beginning with IC (0). (Adapted from Murray MP, Sepic SB, Barnard EJ. Patterns of sagittal rotation of the upper limbs in walking. *Phys Ther.* 1967;47:272-284.)

Phasing

The coupling of motion between the shoulder and elbow is quite distinct during walking.[5] This portion of the text focuses on the

pattern observed at a moderate walking speed (92 m/min). The ipsilateral arm at IC is maximally extended at both the shoulder and elbow, while the leg is reaching forward with a flexed hip. Following a brief delay (5% GC), the shoulder progressively flexes as the hip extends. There is a longer delay in the onset of elbow flexion. Movement of the elbow toward greater flexion begins in mid stance, which may relate to the maximally extended position being 17° of flexion. Maximum shoulder flexion (8°) and elbow flexion (47°) are reached near the end of terminal stance (50% GC). Contralateral foot/floor contact at the onset of pre-swing stimulates both the shoulder and elbow to reverse their motion toward extension. These motions continue throughout the swing period until ipsilateral heel contact, when a maximally extended posture is achieved at both the shoulder (24° extension) and elbow (17° flexion). The phasic correlation of peak arm motion with floor contact by the ipsilateral or contralateral foot is very consistent, with the majority of the subjects exhibiting less than a 0.1-second deviation.

Muscle Control

The activation pattern of shoulder and arm muscles during walking has received only limited study.[3,4,8] The timing of muscle activity varies across subjects.[8] Additionally, the amplitude of muscle activity in the arms increases as subjects walk faster.[4]

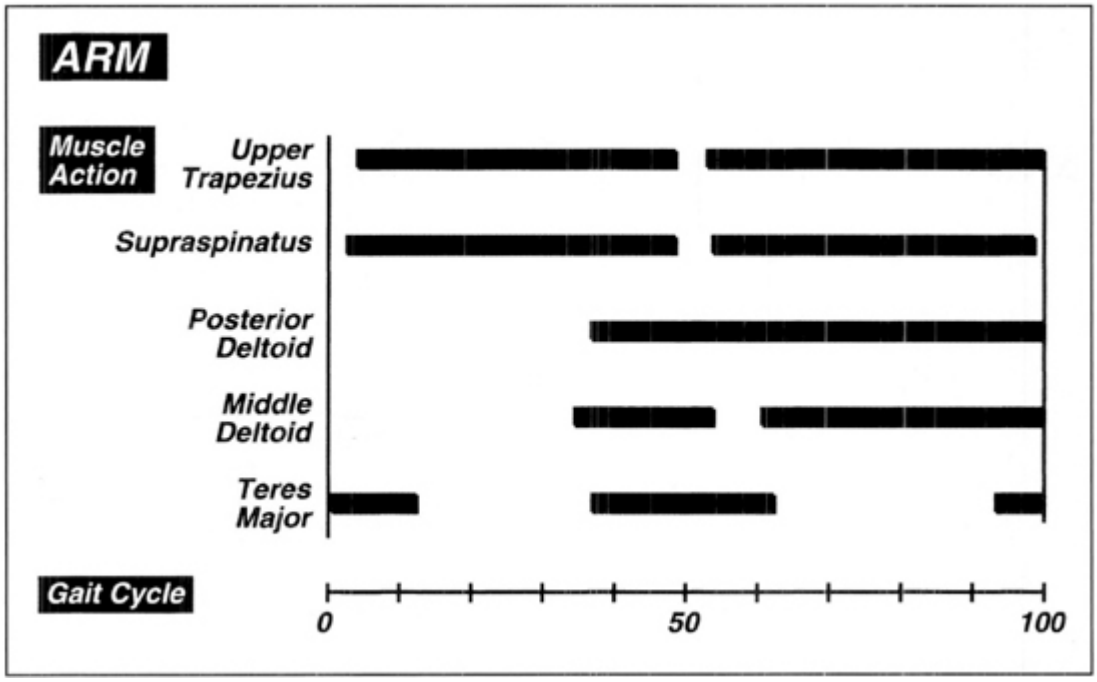

Figure 8-3. Timing of the muscles related to arm swing during walking. Horizontal distance is one GC beginning with IC. (Adapted from Fernandez-Ballesteros ML, Buchthal F, Rosenfalck P. The pattern of muscular activity during the arm swing of natural walking. *Acta Physiol Scand*. 1965;63:296-310.)

The most extensive study of muscle activity in the arm and shoulder revealed activity in only 6 of the 12 muscles recorded.[3] Judging from this study, the supraspinatus and upper trapezius are the most active. Their onset is just after IC and the action continues until the end of terminal swing with only scattered short periods of rest (Figure 8-3).

Middle and posterior deltoid action are nearly synchronous. Their activity begins just before maximum flexion of the shoulder and elbow are achieved (ie, contralateral heel contact). This synergy continues throughout the period of shoulder and arm extension (ie, until ipsilateral heel contact). These 2 muscles are silent during the rest of the stride.

The upper latissimus dorsi and teres major complex exerts 2 bursts of activity. Both occur during periods of double limb stance. The first occurs just prior to ipsilateral heel contact when the shoulder is maximally extended and continues through loading

response. The second occurs just prior to contralateral heel contact when the shoulder is maximally flexed and continues through ipsilateral pre-swing.

None of the other muscles participate in swinging the arm during walking (anterior deltoid, infraspinatus, sternal, and clavicular heads of the pectoralis major, rhomboids, biceps, and triceps).[3,4]

FUNCTIONAL INTERPRETATION OF THE ARMS

The shoulder presents 3 functional patterns during walking: flexion, extension, and support of a dependent arm. Each has a specific pattern of muscular activity.

Support of the arm is provided at both the scapula and the humerus. The upper trapezius actively supports the scapula. Significant EMG in quiet standing has been demonstrated. Humeral support is the role of the supraspinatus.[1] The horizontal alignment of the supraspinatus allows it to draw the humeral head into the socket as it also lifts the humerus.[6]

Extension of the shoulder and deceleration of flexion are dynamic events under distinct control by the posterior deltoid (and teres major). Simultaneous timing of the middle deltoid activity contributes better abduction of the arm, so it will clear the body while following the pull of the extensor muscles. There is a momentary break in activity of the middle deltoid, supraspinatus, and upper trapezius during the period of abrupt transfer of body weight to the opposite limb.

In contrast, the flexion component of arm swing during walking appears to be purely passive. Currently, there is no evidence of flexor muscle activity (anterior deltoid, clavicular pectoralis major, or biceps).[3] One can only speculate on the role of the coracobrachialis, as its actions during walking have not been studied.

The role of arm swing is indicated by the timing of the active component. Dynamic arm extension occurs at the same time the leg

is swinging forward. Each extremity (arm and leg) also moves through a comparable arc (hip 40°, shoulder 32°). Hence, the arm is providing a purposeful counterforce to minimize the rotatory displacement of the body by the locomotor mechanics of the legs, just as Elftman calculated.[2] Actively holding the arm back at the beginning of limb loading (the first 5% of the GC) may be a second deliberate, dynamic stabilizing maneuver.

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Chapter 9

Total Limb Function and Bilateral Synergistic Relationships

The multiple phases of gait represent the serial patterns of motion and muscle control used at the trunk, hip, knee, ankle, and foot to advance the body. During the stance phases, the synergy of actions dissipates the abrupt forces of loading, preserves stability, and maintains forward progression. In swing, the coupling of events ensures foot clearance while also maximizing progression. In the preceding chapters, function at each joint has been detailed. Now, it is appropriate to integrate these actions into a concept of total limb function across the functional tasks of WA, SLS, and SLA. Joint positions constantly change during the GC. In the text that follows, characteristic positions for each joint during the specific phase have been listed. In most cases, values have been rounded to the nearest 5°. Those seeking greater detail may refer to Appendix A for more precise joint position data.

TOTAL LIMB FUNCTION ACROSS THE GAIT CYCLE

Weight Acceptance

During WA, body weight is rapidly transferred from the trailing leg to the outstretched forward limb. Shock arising from the abrupt loading of the limb must be dissipated, while stability and forward progression are maintained. WA occurs during the first 12% of each GC and includes 2 phases: 1) IC and 2) loading response.

Initial Contact (0% to 2% GC)

Positions:	Pelvis: 10° anterior tilt, 5° forward rotation, neutral (0°) frontal plane
	Thigh: 20° flexion
	Knee: 5° flexion (observationally appears extended)
	Ankle: neutral
	ST: neutral
Critical Events:	Heel first contact
	Heel rocker initiated
	Impact decelerated

At the instant the foot strikes the ground, the limb is optimally positioned to absorb some of the shock of floor contact while preserving progression and postural stability. The ankle and ST joints are in neutral alignment, the knee is near full extension (5° flexion), and the hip has approximately 20° of thigh flexion (Figure 9-1). This places the heel closest to the floor.

IC is an intense event as the transfer of body weight to the forward limb is preceded by a 1-cm fall before the heel reaches the floor.[11] The impact of the abrupt foot-to-floor contact generates a spike in the vertical component of the GRF (commonly called the “heel transient”), which reaches an intensity of 50% to 125% of body weight within the first 1% to 2% of the cycle (Figure 9-2).[13-15] The alignment of the GRFV relative to the joints introduces instability of the ankle, ST, and hip joints, while knee stability is enhanced.

At the ankle, the vector is posterior to the joint with its base at the heel. The impact force initiates rapid ankle PF. A prompt increase in the activity level of the pretibial muscles decelerates the motion. Peak intensity of the anterior tibialis reaches 37% MMT during the first 1% GC. At the same time, secondary peaks in activity occur for

the EHL (32% MMT) and EDL (26% MMT). Power absorption increases due to the eccentric activity of the pretibial muscles. The impact vector generates a small arc of eversion at the ST joint, which is controlled primarily by the anterior tibialis muscle while the posterior tibialis is just becoming active. The combined action at the ankle and ST joints provides the initial shock-absorbing response and lessens the impact of the GRF.

At the knee, the vector is anterior to the joint. With the knee already in full extension, the added extensor effect of the impact vector preserves knee stability by opposing the flexion effect of the heel rocker. Hyperextension is avoided by the knee flexion action of the hamstrings (12% to 27% MMT). The flexor moment (0.35 N•m/kg•m) and the power generated (1.0 W/kg•m) at the time of impact, however, are small.

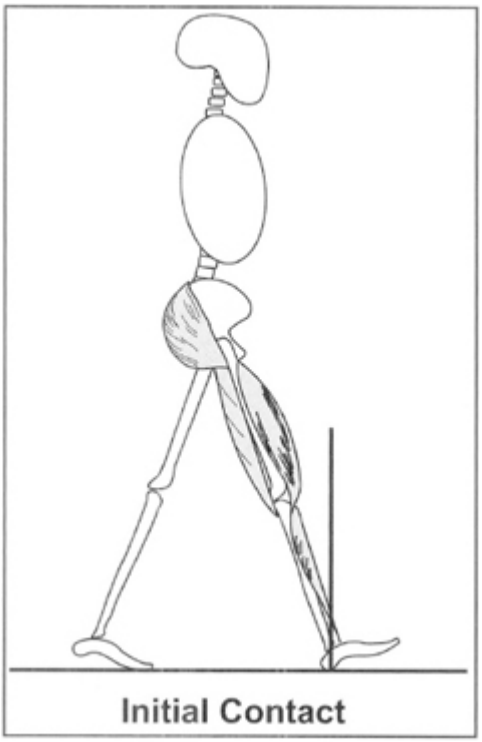

Figure 9-1. IC: Heel contact with hip flexed, knee extended, and ankle at neutral. Vector anterior to the hip and knee and posterior to the ankle joint. Activity of gluteus maximus, quadriceps, hamstrings, and pretibial muscles.

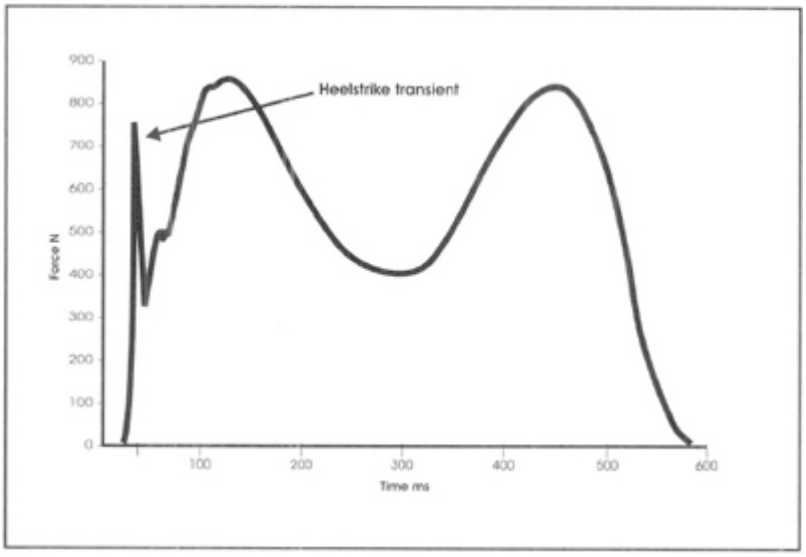

Figure 9-2. Vertical component of the GRF demonstrating a spike in amplitude shortly after heel contact.

The hip, with the thigh in 20° of flexion, is potentially unstable due to the anterior location of the impact vector. Stability is preserved through active support by both the lower gluteus maximus (24% MMT) and adductor magnus (40% MMT) as primary extensors plus added force by the hamstring group (semimembranosus, 27% MMT; semitendinosus, 19% MMT; long head of the biceps femoris, 12%). The resulting extensor moment, however, has only moderate intensity (0.84 N•m/kg•m).

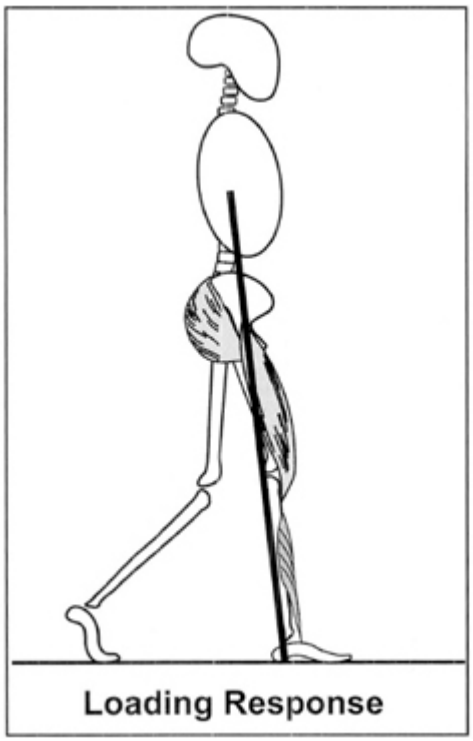

Figure 9-3. Loading response: Heel rocker contributing to forward progression. Hip and knee flexion. Vector anterior to hip, posterior to knee, and at ankle. Activity of the gluteus maximus, quadriceps, and pretibial muscles.

Loading Response (2% to 12% GC)

Positions:	Pelvis: 10° anterior tilt, 5° forward rotation, 5° contralateral drop
	Thigh: 20° flexion
	Knee: 20° flexion
	Ankle: 5° PF then neutral
	ST: 5° eversion
Critical Events:	Heel rocker progression
	Restrained ankle PF
	Restrained knee flexion
	Sustained hip flexion

As the load on the limb increases, forward progression, shock absorption, and stability remain critical components of a successful WA period. At the ankle, the heel rocker serves as a key mechanism for enhancing forward progression. The knee becomes the primary

source of shock absorption. The hip provides a stable interface between the passenger unit (HAT) and the locomotor unit.

Sagittal plane progression follows the heel rocker (Figure 9-3). The intense activity of the anterior tibialis, EDL, and EHL limits the foot drop to 5° by 6% of the GC. The reversal of movement from ankle PF to ankle DF helps preserve the heel rocker. The source of this change in direction is the reduction of heel lever length as the vector advances toward the ankle joint. Pretibial muscle action delays forefoot contact until the tibia attains a vertical alignment at 12% of the GC.

Knee flexion follows the pull of the pretibial muscles. As the foot rolls forward on the heel, the contracting TA and long toe extensors (which originate from the tibia and fibula create a taut link between the shank and foot. As the tibia rapidly moves forward, the knee axis becomes anterior to the body weight vector and initiates knee flexion. Other factors that favor knee flexion are thigh inertia (which delays advancement of the femur), prolonged action of the medial hamstrings, and the gravitational effect of trunk weight on the proximal end of the flexed femur.

As the knee joint axis moves ahead of the body vector, the vastii increase their activity to control the rate and amount of knee flexion. The peak intensity of the quadriceps ranges from 21% to 38% MMT across the 4 vastii heads during this period, and the action is eccentric. Peak knee flexion is limited to 20° by the end of the loading response (12% GC). This is sufficient knee flexion to absorb the shock of limb impact without excessively challenging the vastii in their efforts to ensure that stability is preserved. The RF is generally not active during this phase as its hip flexion action would add to the demands on the hip extensors.[12]

In addition to restraining knee flexion, the quadriceps produces an anterior shear force, which is resisted passively by the anterior cruciate ligament. Simultaneous hamstring muscle activity resists the anterior drawer. All 3 hamstrings are active in early stance, but the medial hamstrings (semimembranosus and semitendinosus) are dominant.

While the ankle and knee are undergoing rapid changes in position during loading response, the hip remains relatively stable

(20° thigh flexion) in the sagittal plane. The anterior location of the body vector relative to the hip joint center necessitates prompt action by the gluteus maximus (25% MMT) and adductor magnus (37% MMT) to prevent further flexion. Low-level hamstring activity (9% to 23% MMT) also provides assistance. Anterior tilt of the pelvis is resisted by brief activity of the abdominal muscles.

Coronal plane demands at the hip and knee relate to the strong abductor moment that follows the rapid transfer of body weight onto the forward limb. The stimulus is the reduced support of the trailing side of the pelvis. At the hip, contralateral pelvic drop is limited to 5° by the strong response of the ipsilateral hip abductors (gluteus medius, upper gluteus maximus, and tensor fascia lata), which peak in activity during this phase. Calculations of the abductor muscle force supporting the pelvis average 1.5 times body weight (range 1.02 to 1.8 body weight).[6,9,10] This protective moment is greater at the hip than the knee because the lever arm between the body vector and the joint line is longer.

The controlling abductor moment at the knee is provided by a combination of passive tension in the IT band and dynamic action of both the gluteus maximus and tensor fascia lata muscles, which insert into the band. There is the possibility of further dynamic lateral protection of the knee during the period of BFLH action, but this muscle usually ceases its activity during the first half of WA.

The lateral location of the calcaneus relative to the weight-bearing axis of the tibia and body vector contributes to the need for an invertor moment to control the rate of ST eversion. Anterior tibialis and posterior tibialis function eccentrically to restrain the motion to a peak of 5° of eversion.

Transverse plane rotation relates to events at both the foot and hip. The primary effect of ST eversion is internal rotation of the talus. As the ankle mortise follows the talus, a more orthogonal alignment of the ankle axis to the path of progression is gained. Internal rotation of the tibia relative to the femur assists in “unlocking” the knee. While the rotational force arising from activity of the medial hamstrings augments internal rotation of the tibia relative to the femur, the availability of IT band tension and action of the BFLH

provides a counter force. Consequently, little transverse motion actually occurs between the tibia and femur.

ST eversion also unlocks the MT joint. The axes of the CC and TN joints become more parallel, creating a more flexible midfoot capable of adapting to the support surface.

Transverse rotation at the hip generally is identified as pelvic rotation. The presence of a dynamic transverse rotational action at the hip is implied by the difference in the duration of the medial and lateral hamstrings. Activity of the semimembranosus and semitendinosus persists throughout loading response (and into mid stance), while the long head of the biceps femoris ceases shortly after IC. The resulting imbalance toward internal rotation would assist advancement of the other limb in its pre-swing phase.

Figure 9-4. Mid stance ankle rocker: Early interval has tibia vertical with knee and hip flexed. Vector minimally displaced from joint centers. The gastrocnemius and soleus provide support at the ankle while the knee has brief support from the vastii. The hip is stabilized via the extensor component of the gluteus medius and upper gluteus maximus. The late interval has the body over the forefoot with continued heel contact. The ankle is dorsiflexed with the hip and

knee extended. The calf muscles provide primary support for the limb.

Single Limb Support

During SLS, the passenger unit (HAT) progresses over a single stable limb. The contralateral limb is in swing. Forward progression must be allowed while stability is maintained. SLS follows WA, occupies 38% of each GC, and includes 2 phases: 1) mid stance and 2) terminal stance.

Mid Stance (12% to 31% GC)

Positions:	Pelvis: 10° anterior tilt, neutral (0°) rotation, neutral (0°) frontal plane
	Thigh: Extension to neutral (0°)
	Knee: 5° flexion (observationally appears extended)
	Ankle: 5° DF
	ST: Decreasing eversion
Critical Events:	Restrained ankle DF (ankle rocker)
	Knee extension
	Coronal plane hip stabilization

Contralateral toe-off signifies the transfer of full body weight to the forward limb and the beginning of SLS. The foot is now flat on the ground (contact by both the heel and forefoot) and the tibia vertical, but there is still some flexion at the hip and knee (Figure 9-4). Progression of the limb is continued by DF of the tibia over the articular surface of the talus, also referred to as the ankle rocker. As the limb rolls forward, the critical site for dynamic limb stability shifts from the knee to the ankle. The intense muscle action at the hip and knee that was present during loading response rapidly terminates by early mid stance. Limb stability becomes dependent on the actions of the soleus, augmented by the gastrocnemius.

The significant factors contributing to the change in muscular requirements are progressional momentum and vector alignment.

Momentum from the contralateral swinging limb and residuals from the heel rocker draw the ankle into DF. As the ankle dorsiflexes, the body vector moves anterior to the ankle and knee axes and posterior to the hip axis.

The anterior alignment of the body vector relative to the ankle joint provides a strong stimulus for the gastrocnemius and soleus to stabilize the tibia. The soleus increases its intensity to approximately 30% MMT and maintains this level of activity until the end of the phase nears, while the gastrocnemius demonstrates a more progressive increase in intensity through the same period. Appropriately modulated eccentric activity of the soleus and gastrocnemius permits controlled forward progression and prevents tibial collapse.

At the knee, anterior alignment of the vector changes the mode of the quadriceps to concentric action to assist extension. This generates a small burst of positive power (0.49 W/kg•m at 16% GC) as the vastii contribute to progression by pulling the femur forward over the stable tibia. The quadriceps ceases in early mid stance.

The hip continually reduces its flexed posture, moving from an initial 20° of thigh flexion to neutral by 27% GC. This is aided by a modest power burst by the medial hamstrings at the beginning of mid stance (0.72 W/kg•m at 12% GC). Otherwise, active hip extension is limited to continuing activity of the posterior gluteus medius. Hip extension also gains indirectly from the quadriceps' pull on the femur and displacement of the vector posterior to the hip joint. The timing of this latter event depends on the relative verticality of the trunk over the pelvis.

In the coronal plane, tensor fascia lata, gluteus medius, and upper gluteus maximus action stabilizes the pelvis in a level posture, which provides an appropriate base for an upright alignment of the trunk. Transverse rotation of the pelvis is now at neutral as the trunk maintains its alignment over the vertical lower limb.

Terminal Stance (31% to 50% GC)

Positions:	Pelvis: 10° anterior tilt, 5° backward rotation, neutral (0°) frontal plane

	Thigh: Extension to 20° apparent hyperextension Knee: 5° flexion (observationally appears extended) Ankle: 10° DF with heel-off ST: Eversion decreases to 2° by end of phase
Critical Events:	Controlled ankle DF with heel rise (forefoot rocker) Trailing limb posture Free forward fall of the body

As the body rolls forward over the forefoot, the ankle dorsiflexes and the heel rises from the ground, signifying the onset of terminal stance and the initiation of the forefoot rocker (Figure 9-5). The knee completes its extension arc and the thigh reaches a trailing limb alignment. Trunk advancement moves the body vector to its most anterior alignment relative to the ankle and to the limits of metatarsal head support. During most of the terminal stance (31% to 47% GC), the EMG activity of the soleus and gastrocnemius increases as these muscles counter the growing plantar flexor moment created by the falling body mass. Their function is to lock the ankle so that the forefoot serves as the point of rotation. The heel rise that accompanies the forward tilt of the tibia preserves the height of the COG. The plantar flexor moment peaks (1.40 N•m/kg•m at 47% GC) just before the other foot contacts the ground. Recent research using ultrasound indicates that the 5° of ankle DF that occurs during this phase arises from tendinous stretch while the fascicle portion of the muscle appears to act isometrically.[1,3,5,7,8]

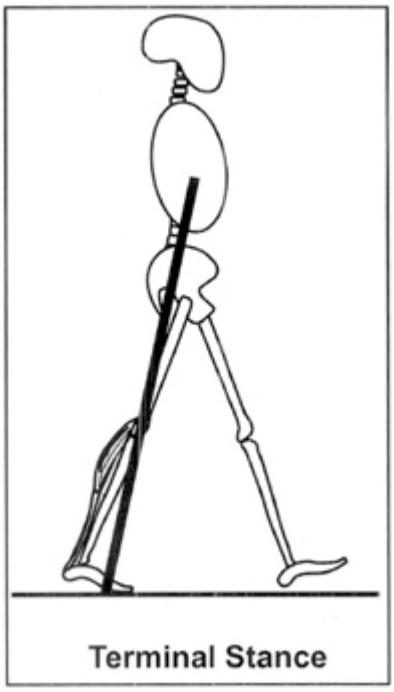

Figure 9-5. Terminal stance: Forefoot rocker with heel rise, ankle DF, and knee and hip extension. Vector anterior to knee and ankle, posterior to hip. Soleus and gastrocnemius are the active extensors.

Seven plantar flexor muscles peak in activity during terminal stance, contributing to the high plantar flexor moment: soleus, gastrocnemius, flexor digitorum longus, flexor hallucis longus, posterior tibialis, peroneus longus, and peroneus brevis. Dynamic stabilization of the ankle is an essential element of heel rise. Extensor stability at the knee and hip is gained passively from the tibial restraint, which preserves the extensor alignment of the vector.

Stability of the foot, ankle, and knee is lost as the end of terminal stance approaches, leading to a rapid decline in the plantar flexor moment. Knee flexion becomes more rapid as tibial advancement moves the knee joint anterior to the force vector. Terminal stance ends as the other limb preserves upright balance by initiating floor contact.

Swing Limb Advancement

Advancing the swing limb involves unloading weight from the trailing leg, lifting the foot from the ground, and moving the limb forward to complete the stride and prepare for the next GC. Finely synchronized muscle actions and joint motions ensure energy-efficient foot clearance and forward progression during the 4 phases: pre-swing, initial swing, mid swing, and terminal swing.

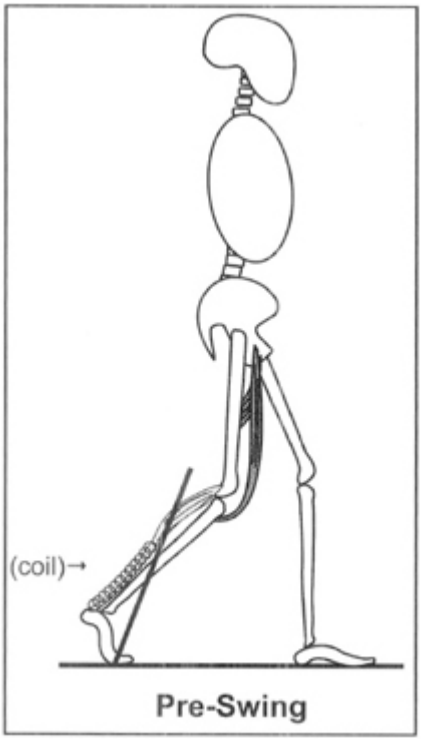

Figure 9-6. Pre-swing: Elastic recoil of the calf muscles advances the tibia over the toe rocker. Metatarsophalangeal joints dorsiflexed, ankle plantar flexed, knee flexed, hip less hyperextended. Vector at MTP joint, posterior to knee, magnitude reduced. The accessory hip flexors are activated.

Pre-Swing (50% to 62% GC)

Positions:	Pelvis: 10° anterior tilt, 5° backward rotation, 5° ipsilateral drop
	Thigh: 10° apparent hyperextension
	Knee: 40° flexion
	Ankle: 15° PF
	ST: Neutral (0°)
Critical Events:	Toe rocker

Knee flexion to 40°

As the trailing limb maintains contact of the metatarsal heads and toes with the supporting surface, it allows the toe rocker to advance the limb (Figure 9-6). Progression of the vector to the MTP joint and unloading of the limb by the abrupt transfer of body weight to the forward limb frees the foot to roll into a high heel rise (4 cm off the ground).[11] The plantar flexor moment that had peaked during the final half of terminal stance, rapidly declines, paralleling the unloading pattern of the trailing limb. Heel rise and tibial advancement are accelerated by elastic recoil of the previously tense plantar flexor musculotendinous unit. This generates a strong power burst (3.7 W/kg•m at 54% GC) that plantar flexes the ankle 15° and advances the tibia forward. The plantar flexed joint position in combination with the heel rise assists in maintaining limb length and reducing the ipsilateral pelvic drop to only 5°.

As the tibia advances, the knee rotates into 40° flexion in large part due to passive forces and, at a minor level, from muscular control by the popliteus, gracilis, and sartorius. If knee flexion threatens to become excessive, the RF responds. This restrains the knee while assisting hip flexion. Advancement of the thigh (hip flexion) is also assisted by the flexor action of the adductor longus and gracilis muscles as they resist the passive abduction caused by weight transfer to the other leg. Activation of the sartorius just prior to toe-off provides an additional source of dynamic hip flexion as well as an abduction and external rotation force to balance the adduction and internal rotation components of the adductors.

Two-thirds of the knee flexion range used in initial swing is attained during this final phase of stance. The actions occurring during pre-swing commonly are called *push-off* and it has been assumed that the body is driven forward. More accurately, this is “limb push-off” with the elastic recoil providing the force that advances the limb in swing. Recent investigators find only minor influence on body progression.[4]

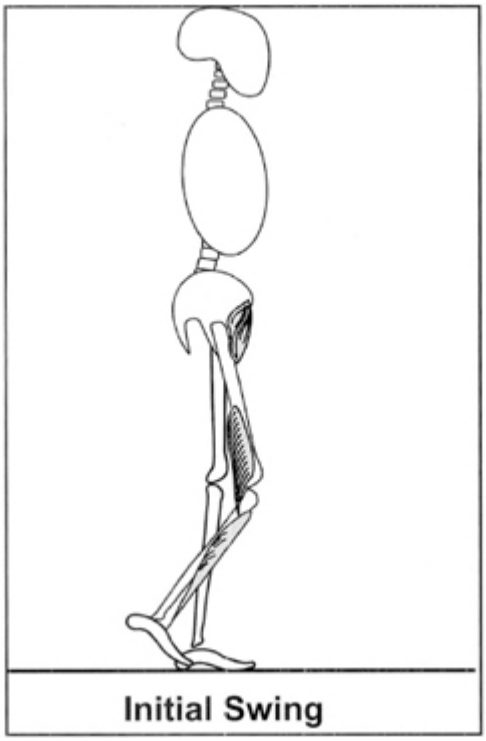

Figure 9-7. Initial swing: Hip and knee flexion increases and ankle plantar flexion reduces. The iliacus and accessory hip flexors, short head of biceps, and pretibial muscles are active.

Initial Swing (62% to 75% GC)

Positions:	Pelvis: 10° anterior tilt, 5° backward rotation, neutral (0°) frontal plane
	Thigh: 15° flexion
	Knee: 60° flexion
	Ankle: 5° PF
	ST: Neutral (0°)
Critical Events:	Knee flexion
	Hip flexion

Lifting of the toes from the ground signals the advancement of the unloaded limb. Knee flexion increases to 60° to raise the foot above the ground. The ankle only reduces its PF to 5° by the end of the phase. Hip flexion advances the thigh to 15° of flexion (Figure 9-7). Foot clearance of the floor is dependent on adequate knee flexion rather than ankle position because the trailing posture of the limb

spontaneously places the foot in a toe-down posture. Rapid advancement of the thigh contributes a significant propelling force.

Hip and knee muscle action during initial swing is variable. The most consistent knee flexor is the BFSH. Because the 2 heads of the biceps share the same tendon, this action often is erroneously attributed to the lateral hamstring (BFLH). Such action, however, would inhibit hip flexion as the long head of the biceps also is a hip extensor. Fine wire EMG recordings clearly differentiate the action of the 2 biceps muscles. Combined hip and knee flexion may be gained by low levels of sartorius or gracilis activity. Independent hip flexion is regularly provided by the iliacus when the pace is fast or slow. This muscle is frequently inactive at the person's customary walking speed. Low-level activity of the adductor longus further augments hip flexion.

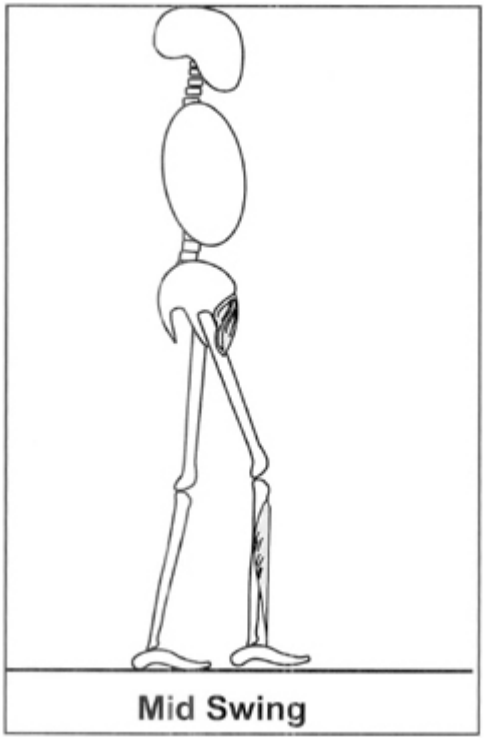

Figure 9-8. Mid swing: Increased hip flexion, reduced knee flexion, tibia vertical, ankle at neutral. Reduced activity of hip flexors and ankle dorsiflexors.

Pretibial muscle action (TA and the long toe extensors) is brisk during initial swing as the muscles begin lifting the foot and toes, with

EDL and EHL experiencing a peak in activity during this phase. The limited motion that is accomplished reflects the inertia that must be overcome.

Mid Swing (75% to 87% GC)

Positions:	Pelvis: 10° anterior tilt, neutral transverse and frontal plane rotation
	Thigh: 25° flexion
	Knee: 25° flexion
	Ankle: Neutral (0°)
	ST: Neutral (0°)
Critical Events:	Ankle DF
	Hip flexion

Limb advancement continues, but the muscle activity is only minimal. Floor clearance is now dependent on ankle and hip position. Active control of the ankle enables the foot to clear the floor. Muscle control at the ankle is a low-intensity continuation of the previously brisk action by the TA, EHL, and EDL. Only minimal activity of the adductor longus and gracilis is present as the thigh achieves 25° of flexion (Figure 9-8). Knee extension is purely passive. At the end of mid swing, the hamstrings begin their action in preparation for terminal swing.

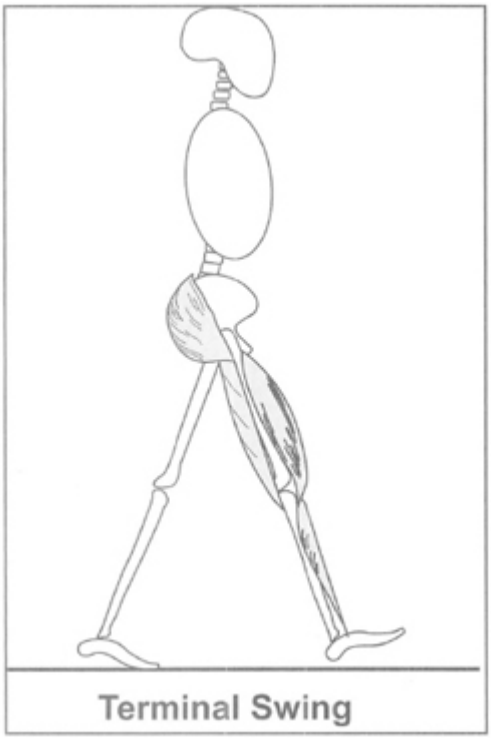

Figure 9-9. Terminal swing: Hip flexed, knee extended, ankle at neutral. Single joint hip extensors, hamstrings, quadriceps, and pretibial muscles active.

Terminal Swing (87% to 100% GC)

Positions:	Pelvis: 10° anterior tilt, 5° forward rotation, neutral (0°) frontal plane
	Thigh: 20° flexion (subtle past retract)
	Knee: 5° flexion (observationally appears extended)
	Ankle: Neutral (0°)
	ST: Neutral (0°)
Critical Events:	Hip deceleration
	Further hip flexion (ie, thigh advancement) is inhibited
	Knee deceleration
	Knee extension
	Ankle DF

This phase prepares the limb for the upcoming demands of IC. Advancement of the thigh is restrained, causing a subtle past retract (backward movement of the thigh from 25° to 20° of flexion). The knee smoothly extends to neutral (0° to 5° flexion) (Figure 9-9). The ankle is maintained at neutral (or may drop into 5° PF).

Muscle activity occurs at all 3 joints. The 3 hamstrings (semimembranosus, semitendinosus, and BFLH) contract with moderate intensity (22% to 38% MMT) during terminal swing to restrain hip flexion. This is their period of peak activity. Their simultaneous knee flexor action avoids excessive hyperextension arising from tibial momentum. By the latter half of terminal swing, the quadriceps (vastii) become active to ensure full knee extension and in preparation for the high-intensity demands of WA. Activity of the pretibial muscles also is brisk to ensure continued ankle DF. As a result of this combination of muscle action, the limb is optimally poised for the onset of weight bearing as the next IC occurs.

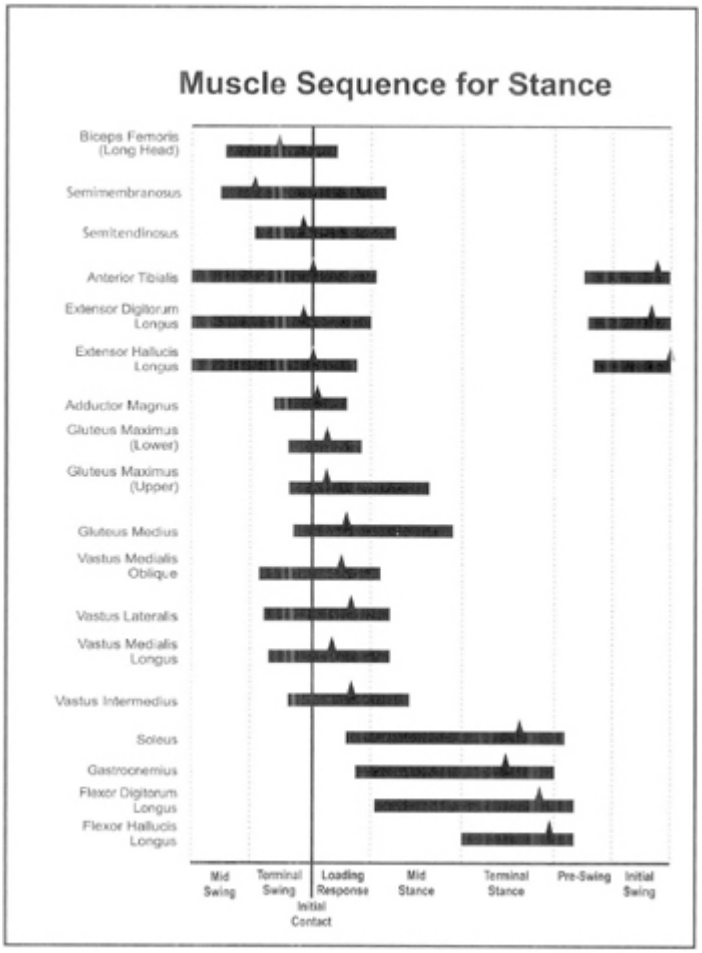

Figure 9-10. Muscle sequence for stance.

SUMMARY

Walking involves a repetitive sequence of motions dynamically controlled by muscles. The pattern of muscle activation varies throughout the GC, ensuring that the basic accomplishments of shock absorption, stability, foot clearance, and forward progression are achieved. Modulation of the intensity of muscle activation refines the control. The next section provides a summary of muscle control during the stance and swing phases, as well as a focused review of foot control.

STANCE MUSCLE CONTROL PATTERN

During the stance phases of gait, the controlling muscles are dedicated to providing weight-bearing stability, shock absorption, and progression over the supporting foot in a manner that conserves energy. With one exception, the responsible muscles are the extensors of the limb. The extensor muscles follow a dedicated sequence of action that begins in terminal swing and continues through terminal stance (Figure 9-10 and Table 9-1). Three functional synergies are performed: 1) swing-to-stance transition (terminal swing), 2) WA (IC, loading response), and 3) progression over the supporting foot (mid stance, terminal stance, pre-swing).

Table 9-1

Muscle Sequence for Stance (% Gait Cycle)

Muscle	On	Off	Peak
Anterior tibialis	56	13	0
Semimembranosus	81	15	88
Biceps femoris long head	82	5	93
Semitendinosus	88	17	98
Vastus medialis oblique	89	14	6
Vastus lateralis	90	16	8
Vastus medialis longus	91	16	4
Vastus intermedius	95	20	8
Adductor magnus	92	7	1
Gluteus maximus, lower	95	10	3
Gluteus maximus, upper	95	24	3
Gluteus medius	96	29	7
Posterior tibialis	0	50	44
Soleus	7	52	43
Gastrocnemius	9	50	40
Flexor digitorum longus	13	54	47
Peroneus longus	15	51	41
Peroneus brevis	20	55	46
Flexor hallucis longus	31	54	49

Terminal Swing

The 3 hamstrings (semimembranosus, semitendinosus, and BFLH) follow their activation in mid and terminal swing with a rapid rise to peak intensity in terminal swing. The accompanying change in motion is deceleration of hip flexion and knee extension. This prepares the limb for stance by limiting the thigh position to 20° flexion and prevents knee hyperextension. In late swing, the hamstrings reduce the intensity of their activity so that excessive knee flexion is avoided at the end of the phase.

In preparation for stance, 3 other muscle groups begin their activity in late terminal swing. The 2 single joint hip extensors (adductor magnus and the lower fibers of the gluteus maximus) become active as hamstring activity diminishes. By crossing only the hip joint, these muscles continue deceleration of the femur without having a flexor influence on the knee. Activation of the 4 vastii (lateralis, intermedius, medialis longus, and medialis oblique) counteracts the flexor effect of the hamstrings to ensure optimal knee extension for IC. Increased activity of the pretibial muscles (TA and long toe extensors) positions the foot for the pending heel rocker action.

Initial Contact and Loading Response

With floor contact, action of the single joint hip extensors (adductor magnus and lower gluteus maximus) rapidly reaches peak intensity and this level of effort continues into early loading response. Through their insertion on the femur, the adductor magnus and lower gluteus maximus impart an extensor effect on the knee as well as the hip. The hip abductor muscles (upper gluteus maximus and gluteus medius) enhance femoral stability as they respond to the contralateral drop of the pelvis. Also, the upper gluteus maximus has a direct extensor action on the knee through its insertion into the IT band.

TA action reaches its peak intensity immediately following heel contact to eccentrically restrain the rate of ankle PF. To augment this

action, EHL and EDL have a secondary peak in intensity during a similar period. This provides a heel rocker to initiate knee flexion for shock absorption during WA.

The 4 vastii (lateralis, intermedius, medialis longus, and medialis oblique) rapidly increase their action to peak intensity. Their function is to limit the knee flexion that was initiated by the heel rocker and ensure stable WA. The vastii relax in early mid stance once the initial knee flexion wave has been restrained. Thus, the hip and knee extensors ensure stability as body weight is transferred onto the limb during WA. Shock absorption is provided by the heel rocker stimulus and the vastii restraint of the resulting knee flexion. Progression is preserved by the heel rocker mechanics under the control of the pretibial muscles. Having accomplished their tasks, the hip extensor, knee extensor, and pretibial muscle groups relax.

Mid Stance, Terminal Stance, and Early Pre-Swing

While there is a brief period of vastii muscle action during early mid stance to assist knee extension, the primary responsibility for limb control is transferred to the ankle extensor muscles to provide graded progression over the supporting foot. For the rest of the weight-bearing period, the ankle plantar flexor muscles assume full responsibility for limb stability.

The soleus is the first to be activated (7% GC), followed shortly by the gastrocnemius (9% GC). Once the foot becomes stable as a result of a foot flat posture at the end of loading response, the tibia becomes the moving segment. Gastrocnemius and soleus action provides a plantar flexor force to restrain the rate of tibial advancement. Two functions are accomplished. Passive knee extension is induced by having the tibia move forward more slowly than the femur. This also assists hip extension. As a result, there is no need for action by either the hip or knee extensors during the latter portion of mid stance through terminal stance. Secondly, modulated soleus and gastrocnemius activity balances the need for tibial stability with that of forward progression. During terminal

stance, heel rise increases the demand on both the contractile and elastic components of the musculotendinous soleus and gastrocnemius complex. This is evidenced by the peak in plantar flexor muscle activity during this period and the subsequent elastic recoil during weight transfer.

The final source of sagittal limb control is the toe flexors. It starts with flexor digitorum longus at the initiation of mid stance (13% GC) as the foot flat posture allows the toes to contact the ground. Then, the flexor hallucis longus becomes active as body weight rolls toward the first MTP joint. Action of the toe flexors enlarges the forefoot support area by incorporating the base of the first phalanx with the metatarsal heads. Rapid transfer of body weight to the other foot with the onset of double limb stance in pre-swing terminates the action of the plantar flexors, including the toe flexors.

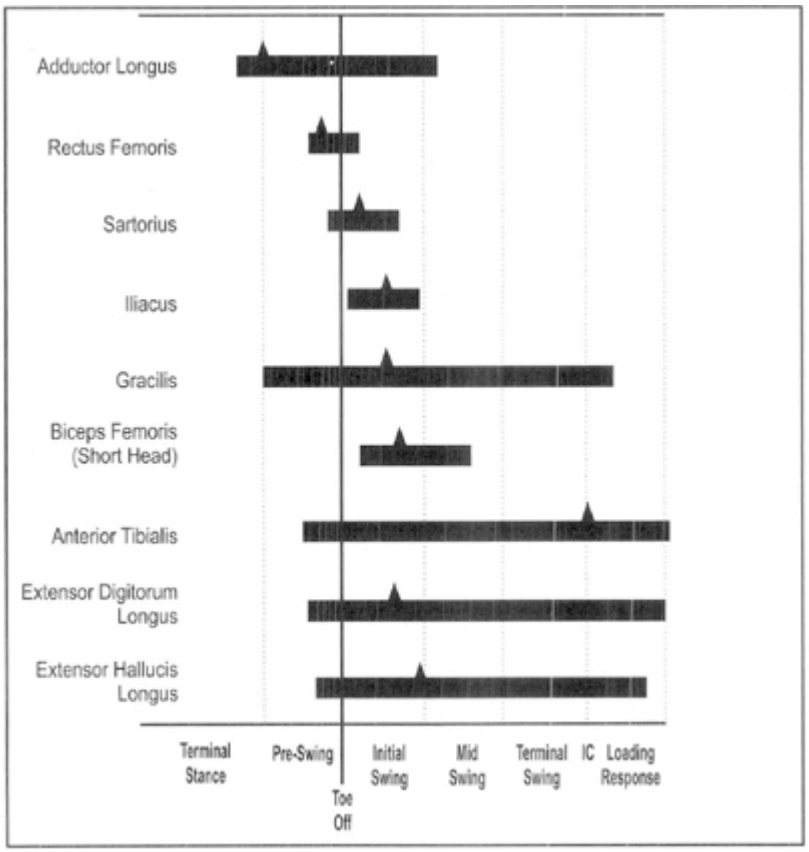

Figure 9-11. Muscle se-quence for swing.

SWING MUSCLE CONTROL PATTERN

Limb advancement relies on 2 patterns of muscle action. The transition from stance to swing is accomplished in pre-swing. This is followed in initial swing with a mass synergy of flexor muscle action that lifts and advances the limb (Figure 9-11 and Table 9-2). The effects of the initial swing effort continue through mid swing with minimal addition of muscular action.

PRE-SWING

The adductor longus muscle becomes active at the end of terminal stance to control the lateral transition of body weight from the trailing limb to the other leg. Its anteriormedial alignment also produces hip flexion, and this appears to be a key role for the muscle as its action continues until the beginning of mid swing. The result is reversal of hip motion from hyperextension toward flexion.

The gracilis and sartorius muscles initiate activity during pre-swing. While they share a common function as hip and knee flexors, the adduction and internal rotation roles of the gracilis counterbalance the abduction and external rotation forces provided by the sartorius.

RF activation often occurs in late pre-swing. Its role is deceleration of excessive knee flexion when the passive events are overly effective. The hip flexor capability of the RF also assists limb advancement.

Table 9-2

Muscle Sequence for Swing (% Gait Cycle)

Muscle	On	Off	Peak
Adductor longus	46	77	50
Gracilis	50	4	69
Rectus femoris	57	65	59
Sartorius	60	71	65
Iliacus	63	74	69
Biceps femoris short head	65	82	71
Anterior tibialis	56	13	0
Extensor digitorum longus	57	12	70
Extensor hallucis longus	58	9	74

The ability of these hip flexors to influence limb advancement is augmented by the passive events that also are occurring. With the onset of double stance, the limb is rapidly unloaded. As the heel rises farther from the ground and the tibia falls forward, the knee flexes and the thigh advances. The remaining mild plantar flexor force also assists upright balance as toe contact is maintained.

The pretibial muscles (anterior tibialis and long toe extensors) become active in the latter half of pre-swing and rapidly increase their intensity to nearly peak effort. This dorsiflexor action counteracts the residual PF.

All of the actions during this terminal double stance period prepare the limb for swing. Stance stability has been disrupted, a large arc of knee flexion has been provided, and the muscular control to reverse ankle motion has been activated. The evolving flexor synergy includes overlapping activity across the hip flexors, knee flexors, and ankle dorsiflexors.

INITIAL SWING

Ongoing activity of the adductor longus, gracilis, and sartorius is now augmented by the onset of iliacus EMG. Collectively, these muscles advance the thigh. Use of the hip flexor muscles is

inconsistent among individuals and is of low intensity. It appears that the momentum gained in pre-swing often is sufficient to continue thigh advancement through initial to mid swing. Accompanying activity of the BFSH augments the knee flexion component of the initial swing hip and knee flexor synergy to lift the foot for limb advancement.

Increasing activity of the pretibial muscles lifts the foot from its previously plantar flexed position. Peak activity of the EHL and EDL ensures the toes clear the floor.

MID SWING

Thigh advancement continues, despite the lack of substantial hip flexor activity during this phase. Iliacus, sartorius, and RF activity has ceased. Inconsistent continuation of the gracilis action sometimes occurs at this time. Residual momentum from initial swing appears to be sufficient to advance the thigh to 25° flexion.

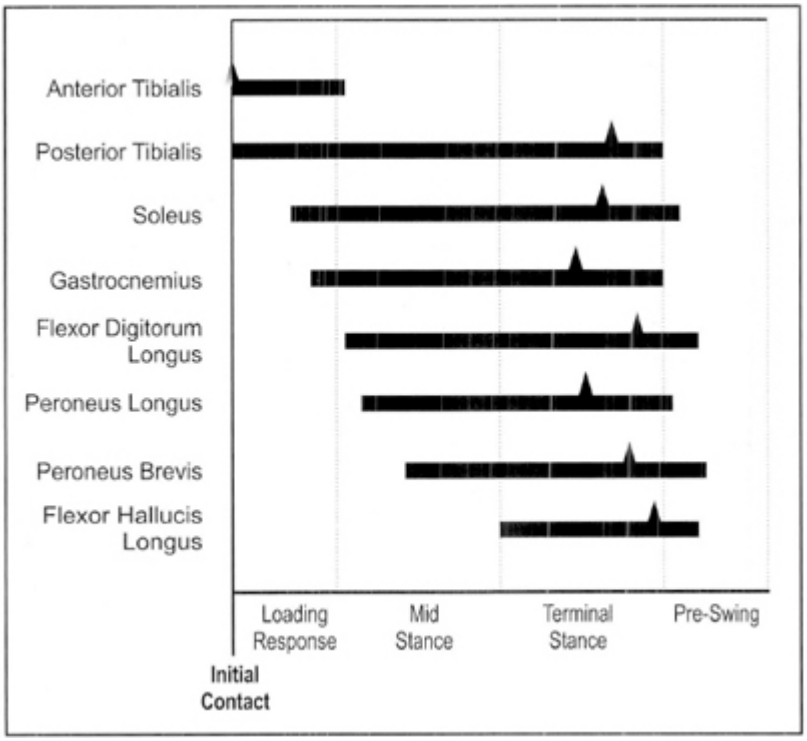

Figure 9-12. Muscle sequence controlling the foot joints during stance.

Control at the ankle also is variable. While peak ankle DF is reached in mid swing, the mean EMG pattern shows a significant reduction in the intensity of the dorsiflexor muscles, particularly the inverting anterior tibialis and EHL. Often the muscles cease activity during mid swing. Again, this implies that momentum generated by the vigorous muscle action in initial swing is sufficient to meet the mid swing demands. Toe clearance occurs with a minimal amount of muscular effort.

Thus, the pattern of swing phase muscle control differs from the actions during stance. The demands of swing stimulate nearly synergistic action of the limb flexors. In contrast, the stance requirements at the hip, knee, and ankle are sequential through overlapping participation by the muscles.

FOOT CONTROL

The sequence of muscular control for the joints within the foot reflects the demands made by body weight rolling across the foot (Figure 9-12 and Table 9-3). Inverting, plantar flexing, and everting muscle groups generate sufficient moments to provide dynamic stability across the ST, MT, and metatarsal joints.

LOADING RESPONSE

Peak TA muscle action following IC contributes an inversion force during the heel support period. This acts to restrain the tendency for the ST joint to collapse into eversion.

Activation of the tibialis posterior at IC adds a more dedicated invertor moment. Its rapid rise in intensity during the first half of loading response is specifically related to ST deceleration and to controlling the magnitude of foot pronation.

Table 9-3

Muscle Sequence Controlling the Foot Joints (% Gait Cycle)

Muscle	On	Off	Peak
Anterior tibialis	56	13	0
Extensor digitorum longus	57	12	70
Extensor hallucis longus	58	9	74
Posterior tibialis	0	50	44
Soleus	7	52	43
Gastrocnemius	9	50	40
Flexor digitorum longus	13	54	47
Peroneus longus	15	51	41
Peroneus brevis	20	55	46
Flexor hallucis longus	31	54	49

Soleus onset in late loading response adds an inversion force for ST control, incidental to its primary role as an ankle plantar flexor. The nearly simultaneous activation of the gastrocnemius, which generates a mild eversion moment, supports the interpretation that ankle control is the primary objective of these muscles.

Mid and Terminal Stance

Flexor digitorum longus activation at the beginning of mid stance is a response to loading of the forefoot. With the toes stabilized by floor contact, the flexor digitorum longus provides a plantar flexor force across the arch to support the MT joints. The intensity progressively increases in response to the higher demands of advancing body weight across the foot and heel rise. Peak action occurs in late terminal stance to reinforce the stability of the forefoot rocker. It recedes following contralateral heel contact.

Peroneus longus onset in early mid stance identifies the need for first ray stabilization to counteract the lift effect of the inverting (soleus and posterior tibialis) muscles on forefoot position. Peroneus brevis activation, which quickly follows that of peroneus longus, adds a more direct eversion force for lateral foot stability. This is needed

as increasing intensity of the inverting muscles introduces imbalance. Total forefoot contact is, thus, assured. The onset of flexor hallucis longus at the start of terminal stance accompanies heel rise and the need to stabilize the first MTP joint for weight bearing. Stable toe contact also enlarges the area of support for the forefoot rocker action. Transfer of body weight to the other limb in pre-swing terminates action of these muscles.

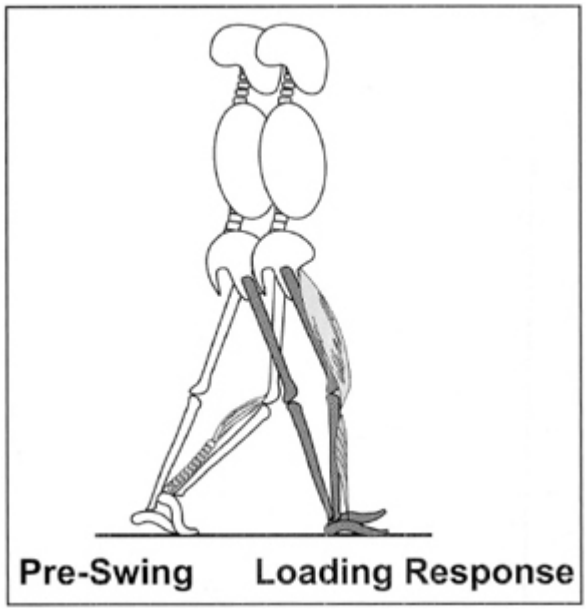

Figure 9-13. Weight transfer synergy includes reference limb IC, loading response, and contralateral limb pre-swing.

THE RECIPROCAL SYNERGIES OF NORMAL WALKING

Walking advances the body along the desired path of progression by the 2 lower limbs reciprocally repeating the same sequence of basic motions with a 50% offset of IC. Detailed analysis of the anatomical and biomechanical events, which characterize the 8 phases of gait, have clearly defined the way the limb accomplishes the basic tasks of shock absorption, weight-bearing stability, and energy conservation. The same analytical approach, however, has not provided a comparable description of body progression. This

inconsistency suggests that progression is a bilateral function of walking, and this has received insufficient attention. To correct this omission, the functions the 2 limbs accomplish during a typical step were reorganized into a series of reciprocal synergies.

Progression, during walking, is accomplished by the body advancing over the supporting foot. Thus, the major weight-bearing phases were selected as the basis for defining the patterns of bilateral synergistic limb function. These 4 phases of stance are IC/loading response (12% GC), mid stance (18% GC), and terminal stance (20% GC). The sum of these time periods totals 50% of the GC. Equivalent percentages of the rest of the GC, offset by 50%, designate the synergistic function of the contralateral limb.

Each phase was analyzed to identify all the events contributing to progression even though the phase had another primary purpose. Each synergy is named for the phase of the weight-bearing limb.

Synergy 1: Weight Transfer (0% to 12% GC and 50% to 62% GC)

Loading response (including IC) is the first phase of the reference limb. Its primary function is shock absorption. Pre-swing is the reciprocal phase and its primary function is preparation of the limb for swing. This synergy is an intense period of double stance, with the weight-bearing actions of both limbs contributing to progression (Figure 9-13).

The loading response of the reference limb employs the heel rocker to advance the tibia and the COP. The abrupt transfer of body weight onto the heel initiates an arc of rapid ankle PF. This is immediately resisted by eccentric contraction of the anterior tibialis. With the muscle's peak EMG occurring at IC, ankle PF is limited to 5° at 6% GC. The recent redefinition of eccentric muscle action by ultrasound analysis implies that the anterior tibialis restrains the ankle with an isometric hold while stretch of the tendon allows a small arc of foot drop. Elastic recoil of the tendon returns the ankle to neutral DF, while the heel still is the area of foot support. Preservation of the heel rocker combined with stretch tension in the

pretibial tendons advances the tibia from a negative 15° angle to vertical 0° by the time of forefoot contact. At the same time, the COP quickly moves across the heel and reaches the ankle axis by the end of the loading response phase.

Advancement of the thigh on the tibia by the eccentric pull of the quadriceps is slightly limited by the 20° knee flexion allowed for shock absorption. The accompanying hip flexion maintains an erect trunk with the vector and tibia both vertical.

The pre-swing synergy by the reciprocal limb provides “push-off.” The rigidly extended limb is converted into a dynamically flexed limb ready for swing. At end of terminal stance, the reference limb was locked in full hip and knee extension by its trailing position over a dorsiflexed ankle with the COP at the forefoot. The entire gastrocnemius-soleus tendon complex is tightly stretched by the forward alignment of the body COM. The abrupt partial unloading of the limb following floor contact by the weight-accepting (leading) foot unlocks the stretched tendon of the trailing limb and generates a major PF power burst by elastic recoil (there is no EMG).[3] This dynamic response plantar flexes the ankle and rolls the trailing foot and shank forward over the toe rocker. This passively flexes the knee and unlocks the hip. The knee gains 40° flexion and the thigh is advanced from its extended position.

Thus, the 2 gait phases of double limb support actively contribute to progression of the body mass. Loading response moves the trunk forward as the limb provides shock absorption. Pre-swing advances the trailing limb and makes a small (4%) contribution to the progression of the COM (identified by kinetic analysis).[4]

Synergy 2: Transition (12% to 31% GC and 62% to 81% GC)

Mid stance is the second phase of reference limb action. This phase also is the first period of single stance. Initial swing and early mid swing are the phases of the reciprocal limb. Mid stance utilizes

the ankle rocker for progression while the reciprocal limb is in swing (Figure 9-14).

At the onset of mid stance, the ankle is at neutral with the tibia upright while the foot is stationary flat on the floor. Both the hip and knee are in 20° flexion. As the body vector advances across the foot, the hip and knee joints regain neutral alignment (0° at the hip and 5° flexion at the knee). During this same period, the ankle moves into 5° DF and the base of the body vector (COP) advances to the forefoot. Ankle DF is restrained by the soleus and to a lesser extent by the gastrocnemius.

The primary intrinsic propelling forces available to minimize hip and knee flexion are late semimembranosus, semitendinosus, and vastus intermedius muscle activity. Each has prolonged EMG, but the intensity is waning. Small extensor power bursts at the hip (0.7 W/kg•m) and at the knee (0.5 W/kg•m) are generated as the mode of muscle contraction changes from eccentric to concentric. Another potential force is residual momentum from pre-swing.

The postural modifications at the hip and knee simplify extensor limb control by aligning the vector anterior to the knee and posterior to the hip. This passive control also gives the hip and knee joints the freedom to quickly respond to changes in limb alignment. Active control of the whole limb is transferred to the ankle plantar flexor muscles.

Initial swing by the reciprocal limb rapidly flexes all 3 joints (hip, knee, and ankle). The limb is advanced by the primary hip flexor (iliacus) or by momentum when the individual is walking at his or her optimum velocity. Low-intensity action of the sartorius and gracilis also assists flexion of the hip and knee. The total 60° of flexion at the knee enables the foot to clear the floor as hip flexion advances the limb.

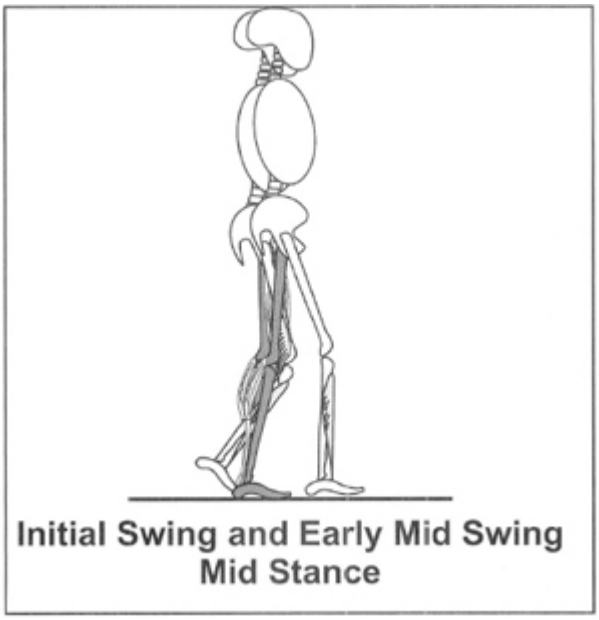

Figure 9-14. Transition synergy includes reference limb mid stance, contralateral limb initial swing, and early mid swing.

Three factors prepare for optimal progression. By the end of this second synergy (31% GC), the COP of the weight-bearing limb is over the metatarsal heads with the body vector slightly anterior to the ankle. The body's COM is at peak height.[2] The rapid hip flexion during initial swing and early mid swing has advanced the reciprocal limb ahead of the stance limb vector. The resulting anterior tilt of the body vector and the anterior alignment of the COM of the swing limb position the body's COM to generate passive (gravity induced) energy for progression over the stance limb.

Synergy 3: Progression (31% to 50% GC and 81% to 100% GC)

Terminal stance is the third phase of reference limb activity and the last half of single stance. The phases of the synergistic limb are late mid swing and terminal swing. This synergy is totally committed to progression (Figure 9-15).

The body's COM falls forward during terminal stance as the foot rotates over the forefoot rocker. Ankle DF increases a mere 3° to 5°.

The virtual ankle lock by the soleus and gastrocnemius muscles creates an anterior foot lever between the forefoot and ankle joint. As the advancing body generates a plantar flexor moment, eccentric plantar flexor muscle action preserves the height of the ankle and body's COM by stabilizing the alignment between the limb and anterior foot lever. The magnitude of progression is displayed by the heel rise.

Starting with the ankle in 5° DF and the vector at the forefoot, the heel progressively rises as the body falls forward. Elevation of the heel is 4 cm by the end of terminal stance.[11] The documentation of the onset of heel rise varies with the mode of measurement. A compression-sensitive heel switch identifies heel rise starting with the onset of terminal stance (31% GC). Visual identification by eye or physical displacement of a heel marker delays identifying the onset of heel rise until expansion of the heel's soft tissues ceases.

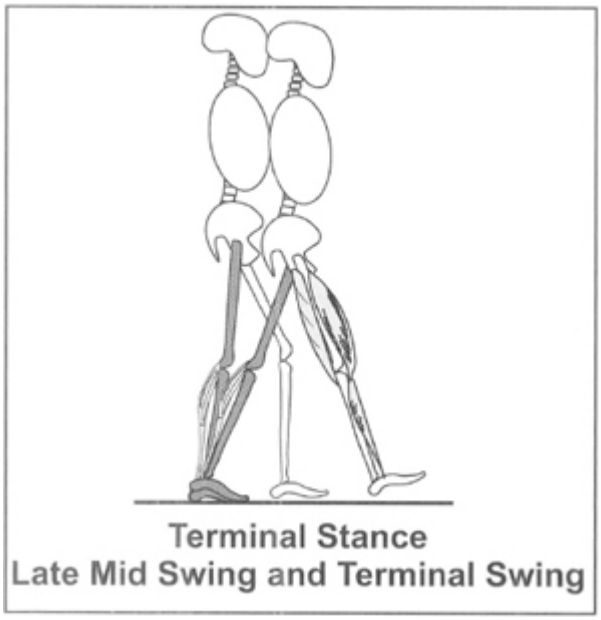

Figure 9-15. Progression synergy includes reference limb terminal stance and contralateral limb late mid swing and terminal swing.

The dominant event during most of terminal stance (31% to 47% GC) is increasing EMG activation of the soleus and gastrocnemius as these muscles oppose the increasing plantar flexor moment of the tilting vector created by the forward fall of the COM. Ultrasound analysis has shown that the muscle fascicles stabilize the ankle by

isometric contraction while tendon stretch allows the ankle to dorsiflex an additional 3°.[3]

Terminal stance ends with the loss of limb instability. By 47% of the GC, the GRF reaches its peak and then begins to wane. Weight-bearing alignment of the limb becomes unstable during the last portion of the phase. Support at the forefoot becomes less secure as the COP advances over the small metatarsal heads and onto the MTP joints. Advancement of the knee axis ahead of the body vector introduces knee flexion and further instability. Floor contact by the reciprocal limb ends terminal stance.

The synergistic function of the reciprocal limb is completion of SLA. Each joint has a unique need. The first action is finalization of hip flexion. By the middle of mid swing, the thigh reaches 25° flexion. This is slightly excessive for optimum floor contact. Prompt activation of the hamstrings (primarily the semimembranosus and long head of the biceps femoris) decelerates the flexing hip and returns it to 20°, which is sustained. Peak muscle intensities of 38% and 22% MMT are reached in mid-terminal swing. The hamstring muscles are the first to be activated because their simultaneous flexor action at the knee avoids excessive extension between the accelerating tibial momentum and femoral deceleration. The intensity of the hamstring muscles soon wanes to minimize opposition to needed knee extension. Hip stability is preserved by the onset of the primary hip extensors. The onset of quadriceps activity in terminal swing completes knee extension. This is accomplished with muscle intensity of 20% MMT. After the ankle attains neutral DF in early mid swing, the pretibial muscles tend to reduce their intensity as tilting of the tibia decreases the pull of gravity. Then in terminal swing, muscle intensity is increased in preparation for the greater demands of loading response.

The dynamic knee extension and ankle DF in combination with preservation of hip flexion increase the anterior alignment of the swing limb's COM relative to the vertical vector of the stance limb. This swing limb activity generates potential (gravity induced) energy, which progresses the stance limb over the forefoot rocker. The moment generated by the swing limb induces the stance limb's plantar flexor muscles to maintain its forefoot lever. The eccentric

action of the soleus and gastrocnemius provides ankle stability not tibial progression.

CONCLUSION

Each phase of gait contributes to progression, even though the force often is a by-product of the primary function in that phase. Synergy of the loading response heel rocker and the push off mechanics of pre-swing advances both the body and limb. Mid and terminal stance provide ankle and forefoot rockers, which optimize the momentum generated by pre-swing elastic recoil and the potential energy created by the swinging limb's advancement. Forward fall of the body mass is the final progressive force (passive).

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Section III

Pathological Gait

Chapter 10

Pathological Mechanisms

The diseases that impair patients' ability to walk may differ markedly in their primary pathology, yet the abnormalities they impose on the mechanics of walking fall into 5 functional categories: deformity, muscle weakness, sensory loss, pain, and impaired motor control. Each category has typical modes of functional impairment. Awareness of these characteristics allows the examiner to better differentiate primary impairment from substitutive actions.

DEFORMITY

A functional deformity exists when the tissues do not allow sufficient passive mobility for patients to attain the normal postures and ROM used in walking. Contracture is the most common cause. Other sources of deformity are abnormal joint contours and congenital disorders such as club foot.

A contracture represents structural change within the fibrous connective tissue component of muscles, ligaments, or the joint capsule following prolonged inactivity or scarring from injury.[6,7,20] The force of 2 fingers is sufficient to move any normal joint through its full range. The need for a stronger push to move the joint signifies a contracture in patients with otherwise normal neuromotor control (Figure 10-1).

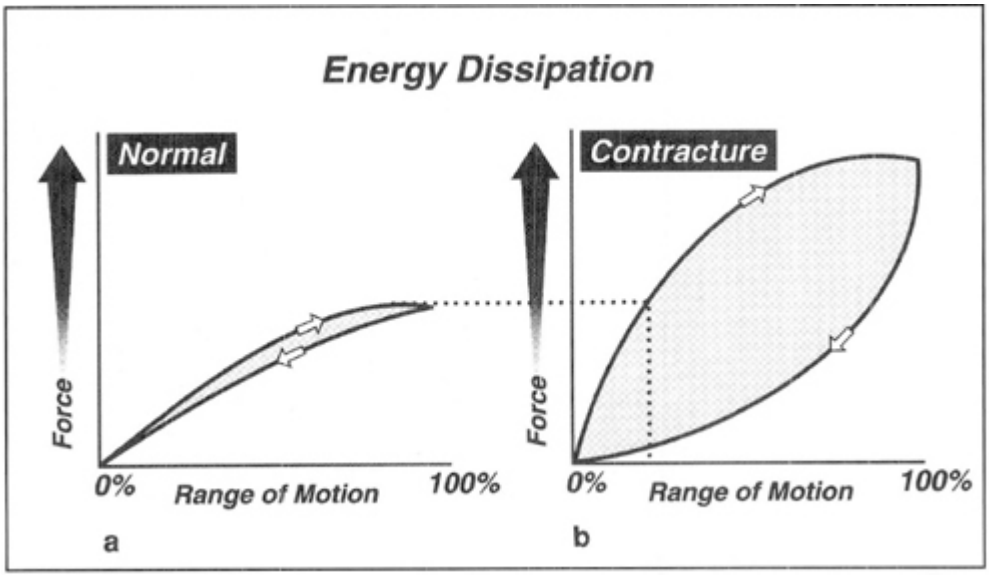

Figure 10-1. Tissue resistance to passive motion. Black lines = forces involved. Tissue stiffness indicated by width of space between flexion (up) and extension (down) force curves. (A) Normal tissue flexibility takes minimal force. (B) Contractures require greater force proportional to their tissue stiffness.

Relative density as well as maturity of the connective tissue leads to 2 clinical contracture patterns: elastic and rigid. Elastic contractures, which often arise from inactivity, yield to forceful stretch of either the examiner or body weight. A rigid contracture has the stiffness to resist considerable force, such as body weight.

While both forms of contractures display excessive resistance to a manual stretch, they differ in their response during the GC. An ankle PF contracture is a prime example. The elastic version presents a confusing situation. It limits motion during mid swing as the pretibial muscles are not programmed to pull harder, yet its less dense nature yields under body weight and allows normal or slightly delayed ankle motion during stance. In contrast, a rigid contracture, enhanced by the scarring from trauma or surgery, blocks tibial progression in stance and foot clearance in swing (Figure 10-2).

Knee flexion contractures provide another good example of the impact of restricted mobility on function. During stance, a knee flexion contracture inhibits advancement of the thigh over the tibia (Figure 10-3A) and greater muscular activity is required to stabilize the flexed weight-bearing knee (Figure 10-3B).[2] A knee flexion

contracture during swing shortens step length due to the reduction in terminal swing knee extension.[8]

MUSCLE WEAKNESS

The patient's problem is insufficient muscle strength to meet the demands of walking. Disuse muscle atrophy as well as neurological impairment may contribute to this limitation. When the cause is a lower motor neuron disease or muscular pathology, weakness is the only impairment. The most frequent motor neuron pathologies include poliomyelitis, Guillain-Barré syndrome, muscular dystrophy, and primary muscular atrophy. These patients have an excellent capacity to substitute, as normal sensation and selective neuromuscular control are intact. Patients with just muscle weakness can modify the timing of muscle action to avoid threatening postures and induce protective alignment during stance. Similarly, they find subtle ways to advance the limb in swing. Each major muscle group has a postural substitution. Patients also reduce the demand by walking at a slower speed. Because the patients do so well when they can substitute, clinicians tend to expect too much from a weakened muscle.

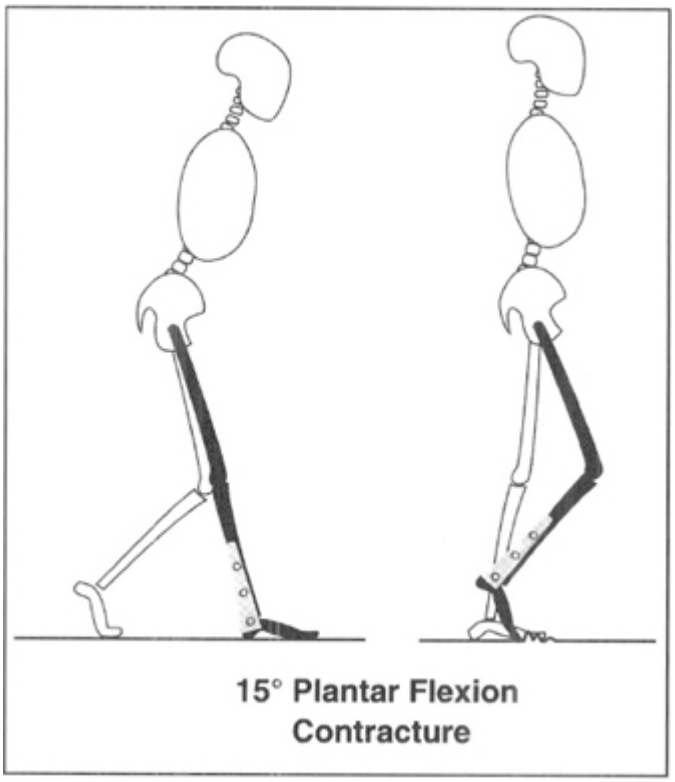

Figure 10-2. PF contracture. In stance, tibial advancement is blocked by contracture at the ankle (rigidity indicated by bolted plate). Swing requires increased hip flexion for floor clearance.

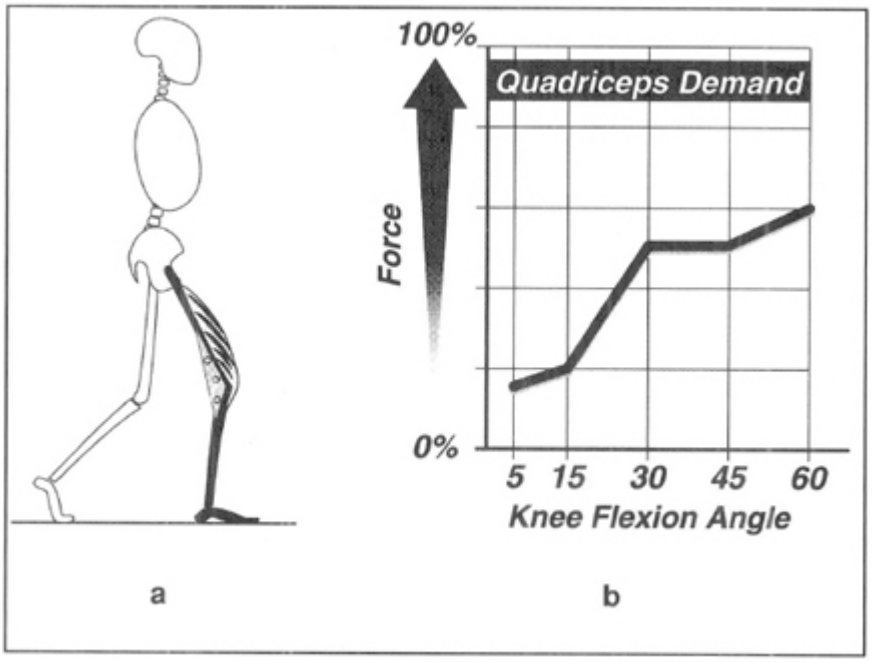

Figure 10-3. Knee flexion contracture (rigidity indicated by bolted plate). (A) Advancement of thigh is inhibited. (B) The demand on the

quadriceps increases with each greater degree of fixed knee flexion. (Adapted from Perry J, Antonelli D, Ford W. Analysis of knee joint forces during flexed knee stance. *J Bone Joint Surg.* 1975;57A:961-967.)

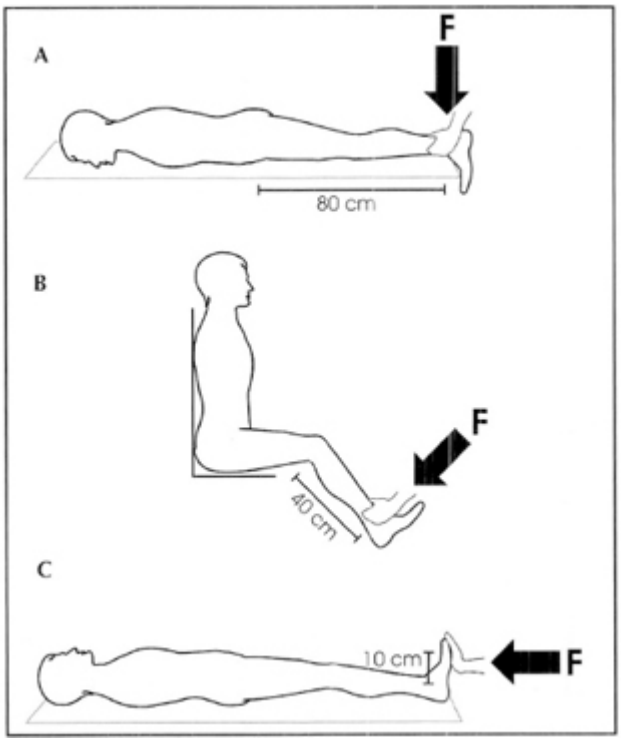

Figure 10-4. During manual muscle testing, the ability to accurately assess the strength of muscle groups is affected by the available lever arm at the (A) hip extensors = 80 cm, (B) knee extensors = 40 cm, and (C) ankle plantar flexors = 10 cm.

Predictions of walking ability commonly are exaggerated by the inability of manual muscle testing to identify the upper levels of normal strength. At the hip, a grade 5 (maximum examiner resistance) represents only 65% of nonparalytic normal.[1] Grade 5 at the knee is 53% of normal. Manual testing of ankle PF strength assesses only 18% of a person's ability to do a single, complete heel rise. The examiner's strength has not changed, but the available lever lengths are vastly different (Figure 10-4). Testing hip extensor strength with resistance applied near the ankle provides the examiner an 80-cm lever. Resistance applied at the ankle to test knee extension provides only a 40-cm lever. Both procedures remain

in practice. It is the responsibility of the examiner to bear in mind each procedure's limitations.

Testing ankle plantar flexor strength has been the most misleading. The available testing lever is limited to the distance between the joint axis and ball of the forefoot, just 10 cm (see Figure 10-4). The gross discrepancy between function and measured strength stimulated alternate approaches. Subjective assessment of the quality of a heel rise was one technique. This was followed by a count of the number of heel rises that could be performed. The first quantified determination of normal heel rise endurance set the threshold as 20 repetitions.[13] A subsequent study elevated the standard to 25 full range repetitions without knee flexion and having only finger tip support for balance.[12]

The earliest display of weakness, grade 4 (good), represents 40% of normal strength.[19] Assessment of plantar flexor strength reveals the ability to perform 10 unilateral heel raises. During normal walking, the muscles function at a 3 (fair) level.[18] This effort, averaging about 15% of normal strength, allows adequate reserve to avoid fatigue. Plantor flexor strength is limited to a single heel rise only. Patients with only 3 strength would have little endurance or reserve, as they must function at 100% of their available strength. Their compromise is to walk more slowly. Grade 3- (fair minus) is approximately 10% of normal.

Hence, strength testing must be judged critically and put in perspective of the testers' limitations. When patients meet the normal manual tests yet still are symptomatic, instrumented strength testing is needed to define the patient's true capability. Otherwise, subtle yet significant limitations will be missed.

Individuals with "borderline" strength deficits will often first show signs of difficulty during the phase that places the highest demand on the weakened muscle. For example, forward tibial collapse and/or failure to achieve a normal heel rise in terminal stance are 2 common signs of calf weakness, as this is the period of highest plantar flexor demand.

SENSORY LOSS

Impaired proprioception obstructs walking because it deprives patients of knowing the exact position of their hip, knee, ankle, or foot and the type of contact with the floor.[11] As a result, the patients do not know when it is safe to transfer body weight onto the limb. Persons with intact motor control may substitute by keeping the knee locked or hitting the floor with extra vigor to emphasize the moment of contact. The mixture of sensory impairment and muscle weakness prevents prompt substitution. Hence, walking is slow and cautious, even with moderate sensory impairment. When there is a greater deficit, the patients will be unable to use their available motor control because they cannot trust the motions that occur.[9] They may try to compensate for proprioceptive deficits by using their visual system to provide information about the position of the involved leg.[11]

Sensory impairment is not visible so it tends to be ignored. Also, grading proprioception is quite gross. There are only 3 grades: absent, impaired, and normal. A grade of normal should not be given unless the responses are both rapid and consistently correct. Both hesitation and the occasional error are signs of impairment. A slow response is equivalent to not having sufficient time to catch an overly flexed knee or inverted foot during walking. Consequently, the assessment of proprioception must be critical.

PAIN

Excessive tissue tension is the primary cause of musculoskeletal pain. Joint distension related to trauma or arthritis is a common situation. The physiological reactions to pain introduce 2 obstacles to effective walking: deformity and muscular weakness.

Deformity results from the tendency of a swollen joint to assume a resting posture, which minimizes tissue tension. Experimentally, the spontaneous resting postures have been shown to be the position of minimal intra-articular pressure. Movement in either direction increases the joint tension.[5] For the ankle, the minimal pressure

posture is 15° PF (Figure 10-5). The knee has an arc between 15° and 60° flexion (Figure 10-6), while the hip's range of least pressure is between 30° and 65° flexion (Figure 10-7).

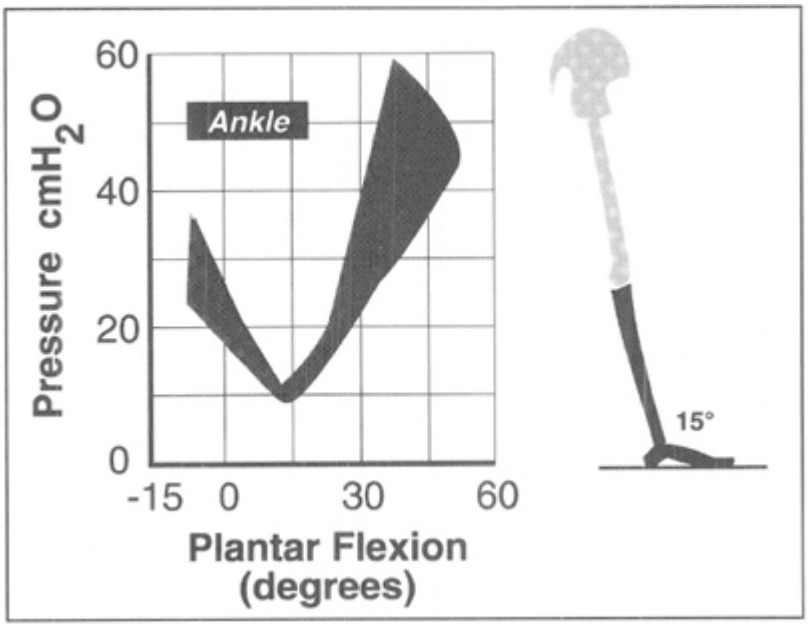

Figure 10-5. Ankle intra-articular pressure of a swollen joint through its range. Minimum pressure at 15° PF represents the joint's natural resting position (greatest volume). (Adapted from Eyring EJ, Murray WR. The effect of joint position on the pressure of intra-articular effusion. *J Bone Joint Surg.* 1964;46A(6):1235-1241.)

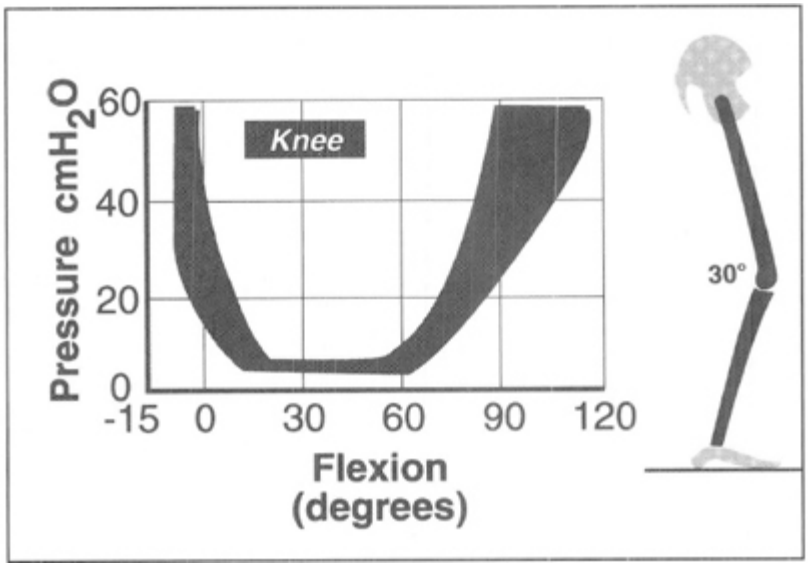

Figure 10-6. Knee intra-articular pressure of a swollen joint through its range. Minimum pressure from 15° to 60° flexion represents the joint's natural resting position (greatest volume). (Adapted from Eyring EJ, Murray WR. The effect of joint position on the pressure of intra-articular effusion. *J Bone Joint Surg*. 1964;46A(6):1235-1241.)

Muscle weakness follows the inhibition of activity by joint distension or pain. Experimental distension of the knee with sterile plasma increased intra-articular pressure while quadriceps activation became progressively more difficult.[4] When increasing pressure prevented all muscle action, full quadriceps function was restored by anesthetizing the joint (Figure 10-8). This reaction indicated that there is a feedback mechanism designed to protect the joint structures from destructive pressure. Patients display the cumulative effect of this protective reflex as disuse atrophy. During gait analysis, the examiner should expect less available strength and increased protective posturing when joints are swollen.

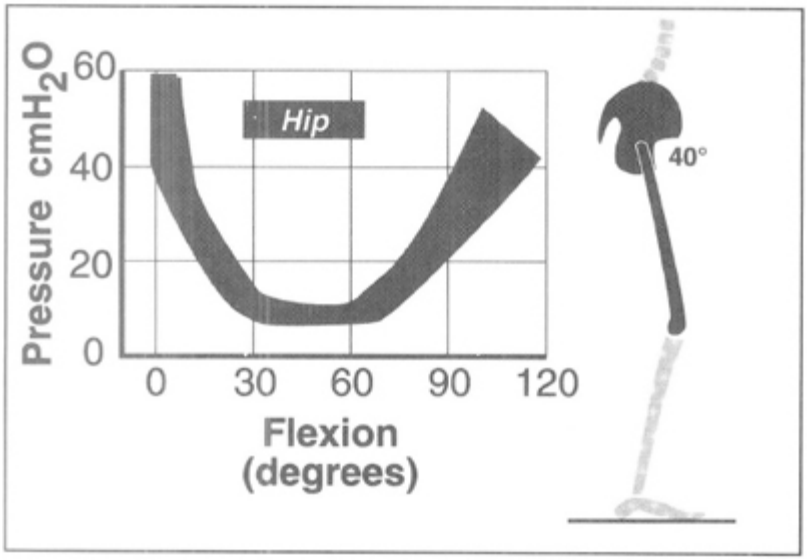

Figure 10-7. Hip intra-articular pressure of a swollen joint through its range. Minimum pressure from 30° to 65° flexion represents the joint's natural resting position (greatest volume). (Adapted from Eyring EJ, Murray WR. The effect of joint position on the pressure of intra-articular effusion. *J Bone Joint Surg*. 1964;46A(6):1235-1241.)

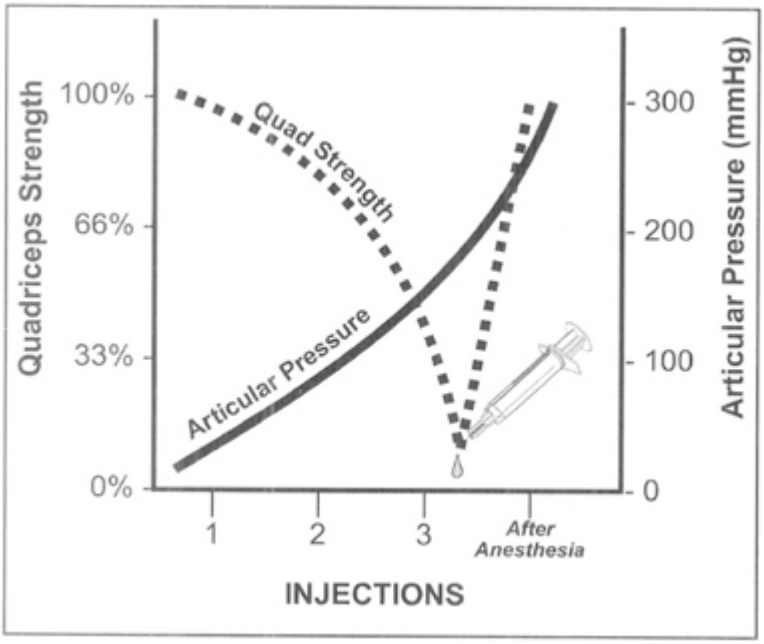

Figure 10-8. Quadriceps inhibition with knee joint distension. Quadriceps strength (top curve) decreases as articular pressure is increased (bottom curve). Injection (syringe) of an anesthetic into the swollen joint restores full quadriceps strength (vertical line). (Adapted from deAndrade MS, Grant C, Dixon A. Joint distension and reflex muscle inhibition in the knee. *J Bone Joint Surg.* 1965;47A:313-322.)

IMPAIRED MOTOR CONTROL

Patients with a central neurological lesion (brain or spinal cord) that results in spastic paralysis develop 4 types of functional deficits in varying mixtures and to differing extents.[16,17] The basic impairments include muscle weakness, impaired selective control, the emergence of primitive locomotor patterns, and spasticity. Motor control can be impaired by anything that injures the motor areas in the brain or the cervical and thoracic segments of the spinal cord. Clinical examples are stroke (cerebral vascular accident), traumatic brain injury, tetraplegia, paraplegia, multiple sclerosis, cerebral palsy, hydrocephalus, infections, and tumors.

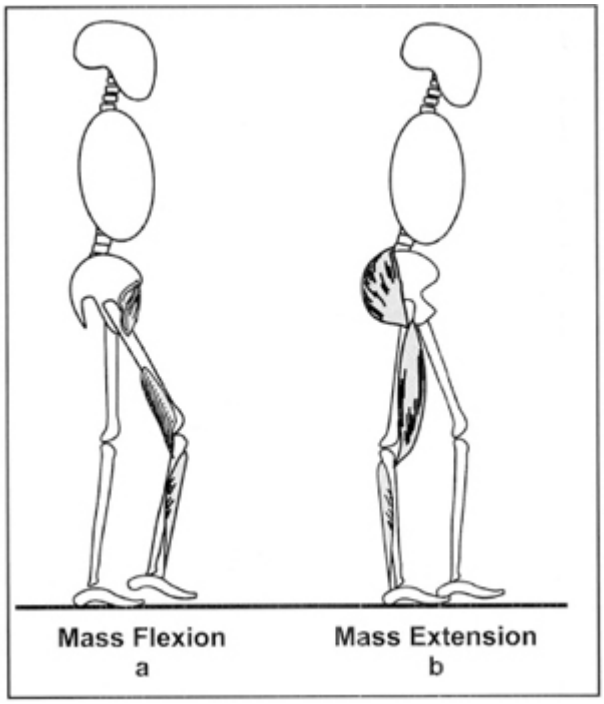

Figure 10-9. Primitive voluntary mass lower limb control. (A) Mass flexion: Activation of hip flexors, knee flexors, and ankle dorsiflexors. (B) Mass extension: Activation of hip extensor (gluteus maximus), knee extensor (quadriceps), and ankle plantar flexors (soleus).

Muscle weakness is the primary finding today.[14] The therapeutic challenge is to identify a way of stimulating muscle action early in the rehabilitation program in order to allow accurate prediction of the depth of neurological loss.

While impairments of selective control may be interpreted as weakness (similar to flaccid paralysis), the reflexes remain intact. This control deficit prevents patients from dictating the timing and intensity of muscle action. The patient may have difficulty activating the unique muscle combinations required for each phase of gait. While the whole limb is generally involved, the control loss is more severe distally. During loading response, for example, vigorous activity of the pretibials (the flexors of the ankle) and knee extensors may not be possible. The ankle most likely will end up in PF.

Primitive locomotor patterns commonly become an alternate source of voluntary control. They allow the patient to willfully take a step by using a mass flexion pattern (ie, the hip and knee flex simultaneously while the ankle dorsiflexes with inversion) (Figure 10-

9A). Stance stability is attained through the mass extensor pattern. Now the hip and knee extensors and the ankle plantar flexors act together (Figure 10-9B). Inability to mix flexion and extension eliminates the motion patterns that allow a smooth transition from swing to stance (and vice versa). Also, the primitive patterns do not let the patient modulate the intensity of muscle action that occurs during the different phases of gait. Weakness may further limit the utility of patterns. For example, when strength of key muscle groups in the flexion synergy (hip flexors, knee flexors, and ankle dorsiflexors) is insufficient, the patient loses the ability to lift and advance the limb during swing. Hence, a toe drag may ensue.

Spasticity obstructs the yielding quality of eccentric muscle action during stance and swing. The presence of spasticity is readily apparent when a quick stretch induces clonus (Figure 10-10A). Hypersensitivity of the muscles to slow stretch, however, may be missed or erroneously considered a contracture because the muscle action is continuous (Figure 10-10B). Soleus and gastrocnemius spasticity lead to persistent ankle PF. Progression is obstructed by loss of the ankle rocker and the inability to rise on the metatarsal heads for the forefoot rocker. Hip flexor spasticity restricts the ability to achieve a trailing limb posture in terminal stance, while sustained quadriceps action during pre-swing inhibits limb advancement and leads to a “stiff-legged” pattern.[3,10] The persistent knee flexion that follows hamstring spasticity limits the effectiveness of terminal swing and restricts thigh advancement in stance.

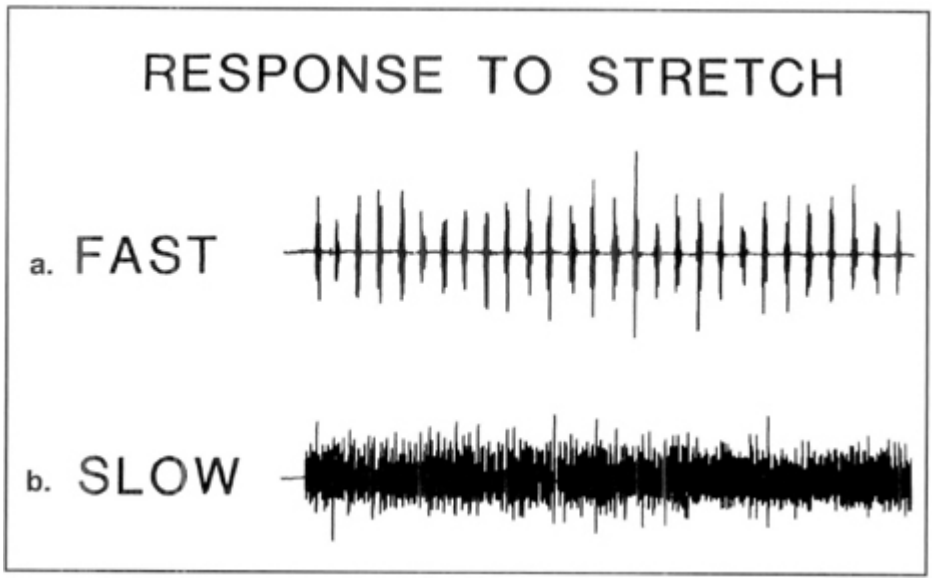

Figure 10-10. Spastic muscle response to stretch (EMG). (A) Fast stretch elicits clonus. (B) Slow stretch generates sustained muscle action, which can mimic a contracture.

Inappropriate phasing results from the sum of the control errors and spasticity. The activity of any muscle may be prolonged or curtailed, continuous or absent. Its onset and cessation may be premature or delayed. Each of these phasing errors may alter the pattern of sequential movements during gait.

The patient's ability to substitute is proportional to the amount of selective control and the acuity of their proprioception. Generally, only mildly involved persons are capable of accommodating to their lesions. Hemiplegia, by having one side intact, offers the best opportunity. Paraplegia from incomplete spinal cord injury is the next most versatile lesion. Spastic quadriplegia is the most disabling.

CONCLUSION

The modern emphasis on early mobilization has markedly changed the patient's functional potential. Contractures are much less severe, and spasticity is less intense at the time when the patient enters into rehabilitation. As a result, muscle weakness is the dominant picture[14] instead of the historical stiff, semi-flexed arm and

leg with only minimal ability to walk. Each patient has a unique mixture of impairments that alters his or her walking ability. Dynamic EMG is the only means of accurately defining the pattern of muscle dysfunction that is contributing to the individual patient's gait errors.[15]

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Chapter 11

Ankle and Foot Gait Deviations

Gait deviations of the ankle and foot have been divided into 4 categories. Floor contact deviations describe the mode of IC between the foot and the floor. The second category, ankle deviations, identifies abnormal joint postures occurring at the talocrural articulation. ST joint deviations describe the third subset of deviations that occur at the junction between the talus and calcaneus. The final grouping, toe deviations, identifies abnormal postures at the MTP joints.

The challenge is to differentiate the extent of the motion variability that is normal from that which is pathological. At the ankle, one standard deviation from the mean averages 5°. As the arcs of normal ankle motion are small, yet functionally critical, a 5° error is clinically significant in some situations. This is particularly true for the heel and ankle rockers.

FLOOR CONTACT DEVIATIONS

Customarily WA begins with a heel-first IC, followed by a controlled lowering of the forefoot to the ground. When IC occurs at a more distal area of the foot or the rate of forefoot lowering increases, the effectiveness of the heel rocker is reduced and the normal pattern of limb loading is disrupted.

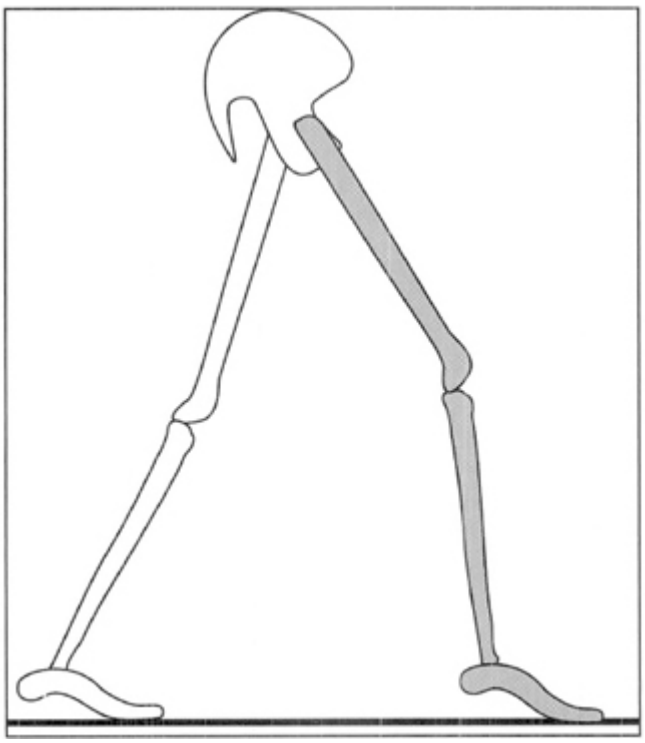

Figure 11-1. Forefoot contact.

FOREFOOT CONTACT

Definition: The forefoot is the initial point of contact with the ground during WA

Phase: IC

Functional Significance: Disrupts heel rocker, forward progression of the tibia, and shock absorption at the knee

Underlying Causes:

* Inadequate pretibial strength or PF contracture contributing to excessive ankle PF approaching 30°
* Combination of excessive ankle PF and knee flexion greater than 30°
* Compensation for heel pain
* Compensation for a short leg

Forefoot contact generally represents a mixture of ankle equinus and knee flexion. Either joint may have the greater deformity or they can be similar. A 15° posture at each joint is sufficient to place the

forefoot lower than the heel at the time the floor is contacted (Figure 11-1). The need for a knee flexion impairment may be limited, however, when the cause of forefoot contact is a short leg.

Forefoot contact can result in 3 loading patterns, depending on the cause (Figure 11-2). The most common pathology is combined spasticity of the hamstrings and ankle plantar flexors. The rapid stretch placed on the spastic hamstring muscles during terminal swing limits full knee extension. The forefoot then serves as the location of IC. In response to the quick stretch placed on the plantar flexor muscles at IC, the ankle may further plantar flex.

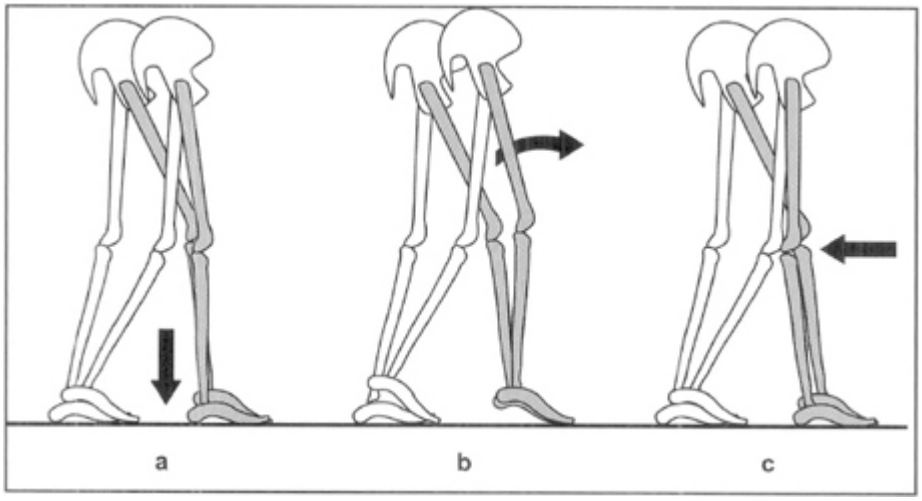

Figure 11-2. Loading response gait deviations from excessive ankle PF. (A) Forefoot contact with rapid drop to foot flat by flexible ankle. (B) Forefoot contact sustained and tibia advances. (C) Forefoot contact followed by heel dropping to ground and knee hyperextending due to rigid ankle PF.

Persons with extreme ankle equinus, such as that associated with flaccid paralysis of the pretibials or a PF contracture, often voluntarily flex their knee to bring the area of floor contact closer to the body vector line. This eases weight transfer. While the excessively flexed knee may initially require additional quadriceps activity to maintain stability, the lack of a heel rocker diminishes the subsequent loading-response demand on the muscle group.

Forefoot contact also may be used when heel pain exists. The high impact forces of limb loading are transferred to the forefoot

instead of the plantar aspect of the calcaneus.

Delayed Heel Contact

Definition: Forefoot precedes heel in contacting the ground

Phases: IC, loading response, mid stance

Functional Significance: Disrupts heel rocker and forward progression

Underlying Causes: Yielding PF contracture or spasticity

While the heel is not the initial site of floor contact, it often drops to the floor later in loading response or even mid stance (see Figure 11-2A). This is characteristic of an elastic contracture yielding as a greater portion of body weight is loaded onto the limb later in stance.

The PF contracture or spasticity that caused an initial heel-off situation yields to body weight in mid stance. The loss of forward momentum may be slight, even imperceptible in persons with otherwise good walking ability.

Having the heel reach the floor after an initial forefoot contact is a common finding in the slower walker with moderate excessive ankle PF. The plantar flexed ankle creates a rigid reverse rocker as the limb is loaded, which drives the heel to the floor and the tibia backwards. The nonvigorous walker lacks the momentum to overcome this decelerating situation and progression is inhibited.

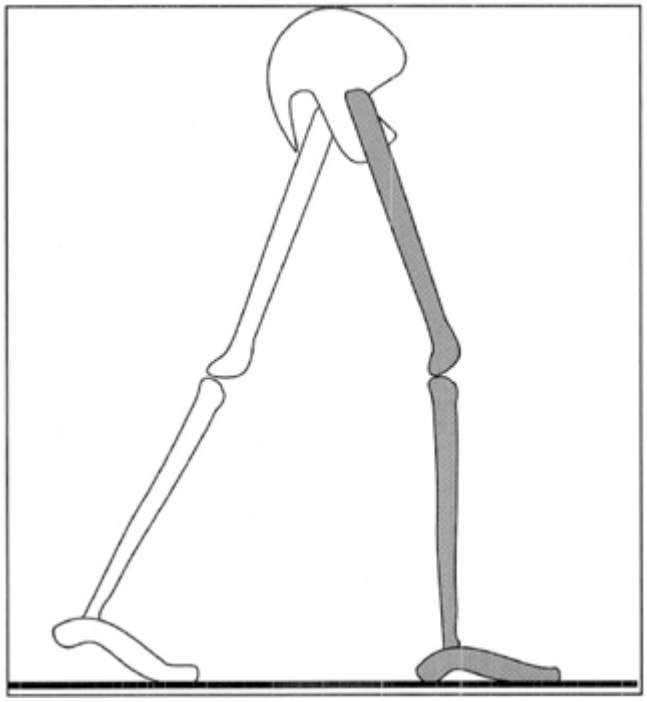

Figure 11-3. Flexed knee contributing to foot flat IC.

Foot Flat Contact

Definition: The heel and forefoot simultaneously contact the floor

Phase: IC

Functional Significance: Limited heel rocker and forward progression

Underlying Causes:

* Any impairment contributing to excess knee flexion
* Compensation for weak quadriceps

Foot flat contact results from incomplete knee extension in terminal swing combined with normal ankle DF (Figure 11-3). In patients with spastic paralysis, this is indicative of the primitive flexor pattern dominating swing limb control. Other causes are a knee flexion contracture and either tight or spastic hamstring muscles.

As the forefoot and heel strike the ground at the same time, the heel-only period is missing. Consequently, there is no heel rocker to induce additional knee flexion. Foot flat contact provides the person

with a stable base of support. While the excessively flexed knee may initially require additional quadriceps activity to maintain stability, the lack of a heel rocker diminishes the subsequent loading response demand on the muscle group. Persons with quadriceps weakness may intentionally contact the ground with a flat foot to reduce the peak demand associated with rapid knee flexion during WA.

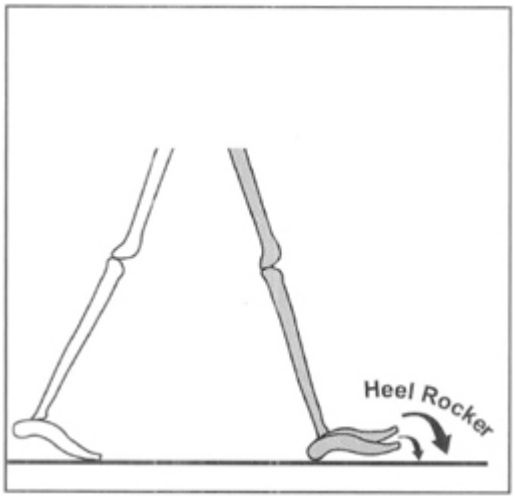

Figure 11-4. Low heel contact arising from excessive PF but normal knee posture.

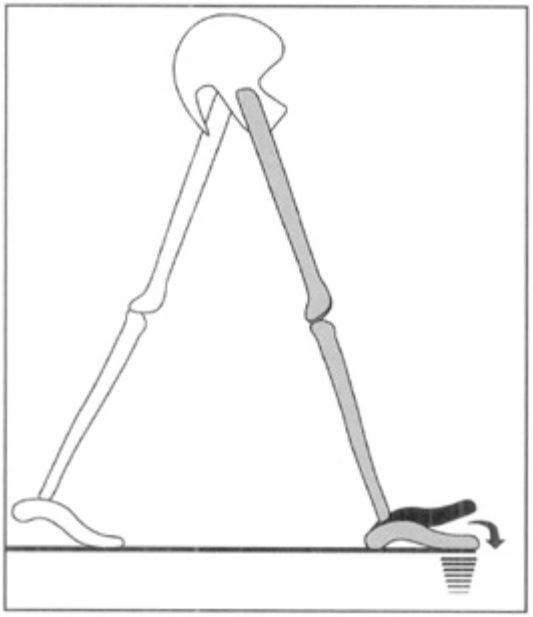

Figure 11-5. Initial heel contact followed by rapid drop of forefoot with audible slap.

Low Heel

Definition: While the heel makes the IC, the forefoot is very close to the floor

Phase: IC

Functional Significance: Reduces the heel rocker and forward progression

Underlying Causes: Any impairment contributing to excess PF

Low heel contact occurs when the foot strikes the floor with the ankle in 10° PF and the knee in its normal position (5° flexion). While the heel still initiates floor contact, the foot is nearly parallel with the floor (Figure 11-4). Hence, the period of heel-only support is abnormally short, leading to an equally brief heel rocker contribution to progression. Visual identification of this gait error requires close observation as IC is by the heel and the final foot and ankle postures are normal during loading response. A PF contracture, TA weakness, and premature action by the calf muscles are possible etiologies (see Figure 11-4).

Foot Slap

Definition: Uncontrollable PF at the ankle following initial heel contact, often accompanied by an audible “slap”

Phases: IC, loading response

Functional Significance: Disrupts heel rocker, forward progression, and shock absorption

Underlying Causes: Pretibial weakness, particularly the anterior tibialis

Following IC by the heel, the forefoot prematurely drops to the floor. The often audible slapping sound associated with the forefoot striking the floor has given rise to the term *foot slap* to describe this deviation (Figure 11-5). As a result, the period of heel-only support is

shortened. The heel rocker is disrupted as the weak pretibial muscles (particularly the TA) fail to draw the tibia forward.

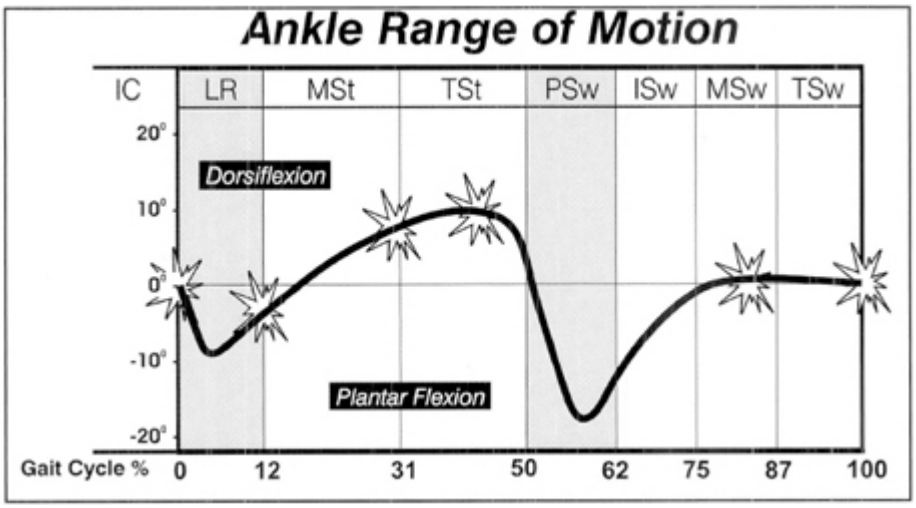

Figure 11-6. Excessive PF impacts all phases except pre-swing and initial swing.

ANKLE DEVIATIONS

Motion at the talocrural joint is best observed in the sagittal view (standing to the side of the patient). The 2 most common deviations, excess PF and excess DF, are characterized by positions that exceed the normal joint posture for a given phase. Excess PF and DF frequently occur in conjunction with an abnormal pattern of contact between the foot and the floor, which can also be described as a deviation (eg, premature heel off or delayed heel off). While many ankle deviations arise from impairments in muscles associated with the talocrural joint (eg, plantar flexor weakness), occasionally deviations arise due to abnormal postures occurring proximally (eg, a “drag” that can arise from the knee not flexing adequately during initial swing). Contralateral vaulting, a deviation of the opposite ankle, also is described under this section as it is reflective of reference limb pathology.

EXCESS PLANTAR FLEXION

Definition: PF that exceeds normal for a particular phase

Phases: All except pre-swing

Functional Significance: Disrupts rockers (heel, ankle, forefoot, and toe) during stance. Impairs foot clearance and limb advancement during swing.

Underlying Causes:

* PF contracture or hypertonicity
* Pretibial weakness
* Intentional move to decrease demand on the quadriceps
* Proprioceptive deficits
* Loose-packed ankle posture secondary to pain or joint effusion
* Initiation of extensor pattern in terminal swing

PF of the ankle beyond neutral alignment can introduce a functional error in all of the gait phases, except pre-swing and initial swing (Figure 11-6). Pre-swing is usually not affected as the ankle normally plantar flexes to 15° during this phase. Subtle contractures of less than 5° may show little observable impact on loading response and initial swing as the ankle moves into 5° of PF during these phases.

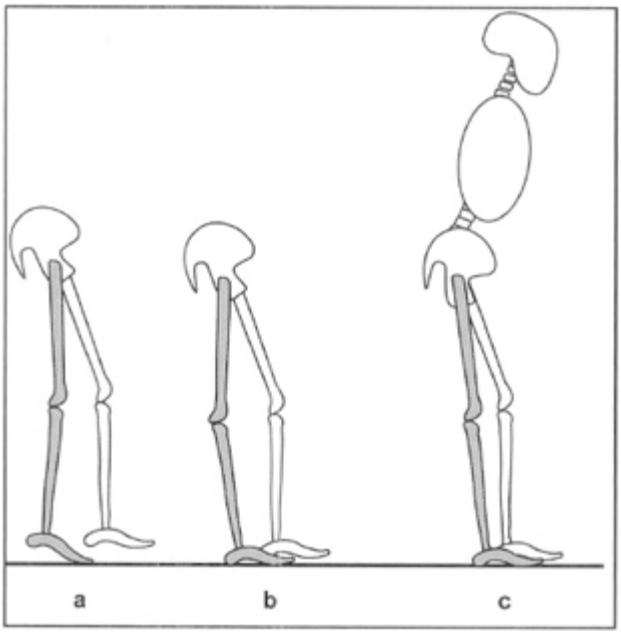

Figure 11-7. Mid stance gait deviations from excessive ankle PF. (A) Premature heel rise. (B) Foot flat with posteriorly restrained tibia. (C) Forward trunk lean substituted for lack of tibial progression

During stance, the primary functional penalty of excessive ankle PF is loss of progression. This leads to a shortened stride length and reduced gait velocity. Stability also is threatened by the difficulty of attaining an upright posture. In swing, excessive PF obstructs limb advancement.

Impact of Excess Plantar Flexion by Phase

Initial Contact/Loading Response

The effectiveness of the heel rocker as a source of shock absorption and progression is compromised by excessive PF. It is also modified by the mode of IC. When normal heel contact is followed by an “instantaneous” foot drop due to impaired pretibial control, the heel rocker is lost (see Figure 11-5). Low heel contact preserves only a small portion of the heel rocker, and consequently, the normal arc of knee flexion is markedly reduced (see Figure 11-4). Forefoot contact arising from a rigid PF contracture will modify the heel rocker in one of two ways depending on quadriceps force. If strength is adequate to support a flexed knee, the individual will maintain good progression by use of a forefoot rocker (see Figure 11-2B). The patient will also gain some shock absorption through the action of the quadriceps. Persons with a rigid PF contracture who lack strength to maintain a flexed knee will have progression interrupted due to the tibia being driven backward as the heel drops to the floor (see Figure 11-2C). When a PF contracture is elastic, the weight of the body will cause the heel to drop to the ground. The heel rocker will be lost (see Figure 11-2A).

Mid Stance

Excessive PF in mid stance inhibits tibial advancement. If a foot flat contact continues, the tibia advances only to the extent of the available passive range. Any limitation that restricts DF to less than 5° by the 30% point in the GC represents an abnormal restraint. With loss of the ankle rocker, progression is proportionally limited, leading to a short step length by the other limb.

Patients have 3 characteristic substitutions for their loss of progression. These include premature heel off, knee hyperextension, and forward trunk lean (Figure 11-7). All represent efforts to move the trunk forward over the rigid equinus. Which strategy is used varies with the patient's gait velocity and knee mobility. A mixture of the 3 adaptations also is common.

Premature heel rise (see Figure 11-7A) is the mechanism used by vigorous walkers with no other major disability. These patients have the ability to propel themselves from a low-heel contact, across an obstructive foot flat posture, and onto the forefoot. Now the heel rise occurs in mid rather than terminal stance. Exact timing varies with the magnitude of the PF contracture and the momentum available. The duration of foot flat is correspondingly limited. The extra time used in this effort results in a moderate reduction in walking speed. A velocity approximating 70% of normal (% N) is common.

Knee hyperextension can overcome the posteriorly aligned tibia when there is sufficient ligamentous laxity (see Figure 11-7B). The knee hyperextends as the femur follows body momentum and rolls forward over the immobile tibia. Walking vigor is not a factor in the use of these substitutions. It is common with cerebrovascular accident (CVA), incomplete spinal cord injury, and cerebral palsy. The range of knee hyperextension can increase in the growing child and in the more vigorous spastic patient. Their dynamics produce sufficient repetitive strain for the tissues to yield.

Cast immobilization following a fracture of the tibia or ankle not infrequently causes a rigid 15° PF contracture. Delayed heel rise increases stress on the posterior knee ligaments. If they yield under strain, knee hyperextension may develop.

Forward lean of the trunk with anterior tilt of the pelvis is the final substitution available (see Figure 11-7C). It is frequently used by persons who are more disabled and who have a slow gait velocity

(15% N). The substitution pattern serves more to maintain balance over the plantigrade foot with fixed ankle PF than to enhance progression. Stance stability is attained, but a significant demand is imposed on the hip and back extensors. When the patient has persistent forefoot support following limb loading, the ankle rocker is absent. Body weight advances as the patient rolls across the forefoot and the patient proceeds immediately into terminal stance.

Terminal Stance

The effects of excessive PF on terminal stance gait mechanics depend on the patient's ability to roll onto the forefoot. If the patient cannot attain a heel rise, the advancement of the body is limited to the extent that knee hyperextension or trunk lean and pelvic rotation improve forward reach of the opposite limb. In contrast, the vigorous walker who advanced from low-heel strike to premature heel rise will have a seemingly normal motion pattern in terminal stance. Excessive heel rise is an inconspicuous event (Figure 11-8). It also raises the pelvis and consequently increases the magnitude of force under the contralateral leg at IC.

Step length is shortened. The loss may be mild in the vigorous walker but can be severe in patients who never attain good stability on the forefoot.

Pre-Swing

If forefoot support was attained during terminal stance, there will be no significant gait abnormalities in pre-swing. The alignment to initiate knee flexion is present.

Patients who maintain heel contact throughout terminal stance may develop a late heel rise after body weight has been transferred to the other limb. Otherwise, the heel will not rise until the thigh begins to advance for initial swing.

Initial Swing

The diagnosis of excessive ankle PF in initial swing is hidden by the natural toe-down posture of trailing limb alignment. Excessive ankle PF in initial swing has no clinical significance unless it is

extreme. The trailing posture of the tibia tends to minimize the effect increased ankle PF has on toe drag.

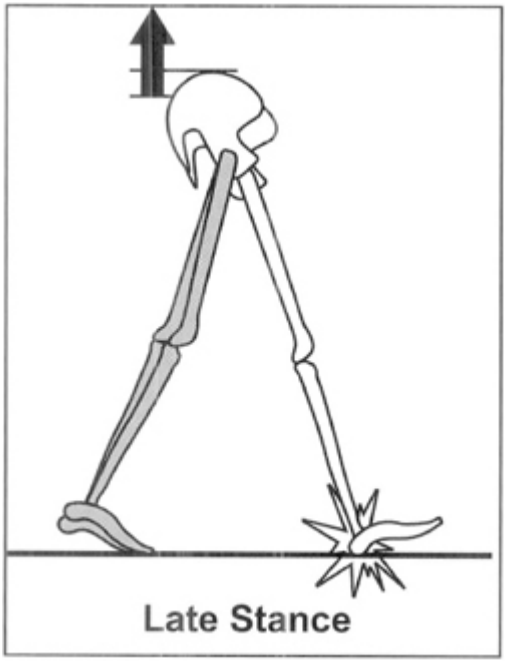

Figure 11-8. When the patient has excess PF and is able to roll onto the forefoot, the terminal stance gait deviations are increased heel rise, elevated pelvis, and sharper loading on the contralateral limb.

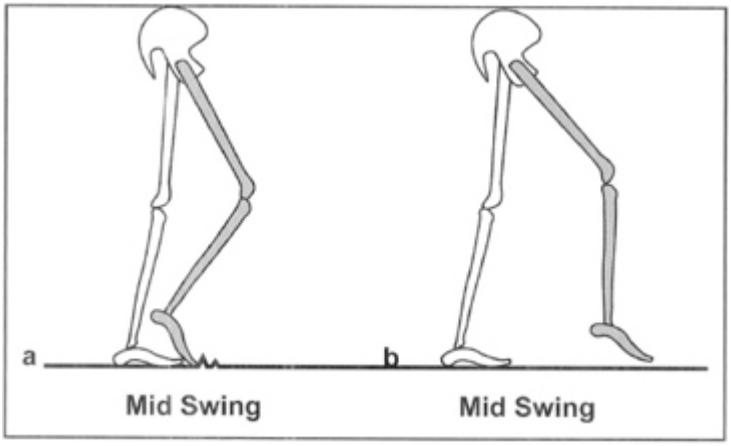

Figure 11-9. Mid swing gait deviations from excessive ankle PF. (A) Toe drag is the immediate reaction. (B) Increased hip and knee flexion substituted.

Mid Swing

Excessive PF in mid swing drops the forefoot below horizontal. The immediate effect is toe drag on the floor and inhibition of limb advancement (Figure 11-9A). As a result, swing is prematurely terminated unless there is adequate substitution to preserve floor clearance.

The most direct substitution for lack of adequate ankle DF in swing is increased hip flexion to lift the limb and, hence, the foot (Figure 11-9B). As the thigh is lifted, the knee flexes in response to gravity. Because displacement at the knee is more conspicuous, it often is erroneously considered to be the primary substitution for a foot drag. Knee flexion without hip flexion, however, would direct the tibia backwards and actually increase the foot's equinus posture rather than lift the toe.

Other substitutions are used to attain floor clearance when compensatory hip flexion is insufficient. These include circumduction, lateral trunk lean, and contralateral vaulting. Each of these compensatory maneuvers increases the energy cost associated with walking as a large mass now needs to be lifted versus only the weight of the foot.

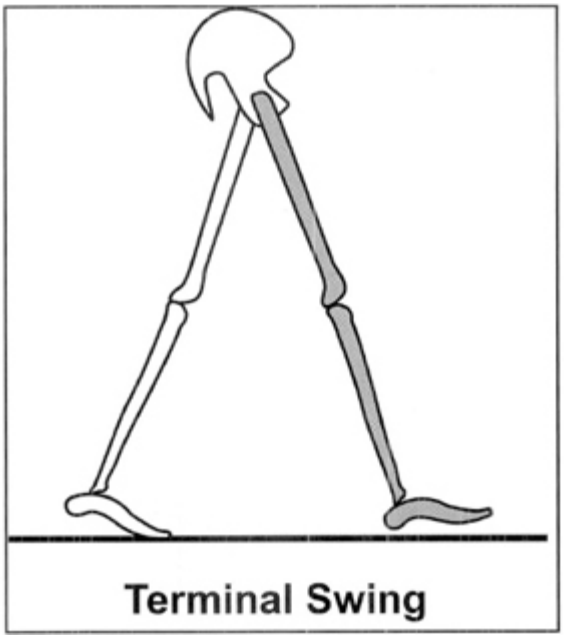

Figure 11-10. Fifteen degree excess PF does not cause toe drag in terminal swing.

Terminal Swing

Excessive ankle PF in terminal swing seldom interferes with floor clearance because the flexed hip and extended knee place the forefoot above the floor (Figure 11-10). Commonly a toe drag that is present in mid swing is corrected by the terminal swing lift of the foot. Hence, persistent toe drag indicates a mixture of excessive ankle PF and inadequate knee extension. The primary impact of excess PF in terminal swing is that the foot is positioned poorly for a heel-first IC.

Detailed Causes of Excessive Plantar Flexion

Pretibial muscle weakness, PF contracture, soleus overactivity (spasticity or pattern control), and voluntary posturing for a weak quadriceps are the 4 basic types of dysfunction that cause excessive PF. Their effects within the GC lead to different patterns of abnormal function (Table 11-1).

Pretibial Muscle Weakness

Failure of the pretibial muscles (primarily the TA) to produce adequate DF force allows the foot to fall in an uncontrolled manner. If only the TA is lost, the foot drop involves just the medial side of the foot. Continued activity by the EHL, EDL, and peroneus tertius produces a mixture of DF and eversion.

The magnitude of passive ankle PF also varies with the age of onset. Adult acquired disability seldom causes more than a 15° equinus posture. This also is generally true for the foot drop resulting from spastic paralysis. In contrast, when flaccid paralysis of the pretibial musculature occurs in early childhood, passive equinus may reach 30°, perhaps more. These differences in magnitude also influence the types of gait errors recorded.

Excessive PF resulting from inadequate TA activity is most apparent and clinically significant in mid swing (see Figure 11-9), IC (see Figure 11-1), and loading response (see Figure 11-2A). Floor clearance is the problem during swing. The heel rocker is altered in

stance. The subsequent stance phases will be normal following all 3 situations if inadequate pretibial muscle action is the only deficit.

Table 11-1

Causes of Excessive Plantar Flexion and Phasing

	IC	*LR*	*MSt*	*TSt*	*PSw*	*ISw*	*MSw*	*TSw*
30° contracture	X	X	X	X	X	X	X	X
15° contracture	X	X	X	X			X	
15° elastic contracture	X	X	D				X	
Spastic calf	X	X	X	X	X			
Pretibial weakness	X	X					X	
Voluntary compensation for quadriceps weakness		X	X					

Key:
X = phases functionally affected by the designated pathology
D = delayed achievement of normal position

Plantar Flexion Contracture

The phases of gait that are altered by a PF contracture vary with the magnitude of DF lost as well as the rigidity of the tissues. A 15° PF contracture is most common because that is the position of minimal joint capsule tension.[3] It may be rigid or elastic. An elastic contracture is created by moderately dense tissues that stretch under the force of body weight but hold against manual testing. Other clinical circumstances, however, may introduce greater deformity. Hence, ankle PF contractures fall into 3 gross categories based on magnitude and rigidity: 30° PF contracture, rigid 15° PF contracture, and elastic 15° PF contracture. Each modifies different phases of the GC.

30° PF Contracture: Floor contact with a flat foot is difficult unless knee extension is excessive. Flexing the knee and striking the floor with the forefoot is much more common (see Figure 11-1) because this adaptation facilitates progression. As the 30° PF deformity exceeds the normal ranges of PF, there will be abnormal function in each phase of gait. There will be no heel contact in stance even with the slow walker. Instead, the forefoot will be the only mode of

support. Stride length will be shortened by the absence of the heel and ankle rockers. Each phase of swing is threatened by toe drag unless the patient has an adequate substitution.

Rigid 15° PF Contracture: Stiffness of the fibrous tissues determines the effect of the contracture. A rigid contracture can cause deviations in 5 of the gait phases. The severity of the deviations also varies with the vigor of the patient's walking ability. The typical picture associated with a 15° PF contracture is a slow walker with a foot flat mode of IC and lack of tibial advancement in loading response and mid stance. Slow walkers will lack the energy to roll up onto the forefoot. Persistent heel contact and an obstructed ankle rocker terminate the patient's ability to advance. Progression is restricted to the extent of available knee hyperextension.

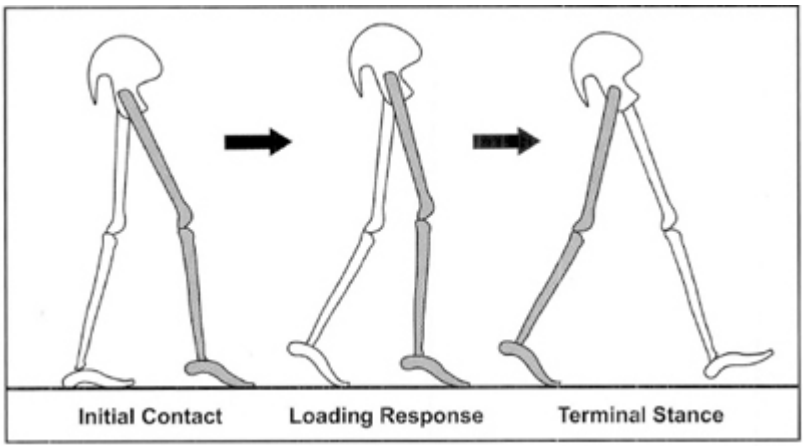

Figure 11-11. Vigorous walker achieves trailing limb posture despite a rigid ankle PF contracture.

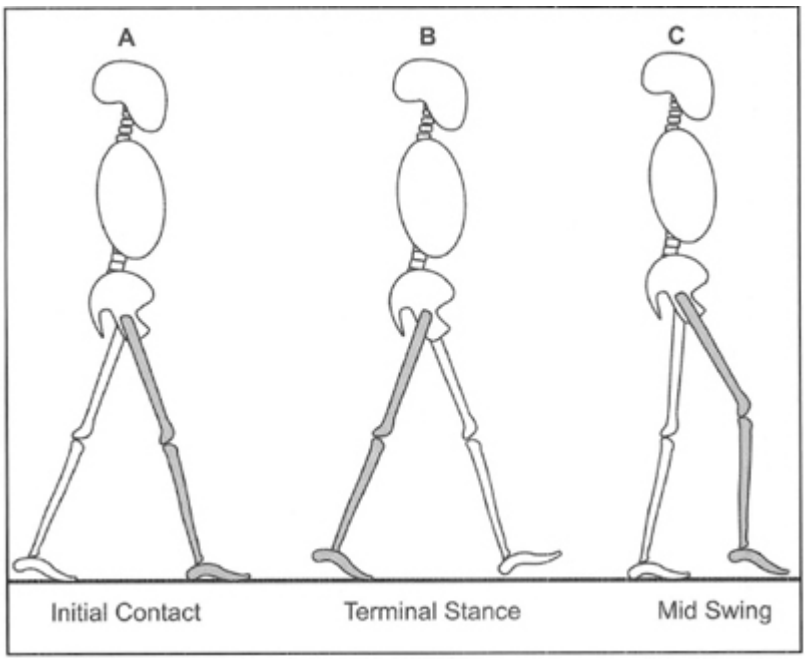

Figure 11-12. Fifteen degree elastic PF contracture. (A) IC foot flat. (B) Terminal stance with nearly normal heel rise. (C) Mid swing with excessively plantar flexed ankle requiring excess hip flexion for foot clearance.

In the vigorous walker, the deviations during mid stance will be obscured by a premature heel off occurring while the limb is still vertical (ie, technically in mid stance). A trailing limb posture is achieved as the body moves over the forefoot rocker, propelled by the energy of the swinging contralateral limb (Figure 11-11).

ST eversion can lessen the apparent amount of PF through 2 mechanisms. DF is a normal component of eversion. In addition, ST eversion unlocks the midtarsal joint, leaving it free to dorsiflex. These actions will reduce the angle between the forefoot and tibia.

In mid swing, there will be a toe-down foot posture similar to the passive drop foot from pretibial muscle weakness. Toe drag will occur if the patient is unable to substitute.

Elastic 15° PF Contracture: The flexibility of this contracture allows the ankle to yield under body weight during stance (Figure 11-12). As a result, the normal WA heel rocker is lacking. During mid and terminal stance, the restraint to tibial advancement presented by the elastic contracture may mimic that of normal soleus activity and, thus, induces no motion

abnormality. The rate of tibial advancement is slowed with stiffer tissues but this, generally, is an imperceptible deviation from normal function. In persons with concomitant plantar flexor weakness, the elastic contracture may serve as a “built-in orthosis” by providing resistance to tibial collapse during SLS. A dynamic EMG recording may be needed to differentiate soleus action from contracture. ST substitution is also common.

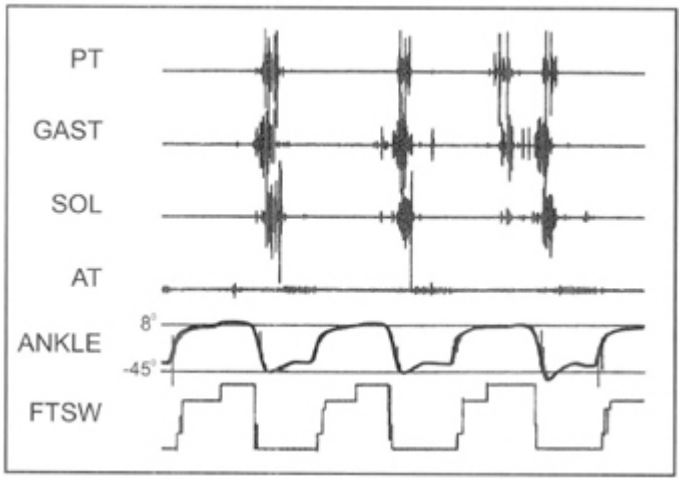

Figure 11-13. Tibialis anterior paralysis as a cause of excessive ankle PF in swing. The extent of the foot drop (45° PF) suggests a coexisting elastic contracture that partially stretched in stance under body weight.

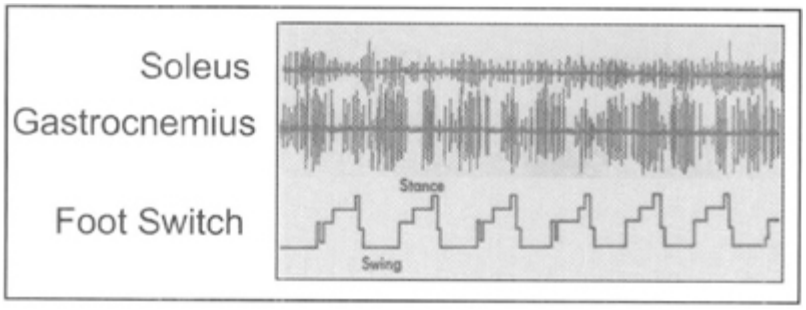

Figure 11-14. Excess PF due to prolonged, out-of-phase gastrocnemius and soleus activity.

In mid swing, the elastic contracture will create excessive PF similar to that of pretibial muscle weakness as the limited force provided by the dorsiflexor muscles is insufficient to stretch the

tissues. Functionally, these muscles are prepared only to rapidly lift the weight of the foot when there is virtually no resistance at the ankle. This effort is equivalent to grade 3 or fair strength.[1] Loading response, however, will not show an uncontrolled drop of the foot. As the secondary events are brief, a dynamic EMG recording may be needed to differentiate the effects of an elastic contracture from the lack of pretibial strength (Figure 11-13).

Soleus and Gastrocnemius Spasticity or Overactivity

With severe spasticity, the soleus and gastrocnemius may be active nearly continuously (Figure 11-14). Then the gait pattern is similar to a PF contracture of similar magnitude.

More commonly, the excessive action of the triceps surae muscles accompanies the primitive extensor muscle pattern. Terminal swing is the phase in which the primitive extensor pattern starts. As the quadriceps begins its action to extend the knee in preparation for stance, there is synergistic activation of the soleus and gastrocnemius. The ankle moves from a mid swing dorsiflexed posture to about 15° of PF. This rigidly plantar flexed ankle posture affects the patient's gait in each phase of stance from IC through pre-swing.

Initial swing and mid swing are periods of normal ankle DF. Activation of the flexor pattern to take a step terminates the extensor muscle action. The ankle promptly dorsiflexes to a near neutral position, and this persists through mid swing. It is this patterned reversal of ankle motion in swing that differentiates the spastic primitive extensor response from a PF contracture.

Voluntary Excessive Ankle Plantar Flexion

As a means of protecting a weak quadriceps from the usual knee flexion thrust of the loading response, patients with normal selective control deliberately reduce their heel rocker. Terminal swing is the usual time that the protective mechanics begin. Premature action by the soleus drops the foot into approximately 10° PF. The gastrocnemius is an inconsistent participant. TA muscle action may

continue to control the rate of the foot drop. The low heel strike at IC minimizes the heel rocker flexion thrust on the tibia. Loading response follows with rapid ankle PF, leading to foot flat and posterior tilt of the tibia as the calf muscles continue their strong activity. As a result, the knee remains extended and continuation of this posture requires little quadriceps effort during the rest of stance. The soleus and gastrocnemius dynamically restrain the tibia to preserve an extended knee throughout mid stance and terminal stance. Muscle intensity is graduated so as to decelerate but not prohibit tibial advancement. A delayed arc of relative DF preserves progression. Peak DF occurs late in pre-swing rather than during terminal stance and heel contact is continued. The progressive ankle DF differentiates the voluntary calf activity from PF contracture or soleus spasticity.

During the other gait phases, ankle DF is normal. The soleus has relaxed and the pretibial muscles become active.

Excess Dorsiflexion

Definition: DF that exceeds normal for a particular phase

Phases: All phases of stance

Functional Significance: During WA, excess DF accentuates the heel rocker, contributing to instability and increased quadriceps demand. During SLS, it disrupts the controlled forward progression provided by the ankle and forefoot rockers and results in an increased demand on the quadriceps to maintain stability. During pre-swing, excess DF shortens the leg length and thus reduces support for the pelvis. Roll-off and forward progression across the toe rocker will be limited. During the remainder of SLA, excess DF has only minimal impact except it requires additional energy and positions the foot poorly for IC.

Underlying Causes:

* WA: Ankle locked at neutral (eg, fusion, rigid orthosis) or secondary to excess knee flexion

* SLS: Weak calf, accommodation for a knee or hip flexion contracture, intentional during terminal stance to lower the opposite limb to the ground
* SLA: Weak calf (pre-swing), ankle locked at neutral (pre-swing), over-activity of pretibial muscles

DF beyond neutral is an abnormal event in all of the gait phases except mid stance and terminal stance. Diagnosing excess DF during these 2 stance phases presents a challenge. A 5° deviation can be considered within the normal variance yet it also may have functional significance as it still leads to considerable tibial tilt.

The term *excessive DF* also is used to indicate the lack of normal PF. This can occur during loading response and pre-swing. Excessive DF has more functional significance in stance than swing.

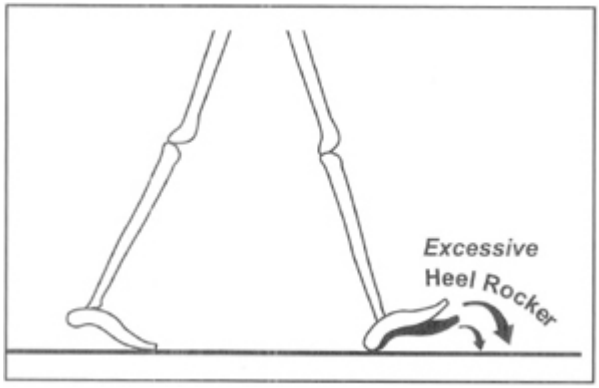

Figure 11-15. Excessive ankle DF at IC presents an exaggerated heel rocker response.

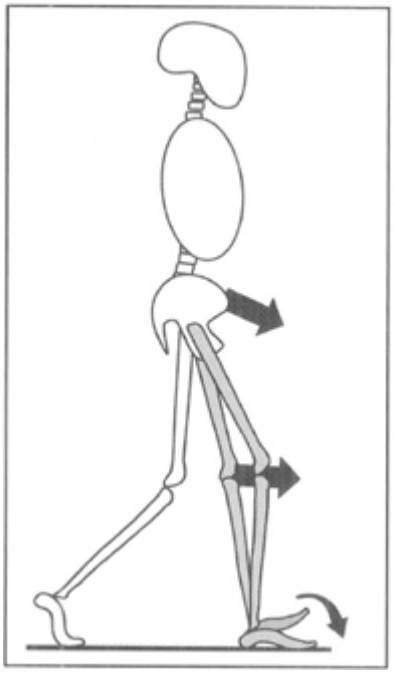

Figure 11-16. Excessive ankle DF during loading response increases the heel rocker, leading to greater knee flexion.

Impact of Excess Dorsiflexion by Phase

Initial Contact

Excessive DF at the time the heel contacts the floor is an infrequent finding. When it occurs, it is a position of instability. An exaggerated heel rocker has been established as the forefoot will be higher above the floor than normal (Figure 11-15).

Loading Response

Two forms of excessive DF are possible. There may be an abnormal form of IC or inhibition of the normal ankle PF.

Floor contact with the knee flexed and a flat foot eliminates the 5° of PF that normally accompanies the heel rocker action. This introduces a passive form of excessive DF as the limb is loaded. The potential for accelerated tibial advancement has been established.

Fixation of the ankle at neutral (0°) during the heel support period advances the tibia at the same rate as the foot falls to the floor, accentuating the heel rocker effect (Figure 11-16). There is a corresponding increase in quadriceps demand.

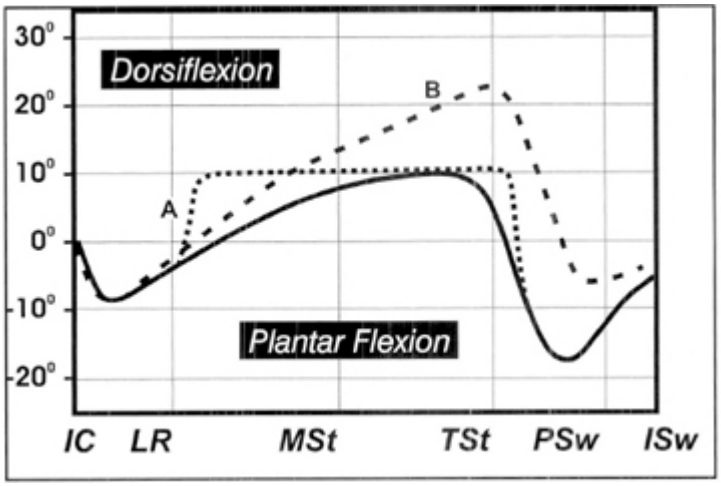

Figure 11-17. Excessive ankle DF in mid stance occurs in 2 patterns. (A) An abrupt change from loading response PF into DF at the onset of SLS due to profound plantar flexor weakness. (B) Progressive increase into excessive DF throughout mid and terminal stance when the plantar flexors are moderately weak.

Mid Stance

Two situations can make excessive DF in mid stance functionally significant. First is an accelerated rate of ankle DF from its initial position of PF (Figure 11-17A). The tibia collapses forward as soon as the vector moves anterior to the ankle joint. As momentum from the swing limb draws the body's COG forward, the tibia follows. Although the final position of the ankle in mid stance may not exceed 10° of DF because of limited passive range, the patient experiences the instability of excessively rapid DF at the onset of SLS versus terminal stance.

The second form of excessive ankle DF is a greater-than-normal angle being attained between the tibia and foot (Figure 11-17B). This is more marked in terminal stance.

Both situations (rate and magnitude) lead to increased quadriceps demand. The lack of tibial control also creates an unstable base for

the quadriceps, which prevents the muscle from fully extending the knee.

Terminal Stance

Excessive DF of the ankle during terminal stance is difficult to identify by observation because 2 actions tilt the tibia forward. These are heel rise and ankle DF. When heel contact continues through terminal stance, the ankle position suddenly becomes visible (Figure 11-18A). Now, even the normal 10° DF may appear excessive. Conversely, as elevation of the foot is more conspicuous than an increase in tibial angle, the combination of heel rise and excessive DF masks the change in ankle position (Figure 11-18B).

Pre-Swing

Whenever the normal 15° of PF is reduced, the ankle is in excessive DF. This most often occurs with prolonged heel contact as the body, by being well forward of the foot, draws the tibia forward (Figure 11-19). Excess DF during this period effectively shortens the limb and reduces support provided to the ipsilateral pelvis. An accentuated ipsilateral pelvic drop may occur during this phase.

Initial Swing, Mid Swing, and Terminal Swing

Seldom does the foot rise above neutral during swing. The only clinical significance relates to the position the ankle will be in at the time of IC.

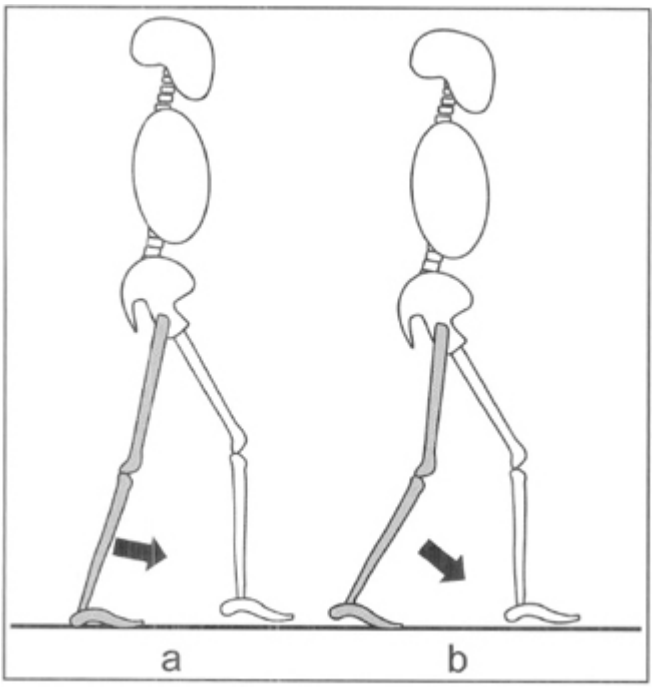

Figure 11-18. Excessive ankle DF in terminal stance can be identified by 2 gait deviations. (A) Prolonged heel contact makes the tibial advancement more apparent. (B) When a low heel rise accompanies excessive knee flexion, the excessive DF may be less observable.

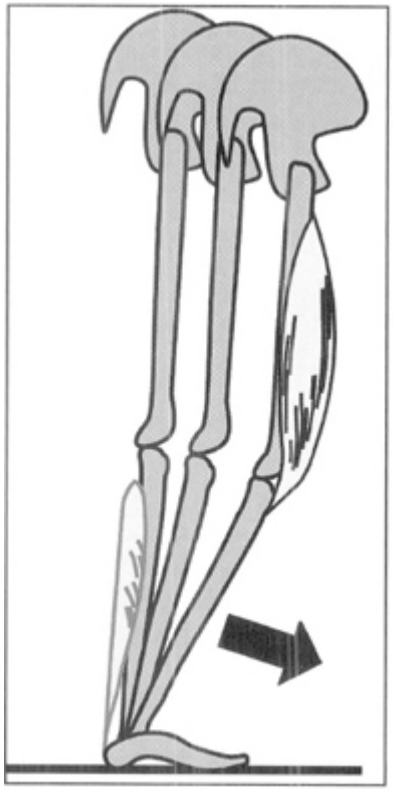

Figure 11-19. Excessive ankle DF in pre-swing represents a loss of the normal PF. Sustained heel contact and an accentuated pelvic drop are common.

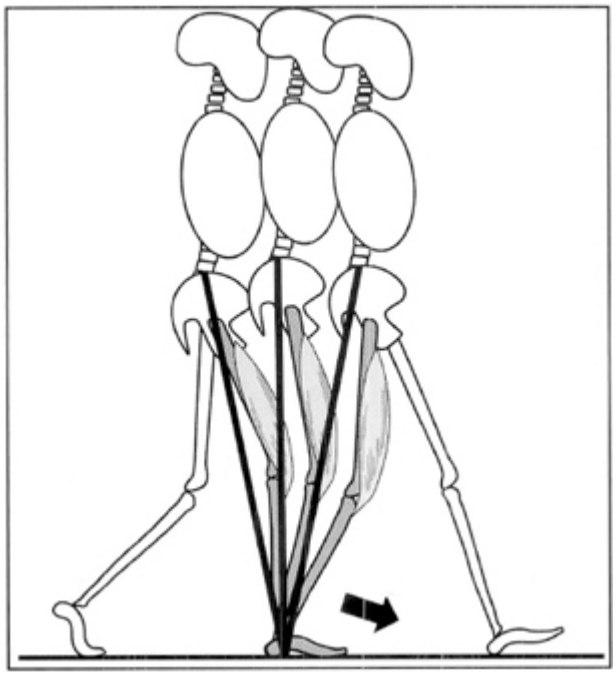

Figure 11-20. Soleus weakness fails to stabilize the tibia, causing sustained knee flexion. Without a stable base, the quadriceps cannot extend the flexed knee.

Detailed Causes of Excessive Dorsiflexion

Two primary conditions lead to excessive DF. These are soleus weakness and fixation of the ankle at neutral. Accommodating to a flexed knee during stance is another cause of excessive ankle DF. The functional significance of these mechanisms involves different phases in the GC.

Soleus Weakness

Loss of tibial stability during the weight-bearing period is the problem. This leads to increased demand of the quadriceps.

Mid stance advancement of the tibia over the foot rapidly moves the ankle into excess DF (exaggerated heel rocker) when the soleus response is inadequate. Forward tilt of the tibia perpetuates the flexed knee posture and the need for continued quadriceps support. The quadriceps activity prevents the knee from collapsing, but it is unable to re-establish knee extension. As the quadriceps acts to advance the femur, the entire proximal body mass (COG) moves forward (Figure 11-20). This moves the body vector farther anterior to the ankle joint and increases the demand on the weak calf muscles. Inability of the plantar flexors to provide adequate restraint leads to further ankle DF, and the sequence of calf muscle demand exceeding capacity continues. Gastrocnemius action contributes a knee flexion effect at the same time it is augmenting the soleus at the ankle.

Terminal stance heel rise also is lost with soleus weakness. This may occur even if the patient had a normal DF arc in mid stance because the intensity of muscular effort normally required during terminal stance is twice that of mid stance. Terminal stance knee extension is lost and replaced with persistent flexion.[5]

The common causes of inadequate triceps surae action are primary muscle weakness (disuse or paralysis) and excessive surgical lengthening of a tight Achilles tendon. The undesirable surgical outcome may result from the restoration of normal range when there is not sufficient neurological control to use such mobility advantageously. A second cause of “over lengthening” could be physiological muscle lengthening by the addition of sarcomeres to the muscle fiber chain in response to the repeated stretch experienced in gait.[6]

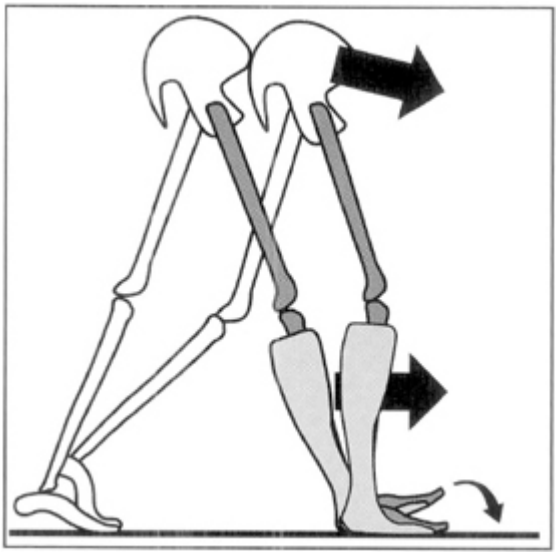

Figure 11-21. A rigid ankle-foot orthosis (AFO) or an ankle fusion set at neutral causes excessive ankle DF at loading response. The effect is excessive knee flexion as the tibia follows the foot.

There generally is gastrocnemius weakness associated with the lack of soleus action, but it is does not directly contribute to the excessive ankle DF. Gastrocnemius action combined with a weaker soleus can accelerate tibial advancement through its effect at the knee.

Substitution for soleus weakness is difficult as momentum draws the limb forward over the supporting foot throughout stance. If the patient has normal quadriceps strength, no effort is made to accommodate the weak calf. Instead, the subject walks with knee flexion. Hence, the need to substitute for excessive DF relates to knee control inadequacy (see the knee section).

If the patient has a weak quadriceps in combination with a weak calf, then walking on a flexed knee will not be ideal as the demand on the quadriceps increases with greater degrees of flexion. Instead, loading response knee flexion will be avoided and knee extension will be maintained throughout stance. Knee recurvatum (hyperextension) may be used to tilt the tibia posteriorly to decrease demands on the plantar flexors while also aligning the body vector anterior to the knee joint to provide passive knee extension stability.

Other measures to reduce plantar flexor demand include the use of shorter steps and a slower walking speed.[4] There is a good correlation between walking velocity and ankle plantar flexor strength.[7]

Ankle Locked at Neutral

Either a pantalar fusion (ankle and ST joints) or an orthosis fitted with a locked ankle joint (PF stop) causes excessive DF by obstructing the normal PF that occurs as the limb is loaded. Right angle rigidity between the tibia and foot increases the heel rocker action (Figure 11-21). The initial early free fall of the foot now carries the tibia with it. As a result, the knee flexes at the same rate as the foot falls, rather than at half of that speed. Quadriceps demand is correspondingly increased. The ability to tolerate the locked ankle thus depends on quadriceps strength.

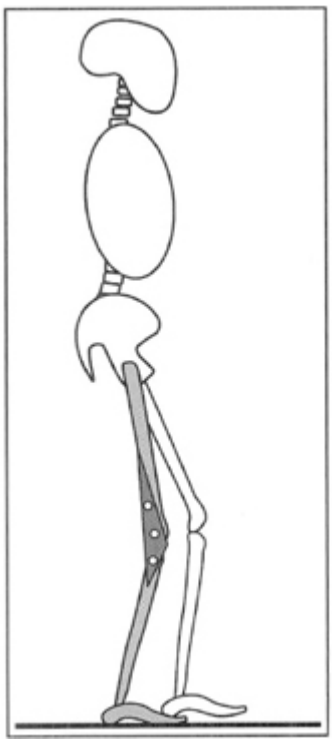

Figure 11-22. Excessive ankle DF allows an upright posture in the presence of a fixed knee flexion contracture.

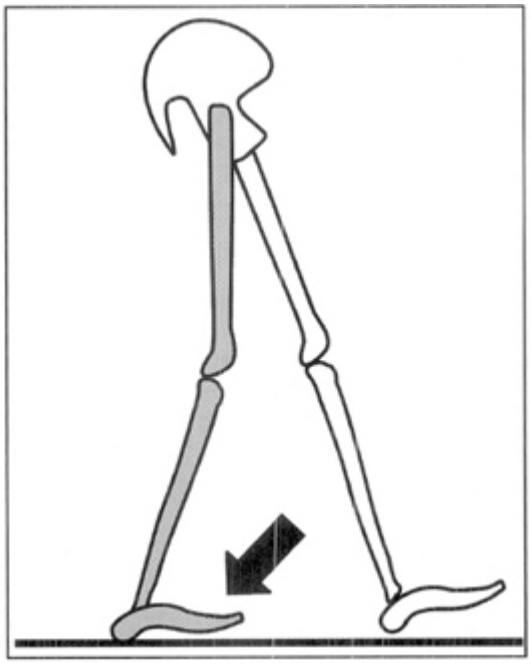

Figure 11-23. Prolonged heel-only contact into SLS curtails trailing limb posture.

Stance Knee Flexion

Persistent knee flexion during the foot flat support period (mid stance) requires ankle DF beyond neutral in order to align the body vector over the foot for standing balance. The amount of DF required is proportional to the flexed knee posture (Figure 11-22).

Prolonged Heel Only

Definition: The heel-only period extends beyond loading response

Phases: Loading response, mid stance, terminal stance, pre-swing

Functional Significance: Disrupts stability and forward progression

Underlying Causes:

* Painful forefoot
* Toe clawing associated with spasticity

This is an infrequent finding. Forefoot contact is avoided by extending the normal loading response pattern of heel only into mid stance and continuing until stance is ended (Figure 11-23). The cause is either a painful forefoot or spastic toe clawing. Weight-bearing pain is minimized by excessive ankle DF. The intensity of the pretibial muscles will be increased and that of the plantar flexor muscles reduced.

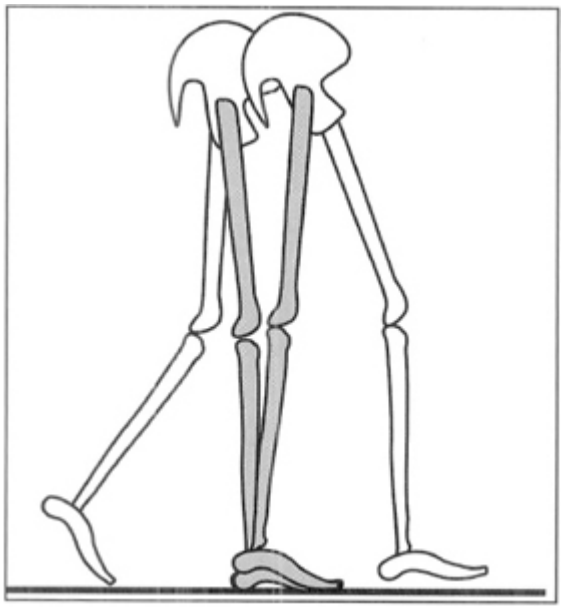

Figure 11-24. Premature heel rise in mid stance due to PF contracture.

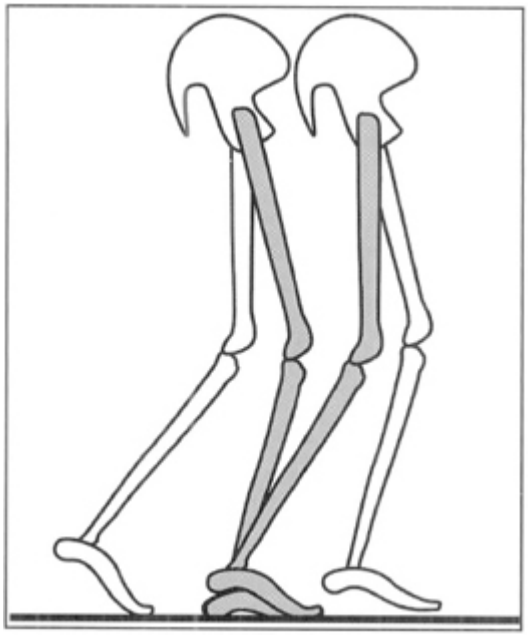

Figure 11-25. Premature heel rise in mid stance due to knee flexion contracture. Note that the ankle is not plantar flexed.

PREMATURE HEEL-OFF

Definition: The heel is not in contact with the ground when it should be

Phases: IC, loading response, mid stance

Functional Significance: Disrupts heel and ankle rockers and associated forward progression

Underlying Causes:

* Increased plantar flexor activity
* Heel pain
* Voluntary substitution for a short limb or to assist floor clearance by the other limb
* Secondary to excess knee flexion

Loss of heel contact is an abnormal event in IC, loading response, and mid stance. The most severe disability is the lack of heel contact throughout stance (continuous forefoot support). Premature heel rise is readily recognizable during the first half of stance but more difficult to identify in late mid stance due to the natural timing of heel rise during terminal stance.

Premature heel rise during loading response or early mid stance can occur following a normal heel-first IC if momentum is sufficient to roll the limb onto the forefoot. The primary causes of premature heel rise are excessive ankle PF (eg, the presence of a PF contracture or spasticity; Figure 11-24) and excessive knee flexion (Figure 11-25). Premature heel rise also may be voluntary. Deliberate PF is used to accommodate a short limb. Vaulting by active, excessive ankle PF raises the body to assist floor clearance by the impaired contralateral limb.

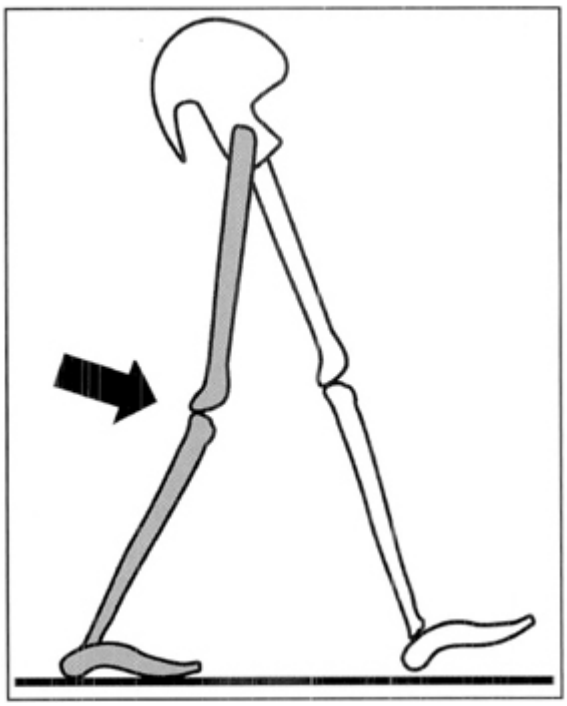

Figure 11-26. Delayed heel rise due to weak calf. Note excessive knee flexion.

NO HEEL-OFF/DELAYED HEEL-OFF

Definition: Absence of a heel rise when the heel should be off the ground

Phases: Terminal stance, pre-swing

Functional Significance: Disrupts forefoot and toe rockers, reduces forward progression, shortens contralateral step length, and limits pre-swing knee flexion

Underlying Causes:

* Weak calf
* Forefoot pain
* Secondary to inadequate toe extension
* Secondary to excess DF

Absence of the normal heel rise is an error seen in terminal stance or pre-swing. Persistent heel contact indicates either plantar flexor muscle weakness or excessive ankle PF with inhibition of limb advancement in a slow walker. Terminal stance represents the

period of peak demand on the 7 plantar flexor muscles of the ankle (soleus, gastrocnemius, TP, FHL, FDL, peroneus longus, and peroneus brevis). Weakness particularly of the soleus, gastrocnemius, and TP may prevent adequate stabilization of the ankle and midfoot for the heel to pivot over the forefoot rocker (Figure 11-26). Limb length is shortened by the loss of the elevated heel height and the ankle is not prepared for pre-swing push off.

Delay of heel rise at the beginning of terminal stance is a more significant sign of a weak soleus than its occurrence at the end of the phase. In late terminal stance, the mere trailing posture of the limb will lift the heel once the ankle reaches the end of its passive DF range.

Pre-swing is a period of double limb support, and body weight is rapidly transferred to the forward limb. The reduced demands on the weak soleus may allow the heel to rise as body weight rolls over the forefoot. By the end of stance, knee flexion also will roll the tibia forward enough to lift the heel. Hence, the timing of the delayed heel rise has functional significance.

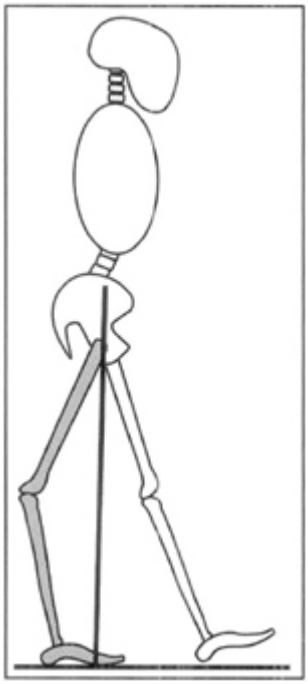

Figure 11-27. Delayed heel rise secondary to knee hyperextension. The knee is locked by anterior alignment of the body vector and posterior tilt of the tibia, which shortens the vertical height of the leg.

MTP DF is a prerequisite for a normal heel rise. Toe flexor spasticity or contracture may prevent the toes from extending in terminal stance, and thus block heel rise.

Persons with pain in the forefoot region may avoid raising the heel. Lifting the heel would concentrate the forces under the small forefoot area, leading to an increase in pressure in the region of pain.

Knee hyperextension can also delay heel off because the body weight vector falls anterior to the knee and inhibits normal tibial advancement for knee flexion. Pre-swing knee flexion is delayed until the foot is completely unloaded as the backward thrust on the tibia has locked the knee (Figure 11-27).

DRAG

Definition: Contact of the toes, forefoot, or heel with the ground during swing

Phases: Initial swing, mid swing, or terminal swing

Functional Significance: Disrupts forward progression and foot clearance and may precipitate a fall

Underlying Causes:

* Limited hip flexion
* Limited knee flexion
* Excess PF

Failure to adequately lift the foot for ground clearance may be a brief error in initial swing or it may continue throughout the swing period. Toe drag in initial swing causes tripping as advancement of the limb is delayed. If the foot cannot reach its forward position to receive the advancing body weight, the subject will fall. Persistent toe drag into mid swing shortens the swing phase.

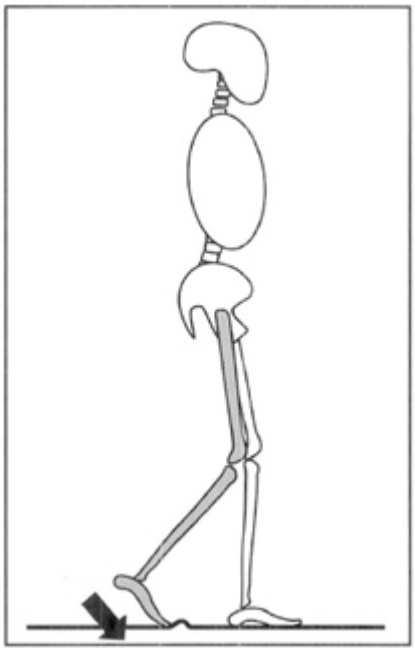

Figure 11-28. Inadequate knee flexion during initial swing fails to lift the lower leg for toe clearance.

During initial swing, the most frequent cause of toe drag is inadequate knee flexion (Figure 11-28). Knee flexion may be limited due to knee extensor spasticity, weak flexor pattern, or difficulty achieving a normal trailing limb posture (please refer to Chapter 12).

During mid swing, a drag can arise from either inadequate hip flexion or failure to achieve a neutral ankle position (see Figure 11-9). Foot clearance in mid swing is generally limited to only 1 to 2 cm, and thus, even subtle position changes can alter the limb's ability to clear.

A drag in terminal swing is less common. Failure to adequately clear the foot during mid swing may contribute to continued problems in terminal swing.

CONTRALATERAL VAULTING

Definition: Prematurely rising onto the forefoot of the contralateral stance limb during SLA of the reference limb

Phases: Initial swing, mid swing, terminal swing

Functional Significance: Decreases contralateral limb stance stability and increases calf muscle demand

Underlying Causes: Voluntary compensation by the other limb to assist the reference leg in clearing the ground if the limb is too long (eg, leg length discrepancy) or inadequate knee flexion

Deliberate premature contralateral heel rise lifts the body and assists with floor clearance when the reference limb is too long. A leg length discrepancy is one common cause of contralateral vaulting. A second source is failure to achieve the normal 60° of knee flexion required for foot clearance during initial swing. In mid swing, inability to attain a neutral ankle posture represents a third situation that may necessitate a compensatory contralateral vault (Figure 11-29).

Contralateral vaulting increases the muscular demand on the stance limb plantar flexors as the associated increase in ankle PF is not optimal for generating plantar flexor muscle force. Additionally, stability under the contralateral stance limb is reduced as balance must be maintained across a relatively small surface.

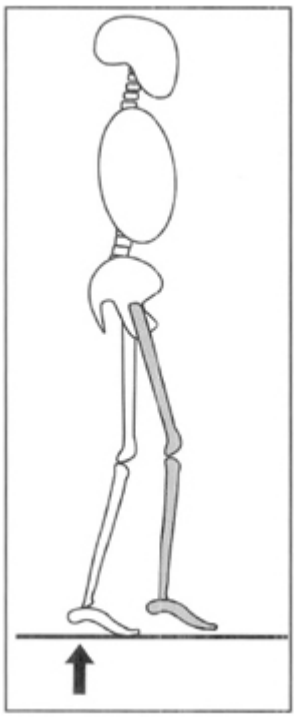

Figure 11-29. Vaulting on the left limb to allow the equinus right foot to clear the ground.

SUBTALAR JOINT DEVIATIONS

Motion at the junction between the talus and calcaneus is best observed in the coronal view (standing behind the patient). Excess inversion (the calcaneus rotated medially relative to the talus) and excess eversion (the calcaneus rotated laterally relative to the talus) are identified when the expected joint posture for a given phase is exceeded. As the normal arc of motion at the ST joint is relatively small, this is sometimes difficult to detect. Subtle changes in medial arch height, as well as the contact pattern of the foot with the floor, may aide in identifying these frontal plane deviations.

EXCESS INVERSION

Definition: Inversion of the calcaneus or forefoot that exceeds neutral for a particular phase

Phases: All phases

Functional Significance: Excess inversion moves the body weight to the lateral side of the foot, which provides an unstable base of support. During WA, the associated rigid midfoot is less able to absorb shock. During SLA, the primary penalty is difficulty with foot clearance as the lateral side of the foot drops.

Underlying Causes:

* Overactivity of the inverting muscles
* Equinovarus contracture
* Patterned movement
* Skeletal deformities

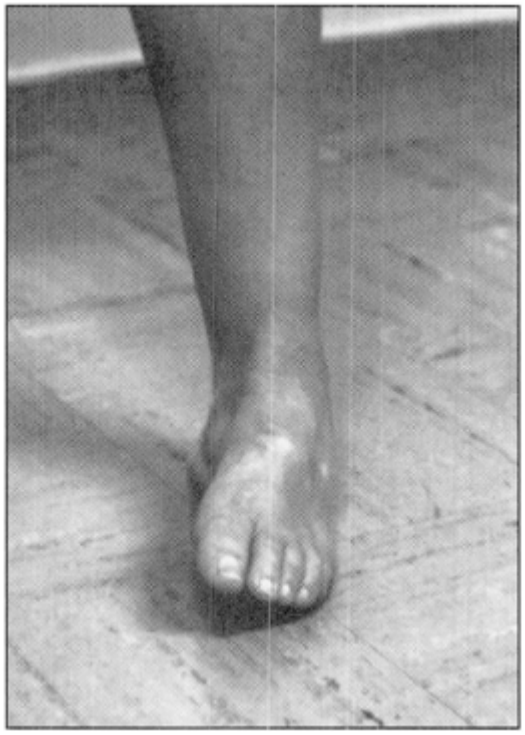

Figure 11-30. Mid stance excess inversion. First metatarsal lifted from floor and height of arch increased.

Because all of the muscles controlling the ankle cross the ST joint as they insert on the foot, inversion abnormalities commonly occur in combination with ankle dysfunction. Forefoot and/or hindfoot postures can be altered. Excessive inversion is also commonly called *varus*. Inversion of the heel is displayed by medial tilt of the calcaneus under the talus. Deviations in heel support are most visible from a posterior view of the foot. Forefoot deviations are identified by the floor contact pattern of the metatarsal heads. Particular attention must be directed to the first and fifth metatarsals, which represent the outer margins of the forefoot. Forefoot varus during stance is characterized by elevation of the first metatarsal head from the floor (Figure 11-30). Arch height also tends to be increased with inversion. In addition, the forefoot may be adducted.

Abnormal muscle control is the leading cause of excess inversion. Five muscles cross the ST joint on the medial side. All are aligned to invert the foot (Table 11-2). Four of the muscles (all but the anterior tibialis) also are plantar flexors. Hence, equinovarus is a common pathological finding when these muscles are overly active. Gait

deviations generally result from the muscles being active at a time they normally are relaxed. The most common timing error is premature or prolonged action. Occasionally, there is a reversal of phasing (ie, stance instead of swing or vice versa). Muscles that have significantly different levels of normal activity (ie, soleus) also introduce gait deviation by an increase in intensity.

Abnormal Tibialis Anterior and Toe Extensor Activity

In persons with spasticity, inversion of the forefoot at IC may either persist into the loading phase or cease. TA action in swing is part of the primitive flexor synergy but not the toe extensors. This causes swing phase foot inversion (varus). When the patient's limb control is dominated by the primitive locomotor patterns, the TA ceases to contract with the onset of the extensor pattern in stance (Figure 11-31). As a result, the inversion in swing abruptly reverses to neutral or even eversion.

Table 11-2

Causes of Excessive Varus and Phasing

	IC	*LR*	*MSt*	*TSt*	*PSw*	*ISw*	*MSw*	*TSw*
Anterior								
TA			X	X	X	X		
Toe extensors	O	O				O	O	O
Posterior								
Soleus	X	X						X
TP						X	X	X
FHL	X	X				X	X	X
FDL	X	X				X	X	X

Key:
X = phases of inappropriate activity contributing to pathology
O = inactivity contributing to varus

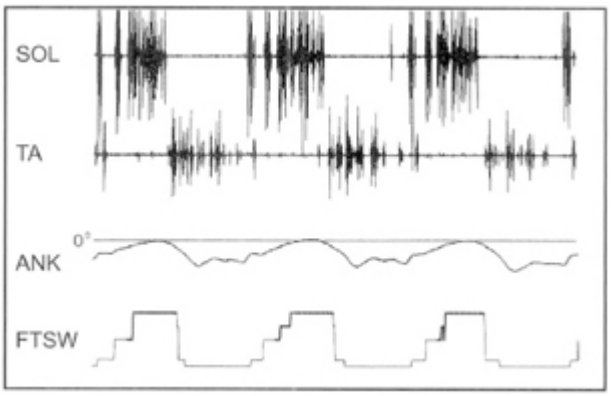

Figure 11-31. Primitive muscle pattern. Anterior tibialis EMG starts with the onset of swing and decreases to a nonsignificant intensity in terminal swing. Soleus (SOL) action begins prematurely with clonic beats at onset of stance and ceases with onset of swing. ANK = ankle electrogoniometer. 0° = neutral. Stance DF just to zero and PF in swing indicate a contracture that the moderately active TA could not oppose. FTSW = footswitch of reference limb. Stance pattern indicates IC with MT5, progression to H-5, H-5-1, and prolonged first and fifth metatarsal (heel off). Baseline is swing interval.

Prolonged inversion into mid and terminal stance may include stance action of the TA as well as the other inversion forces (Figure 11-32). Most commonly, the patient has an equinovarus. This accounts for 75% of the pathological varus in cerebral palsy[9] and probably a much higher percentage in patients who have experienced a stroke.[8]

Swing phase inversion with good ankle DF is evidence of strong TA action without toe extensors' participation (Figure 11-33). This is the common deviation in individuals with spasticity. While visually prominent, there is no functional significance to swing phase varus except at the end of terminal swing. Then foot posture determines the mode of floor contact.

When the pretibials are weak, the foot assumes an equinovarus position during swing. As the natural ankle alignment is slightly oblique, passive PF (ie, the passive drop foot) includes a small amount of inversion. Weak unassisted TA action is capable of inverting the foot but lacks the strength to accomplish full DF.

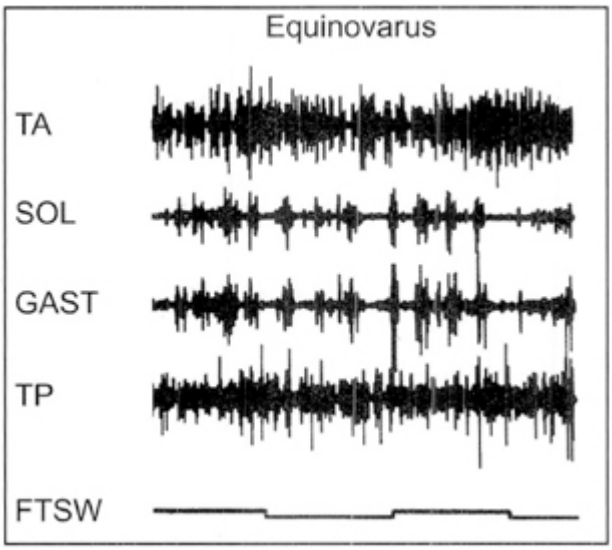

Figure 11-32. Stance pattern is equinovarus with contact only by the fifth metatarsal (elevated region on foot switch [FTSW] indicates fifth MT contact). EMG shows continuous activity of the tibialis anterior (TA), tibialis posterior (TP). Soleus (SOL) and gastrocnemius (GAST) action is clonic and continues to swing. Hence, there are 4 dynamic sources of varus.

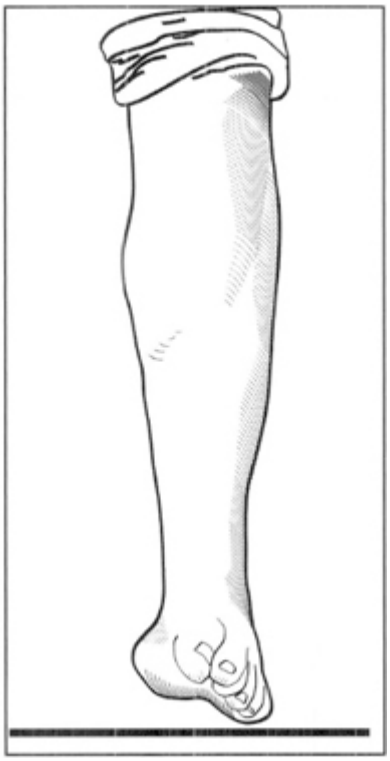

Figure 11-33. Inversion of the foot during swing gives the appearance of equinus because the lateral border of the foot drops

down.

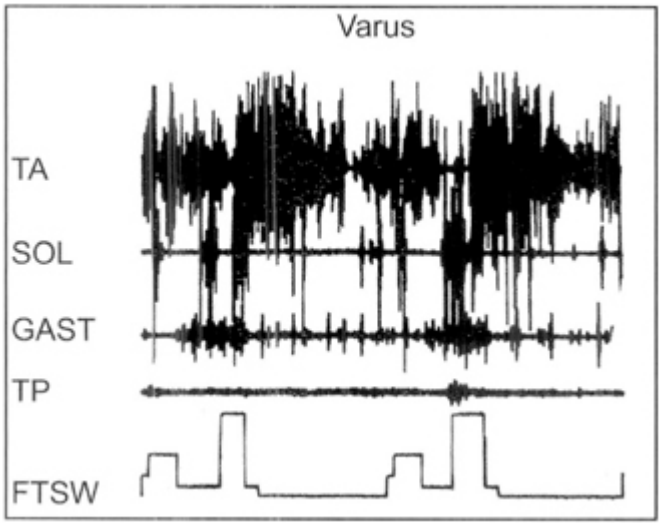

Figure 11-34. Tibialis posterior (TP) inactive though the foot support is varus. Tibialis anterior (TA) strongly active in stance and swing. Soleus (SOL) action is clonic and intermittent. Gastrocnemius (GAST) action is of low intensity with intermittent low spikes in swing. Footswitch (FTSW) pattern shows an unstable varus foot support pattern: H-5, MT-5, and 5-1 (heel off).

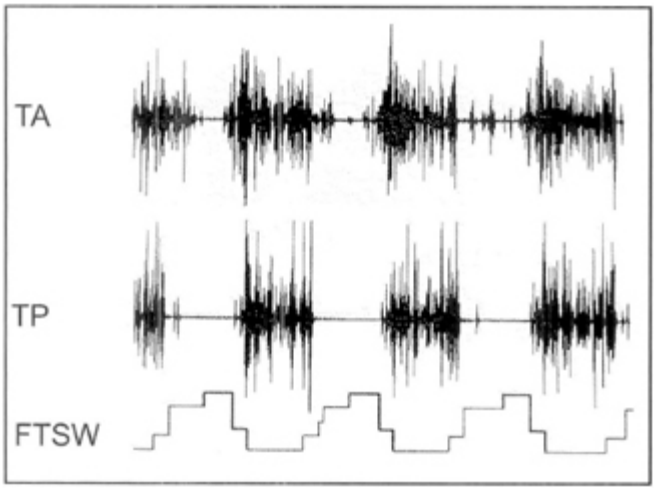

Figure 11-35. Tibialis posterior (TP) out of phase and synchronous with tibialis anterior (TA) throughout swing. Footswitches (FTSW) display a slightly inconsistent normal sequence of pattern (H, H-5-1, 5-1).

Abnormal Tibialis Posterior Activity

While normal gait involves the TP during stance, the activity of this muscle in patients is very inconsistent in both timing and intensity. Hence, one cannot assume a patient's varus is related to excessive or premature TP action. TP may be totally quiescent (Figure 11-34). When active, however, it is a significant source of ST varus as it has the longest leverage among the 5 inverting muscles (see Figure 4-15). Phase reversal to swing action occurs in about 11% of patients with cerebral palsy (Figure 11-35).[2]

Abnormal Soleus Activity

While primarily positioned for PF, the soleus also has good inversion leverage at the ST joint. This becomes significant because of the muscle's size (five times the size of the TP). Two abnormalities in soleus action lead to its contribution to inversion. Premature onset in terminal swing as part of the primitive extensor synergy prepositions the foot into varus and then maintains this undesirable posture throughout stance (Figure 11-36). The primitive control pattern also activates the soleus at a higher intensity during loading response and mid stance than is normally needed before the terminal stance heel rise. As this is greater than the demand presented by the body vector, equinovarus results. Spasticity commonly increases the intensity of the soleus response.

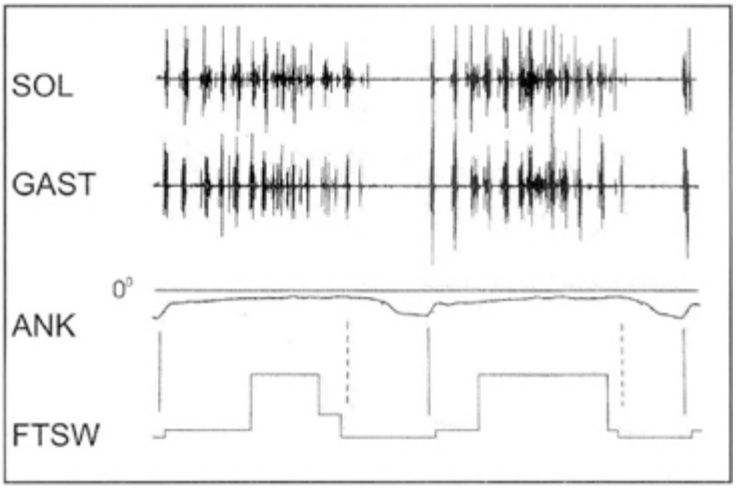

Figure 11-36. Soleus (SOL) and gastrocnemius (GAST) premature action. A clonic pattern of EMG begins with IC and persists through stance. Ankle motion (ANK) is continuous equinus (below 0°). Foot switch (FTSW) indicates varus (MT5 IC) progressing to equinus (prolonged MT5-1).

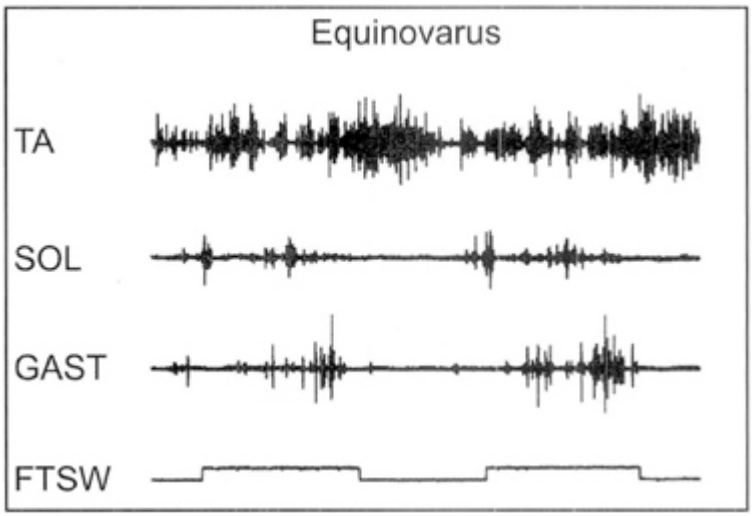

Figure 11-37. Soleus (SOL) and gastrocnemius (GAST) activity reduced by contracture. EMG sparse and low intensity. The combined effect is an equinovarus foot with support by only the fifth metatarsal (by FTSW).

A PF contracture will contribute to excess inversion across all phases of the GC. If it is elastic, it may be reduced during stance due to the weight-bearing forces. A PF contracture can reduce the level of soleus and gastrocnemius activity required to stabilize the ankle by providing static resistance to tibial collapse (Figure 11-37).

Abnormal Flexor Hallucis Longus and Flexor Digitorum Activity

These muscles commonly are included in the primitive extensor pattern and, thus, are prematurely activated in terminal swing. In addition, spasticity is frequently present in these muscles. As was described for the soleus, this leads to increased intensity of muscle action as well as premature timing. Both factors contribute to varus.

Such overactivity is implied by the toe clawing sometimes observed in stance.

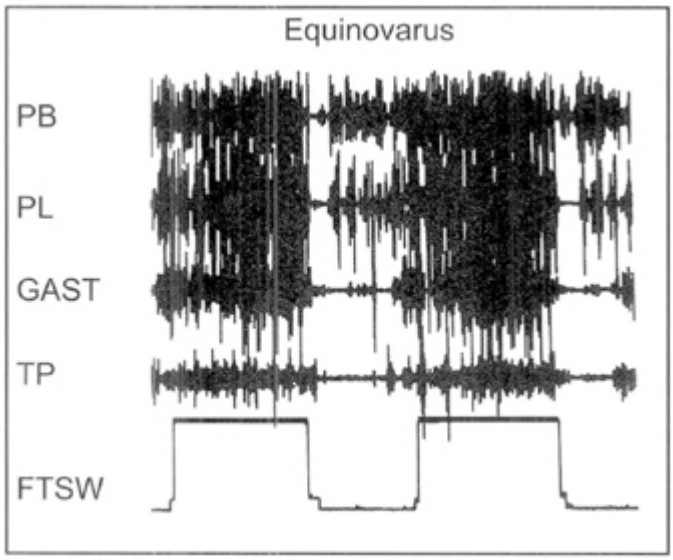

Figure 11-38. Strong activity in peroneus longus (PL) and peroneus brevis (PB) provides lateral stability during weight bearing on an inverted foot. Gastrocnemius (GAST) and tibialis posterior (TP) are also active through stance. Footswitch (FTSW) shows forefoot support (5-1) with MT5 at start and end of stance.

Abnormal Peroneus Longus and Peroneus Brevis Activity

Patients who are able to walk with either varus or equinovarus display strong peroneal action by EMG testing. Generally, both peroneals are active but either may dominate. Gastrocnemius activity also may be strong in equinovarus. Thus, insufficient evertor muscle action is not a common component of varus gait (Figure 11-38).

When peroneal muscle action is insufficient to meet the stance phase demands, the foot will be incapable of safely accepting body weight. The foot and ankle will twist severely and generally there will be pain as the lateral tissues are strained.

Excess Eversion

Definition: Eversion of the calcaneus or forefoot that exceeds normal for a particular phase

Phases: All phases

Functional Significance: During WA, excess outward tilt increases rotary stress in the ankle mortise and at the knee. Excessive eversion during SLS unlocks the midfoot and prevents achieving the rigid forefoot required for heel off and forward progression. When ankle mobility is limited, excess eversion can be used to gain increased DF range during stance.

Underlying Causes:

* Weak inverters
* Compensation for a PF contracture to gain increased DF ROM
* Peroneal hypertonicity
* Skeletal alignment variations resulting in a low arch
* Valgus deformity

Excessive eversion is commonly referred to as valgus. Excessive eversion during stance most frequently results from total weakness of the inverters (eg, anterior tibialis, TP, soleus) whether the primary pathology is flaccid or spastic paralysis, rather than excessive peroneal action (Figure 11-39). TA and TP normally modulate the rate and amount of ST eversion during WA. Weakness of these 2 muscles leads to a visually apparent eversion “thrust,” with the ST joint abruptly everting as weight is loaded onto the limb.

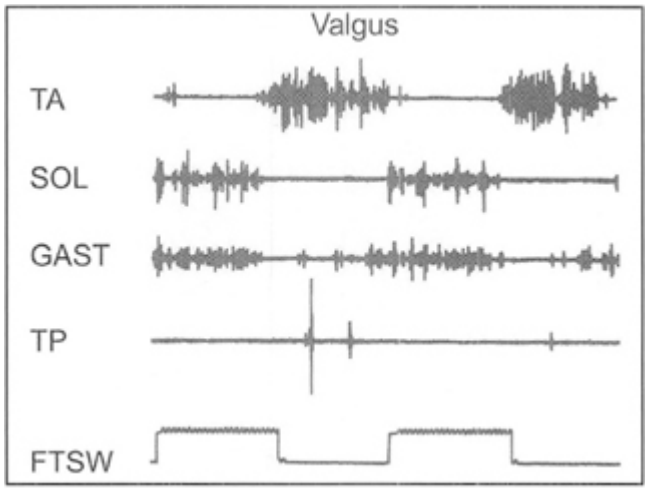

Figure 11-39. Everted foot due to weak inverter muscle action. Tibialis posterior (TP) inactive. Tibialis anterior (TA) activity occurs in swing only (loading response action is lacking). Soleus (SOL) and gastrocnemius (GAST) EMG of low intensity. Footswitch (FTSW) shows eversion: H-1, great toe (wavy overlay).

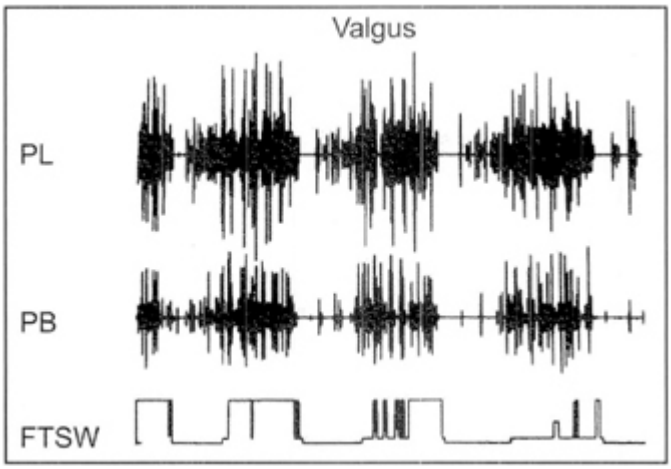

Figure 11-40. Peroneus longus (PL) and peroneus brevis (PB) excessive activity. Intense, premature onset, high amplitude, and dense EMG. Footswitches (FTSW) show eversion: H-1.

Eversion of the calcaneus decreases the support to the head of the talus. This allows it to drop, which causes internal rotation of the tibia. Internal rotation of the tibia also may be accentuated as the ankle mortise follows the path of the head of the talus. Rotary strain on the midfoot and knee results.

As body weight advances onto the forefoot, depression of the midfoot becomes more apparent as ST eversion also has unlocked

the midtarsal joints, allowing them to sag into DF. Whether the arch collapses or persists depends on its structural integrity (ligamentous laxity). The “too many toes” sign, one indicator of a weak TP, can be observed during the stance period from behind. An excessive number of toes will appear lateral to the ankle joint on the involved limb due to the forefoot abduction, which is associated with the valgus. Excess eversion in terminal stance also disrupts the normal heel rise as the axes of CC and TN joints are not optimally aligned to achieve a rigid forefoot.

Very infrequently the cause of stance phase valgus is premature, strong peroneal muscle action accentuating the elevation of the lateral foot (Figure 11-40). With weakness of the other plantar flexors, occasionally peroneal muscle action can lead to a calcaneovalgus ankle/foot posture.

Eversion in swing always is abnormal as the natural imbalance in the dorsiflexor muscles tends toward varus. Patients disabled by flaccid and spastic paralysis present different everting mechanisms. Flaccid TA paralysis, such as occurs in poliomyelitis, may create eversion in swing.

Strong toe extensor and peroneus tertius muscles can suspend the lateral side of the foot while a weak or absent TA muscle allows the medial side to drop. Floor clearance is less complete and the foot is everted. This posture persists through all 3 gait phases.

TOE DEVIATIONS

During normal gait, the toes serve as the final point of contact between the lower limb and the ground prior to lifting the foot for swing. Deviations occurring at the junction between the heads of the metatarsals and the phalanges often block the smooth transition of body weight across the toe rocker (eg, claw toes or toes that inadequately extend). Sometimes, however, toe deviations facilitate a more normal gait pattern by helping the foot clear the ground in the presence of weakness (eg, excess toe extension when the anterior tibialis is weak).

Excess Toe Extension

Definition: Extension of the toes that exceeds normal for a particular phase

Phases: All phases

Functional Significance: During WA, excess toe extension may reflect increased use of the long toe extensors if the TA is weak. During SLA, excess toe extension may assist with foot clearance.

Underlying Causes:

* Weak TA
* Toe extensor hypertonicity

Normally, the MTP joints are positioned near neutral in all phases except for IC (25° extension), terminal stance (25° extension), and pre-swing (55° extension). From initial swing through loading response, observation of toe extension well beyond neutral serves as a “red flag” that the anterior tibialis may be weak. A compensatory increase in activity of the EHL and EDL assists with foot clearance in swing and controls forefoot lowering during WA.

When excess toe extension leads to rubbing inside of the shoe, calluses or skin abrasions may develop. An extra-depth toe box may be required to safely encompass the toes.

Limited Toe Extension

Definition: Less extension at the MTP joint than is normal for a particular phase

Phases: Terminal stance, pre-swing

Functional Significance: Interferes with forefoot and toe rockers and reduces contralateral step length

Underlying Causes:

* Contracture
* Hallux rigidus
* Toe flexor spasticity

* Forefoot pain
* Any factor limiting heel off in terminal stance or pre-swing

Contractures and arthritic changes (eg, hallux rigidus) at the MTP joint obstruct people's ability to role over their forefoot. If just the great toe is involved, the foot will be displaced into varus. Applying a rocker (toe taper) to the anterior aspect of the shoe's sole can alleviate progression problems.

Spasticity of the toe flexors (FHL, FDL) is aggravated by ankle DF and/or MTP extension. This can be easily overlooked in the patient who has equinus. The excessive activity can block normal toe extension in terminal stance and pre-swing.

Pain in the forefoot (eg, metatarsalgia) may result in a reduced heel off in an effort to avoid concentrating pressure across the painful region. Similarly, if the heel fails to rise from the floor at the end of stance due to plantar flexor weakness, toe extension will not be required.

CLAWED TOES

Definition: Flexion of the interphalangeal joints

Phases: Terminal stance, pre-swing

Functional Significance: Disrupts the smooth transition of body weight across the forefoot and toe rockers and reduces contralateral step length

Underlying Causes:

* Toe flexor hypertonicity
* Imbalance between the strength of the long toe flexors and the intrinsic foot muscles
* Compensation for a weak gastrocnemius and soleus

Muscle imbalance with or without weakness can contribute to clawed toes. Inappropriately timed or high-amplitude activity of the FDL and FHL can lead to clawed toes in both stance and swing. In late stance, this posture can block the ability to rise onto the heel or contribute to pain in the forefoot region.

Peripheral motor neuropathy (as with diabetes mellitus) often is associated with a force imbalance between the long toe flexors and the intrinsic foot muscles, resulting in clawed toes. When sensory neuropathy is present in the feet, the clawed toes may pose a risk factor for ulcers on the dorsum of the toe surface unless footwear adequately accommodates the increased height of the toes.

The posterior course of FDL and FHL relative to the ankle joint positions these muscles to serve as plantar flexors. When the soleus and gastrocnemius are weak, the long toe flexors may exhibit increased muscle activity to augment the plantar flexor force. Their primary action as toe flexors leads to clawing.

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Chapter 12

Knee Gait Deviations

The most common types of knee dysfunction occur in the sagittal plane. Inappropriate arcs of motion result in excessive or inadequate flexion or extension. Less frequent are the deviations in the coronal plane (excessive valgus or varus). Excessive transverse plane rotation within the knee is reported but the findings vary with the method of measurement. This results in a major inconsistency between laboratories, although each facility has confidence in its technique.[1]

SAGITTAL PLANE DEVIATIONS

Six terms have been used to identify abnormal sagittal plane knee motion patterns: *limited knee flexion, knee hyperextension, extensor thrust, excess knee flexion, excess contralateral knee flexion,* and *wobble*. The first 3 terms reflect a bias toward maintaining a more extended knee than is normally expected in a given phase (limited knee flexion, knee hyperextension) or rapidly moving the knee toward extension (extensor thrust). The next 2 terms, *excess knee flexion and excess contralateral knee flexion*, indicate that the ipsilateral or contralateral knee is more flexed in a given phase than is normally expected. Wobble defines an alternating movement between knee flexion and extension (Table 12-1).

Table 12-1

Phasing of Sagittal Plane Gait Deviations at the Knee

	IC	*LR*	*MSt*	*TSt*	*PSw*	*ISw*	*MSw*	*TSw*
Limited knee flexion		X			X	X		
Knee hyperextension	X	X	X	X	X			
Extensor thrust		X	X					
Excess knee flexion	X	X	X	X				X
Excess contralateral knee flexion					X	X	X	X
Wobble		X	X	X				

Key: X = phases affected by the designated pathology

LIMITED KNEE FLEXION

Definition: Less than normal knee flexion for a particular phase

Phases: Loading response, pre-swing, initial swing

Functional Significance: During loading response, limited knee flexion reduces the normal shock-absorbing mechanism and the demand placed on the quadriceps. During pre-swing and initial swing, limited knee flexion interferes with foot clearance and may contribute to a foot drag.

Underlying Causes:

* Quadriceps weakness (loading response)
* Quadriceps or plantar flexor spasticity (loading response, pre-swing, initial swing)
* Knee or patello-femoral knee joint pain
* Knee extension contracture
* Limited thigh advancement due to over-activity of the hamstrings or weakness of the hip flexors
* Impaired proprioception
* Primitive muscle synergies interfering with the ability to rapidly combine knee flexion with extension at adjacent joints

In 3 of the 4 gait phases in which flexion is a normal event, the loss of this motion is functionally significant. These phases are loading response, pre-swing, and initial swing. In mid swing, limited knee flexion has little functional impact. The pathologies contributing to a lack of flexion in stance and swing differ markedly. Each situation also leads to very different substitutions.

During loading response, limited knee flexion is evidence of intrinsic pathology (eg, a knee extension contracture). But the complete absence of knee flexion generally is a voluntary substitutive action (eg, to reduce the demand placed on weakened quadriceps or to avoid pain associated with rapid joint movement).

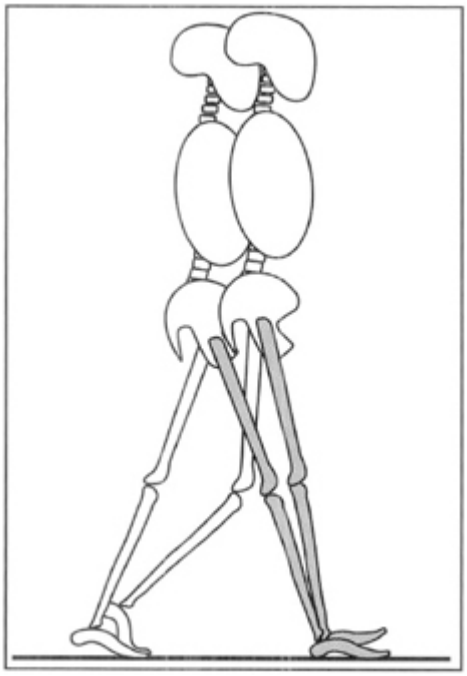

Figure 12-1. Limited knee flexion in loading response disrupts the normal shock-absorption mechanism.

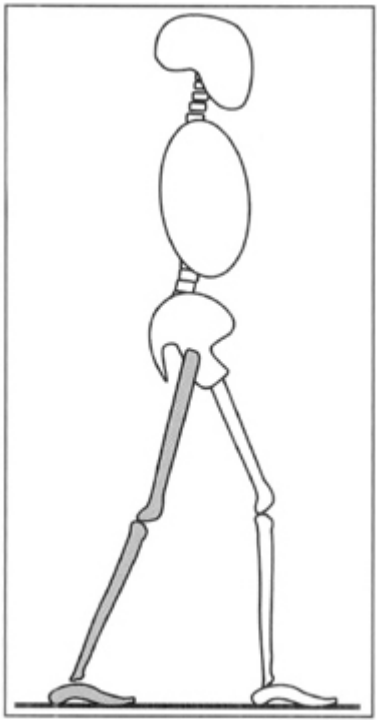

Figure 12-2. Limited knee flexion in pre-swing fails to prepare the limb for swing.

Failure to flex the knee more than 5° or 10° reduces the limb's shock-absorbing capability and can be a source of pain. The relatively rigid limb transfers the impact of body weight falling directly from the femur to the tibia without muscular cushioning (Figure 12-1). The significance of this exchange varies with gait velocity. If the patient is a slow walker, the peak load will not exceed body weight because no significant acceleration is added. Patients capable of walking rapidly may experience damaging micro-trauma to the articular cartilage and underlying bone.[10]

Full knee extension has the advantage of being the most stable weight-bearing position as the body vector is anterior to the joint axis at IC and can remain so if the heel rocker is avoided by premature ankle PF. Consequently, absent knee flexion in the loading period is a desirable gait deviation when the quadriceps muscle is too weak to repeatedly restrain a flexing knee.

During pre-swing, failure to adequately flex the knee makes toe-off more difficult. The transition between stance and swing is lost (Figure 12-2). The trailing posture of the limb tilts the foot downward.

Without timely knee flexion, the functional length between the hip and toe (the part of the foot closest to the floor) is longer than the hip-to-heel length of the weight-accepting lead limb. Substitutions for the limited knee flexion seek to free the toe by creating a relatively longer contralateral limb. These include vaulting (excessive ankle PF) of the lead limb, as well as pelvic hiking (elevation) and abduction (circumduction) by the trailing limb. All of these actions increase the energy cost of walking.

During initial swing, the lack of adequate knee flexion causes toe drag with a corresponding inability to advance the limb (Figure 12-3). This difficulty is a continuation of the situation in pre-swing, which is made worse by hip flexion. In an attempt to advance the trailing limb, it becomes more vertical, and the toe drag is increased. The substitutions used in pre-swing allow the trailing limb to swing forward. After the toe has moved forward of the other limb, the need for knee flexion is reduced.

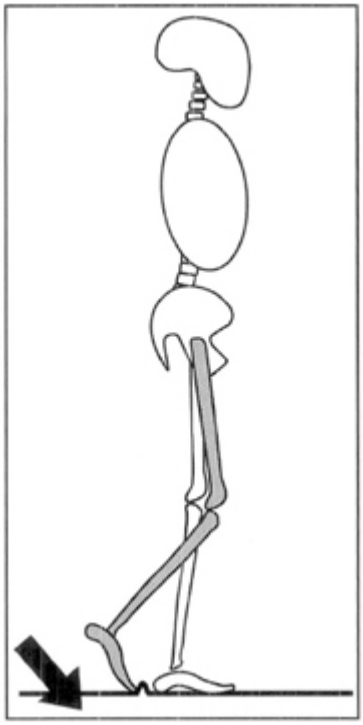

Figure 12-3. Limited knee flexion during initial swing effectively lengthens the limb, resulting in toe drag.

The causes of limited knee flexion differ with the phases in which the deviation occurs. The challenge in loading response is the intensity of quadriceps action to support a flexed knee. Passive flexibility is the need in pre-swing and initial swing. The potential causes of limited knee flexion are quadriceps weakness, quadriceps spasticity, hip flexor weakness, pain, contracture, and joint fusion.

Muscular Causes of Limited Knee Flexion

Quadriceps Weakness

The primary function of the quadriceps is to support a flexed knee. When there is insufficient quadriceps strength to meet this demand, a variety of substitutions are used to preserve weight-bearing stability.

During loading response, persons with intact sensory systems (polio, femoral nerve injury, secondary disuse weakness) deliberately avoid knee flexion to preserve weight-bearing stability when the quadriceps is incapable of controlling a flexing knee (MMT Grade 0 to 3). Some patients with only moderate quadriceps weakness (MMT Grade 3+ to 4) also use this same substitution to protect the muscle from repetitive strain in order to walk at a near normal speed.

Loading response knee flexion is prevented by 2 voluntary actions (Figure 12-4). Hip extension by the gluteus maximus and adductor magnus retracts the thigh. Premature ankle PF, initiated by the soleus, blocks tibial advancement so IC by the heel does not stimulate the normal rocker action.

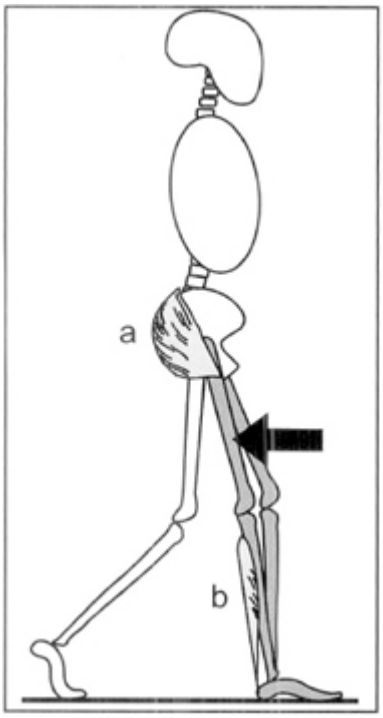

Figure 12-4. Avoidance of loading response knee flexion by retraction of the femur (via increased gluteus maximus and adductor magnus activity) or blocking of tibial progression (by premature soleus action).

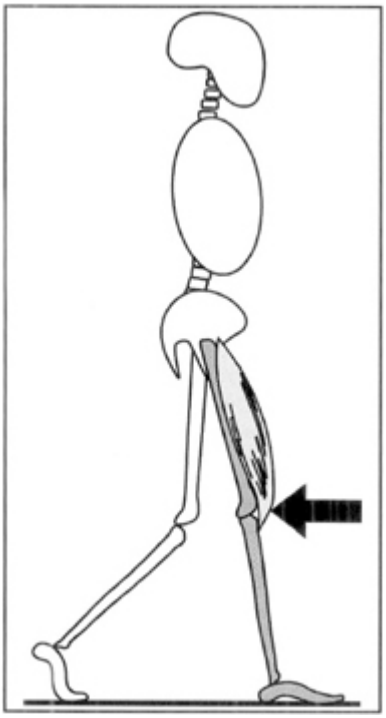

Figure 12-5. Quadriceps overactivity (vastii) can inhibit loading response knee flexion.

During pre-swing, failure to flex the knee relates to combined muscle weakness at the ankle and knee. Normally, the calf muscles act to restrain forward collapse of the tibia. When the gastrocnemius and soleus are weak, the tibia will collapse forward and the knee will assume a flexed posture. If the quadriceps is weak, however, the added extensor demand will not be tolerated. Instead, the patient must keep the knee extended until the limb is fully unloaded by the transfer of body weight to the other limb (end of pre-swing). The knee is maintained in full extension (or hyperextension). This obstructs the passive freedom needed for knee flexion. Occasionally, a momentary toe drag results. The possible delay in toe clearance becomes greater when hyperextension (knee recurvatum) is the source of knee stability.

Quadriceps Spasticity

Excessive muscular action is stimulated by a stretch reaction to rapid passive knee flexion. Loading response, pre-swing, and initial swing are the sensitive phases of gait.

Loading response knee flexion through the heel rocker action induces a rapid stretch of the quadriceps. An excessive response by the vastii inhibits the full flexion range. Premature knee extension results (Figure 12-5).

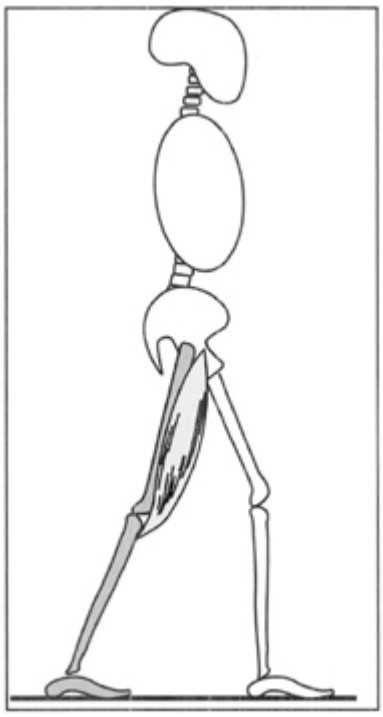

Figure 12-6. Loss of pre-swing knee flexion due to prolonged quadriceps activity.

Throughout the rest of stance, continuation of a primitive extensor pattern accentuates the knee's position. A false impression of hyperextension may be created by a forward lean of the trunk proximally, combined with ankle PF distally while the knee joint actually lacks the backward range.

Sustained quadriceps action into pre-swing obstructs the passive freedom needed for knee flexion. This occurs in persons dependent on a primitive extensor synergy. The primitive extension pattern used in mid and terminal stance to stabilize the knee is too rigid to allow rapid pre-swing knee flexion. Consequently, the knee is not prepared for swing (Figure 12-6).

Initial swing knee flexion can be inhibited by several patterns of quadriceps spasticity. All or part of this muscle group may be involved.[12] The reactive muscle in approximately one-fourth of patients is the RF. It obstructs initial swing knee flexion when the muscle's intensity is excessive or the action is prolonged (Figure 12-7). Persistent vastus intermedius (VI) activity into swing is a second pattern of obstructive quadriceps action (Figure 12-8). The third pattern of quadriceps obstruction to initial swing knee flexion is

prolonged action of all the vastii into a major portion of swing (Figure 12-9). Differentiating over-activity of the RF from that of VI and vastus lateralis is best accomplished using fine wire EMG.[7,8] Surface EMG recording of the RF may inadvertently record cross talk from the underlying muscles.

The over active rectus can be relieved surgically. If the quadriceps muscle is strong, surgical release of the VI also is effective. But total vastii over-activity presents an uncorrectable situation as the vastus lateralis and medialis (longus or VMO) are not expendable (see Figure 12-9). The specific patterns of muscle action, however, cannot be differentiated by motion analysis or palpation. Dynamic EMG is required.

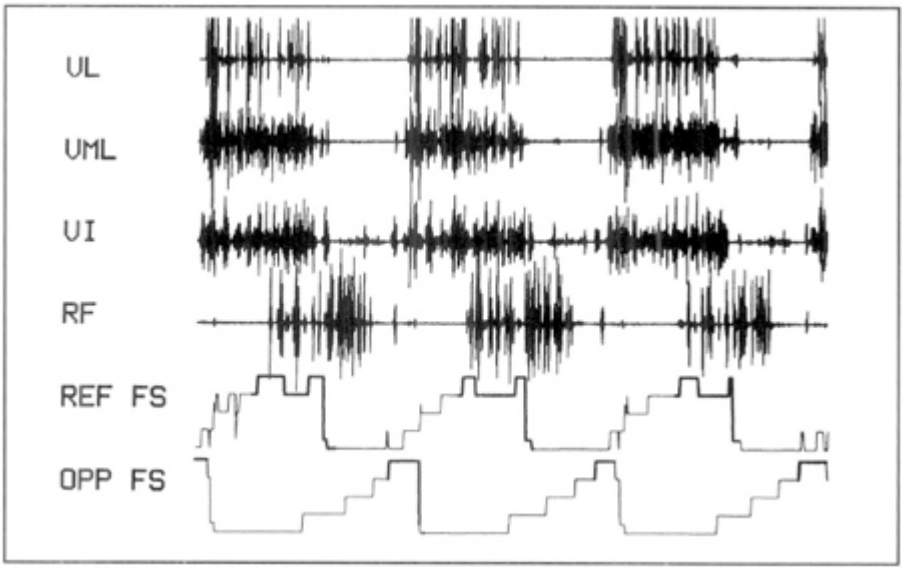

Figure 12-7. Prolonged and intense RF activity inhibits swing phase knee flexion. Prolonged vastii activity terminates during pre-swing. Reference limb footswitches (REF FS) show inconsistent pattern of progression. Overlap of normal opposite footswitch (OPP FS) loading response with end of reference limb footswitch identifies the pre-swing phase.

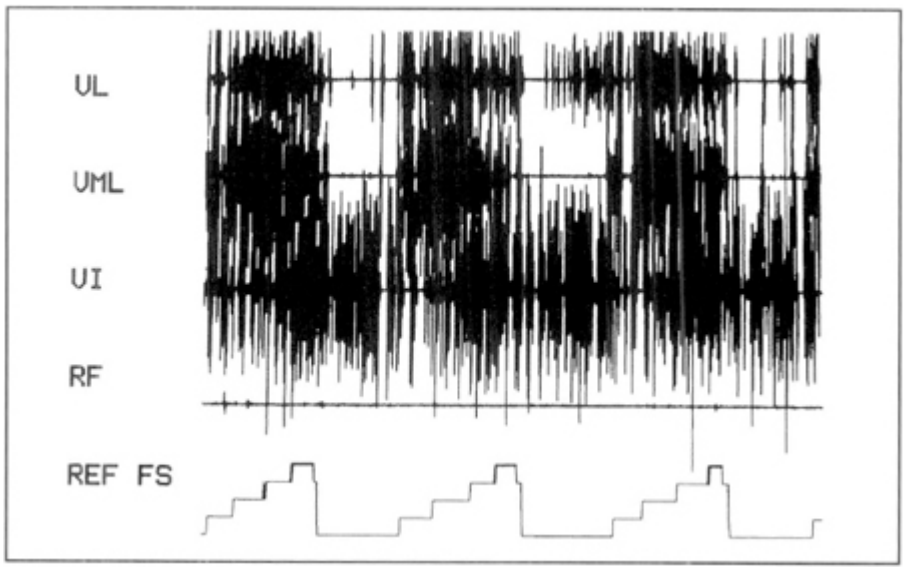

Figure 12-8. Continuous vastus intermedius (VI) activity inhibits knee flexion during swing together with prolonged activity of the vastus lateralis (VL) and vastus medialis longus (VML) into initial swing. REF FS = reference limb footswitches.

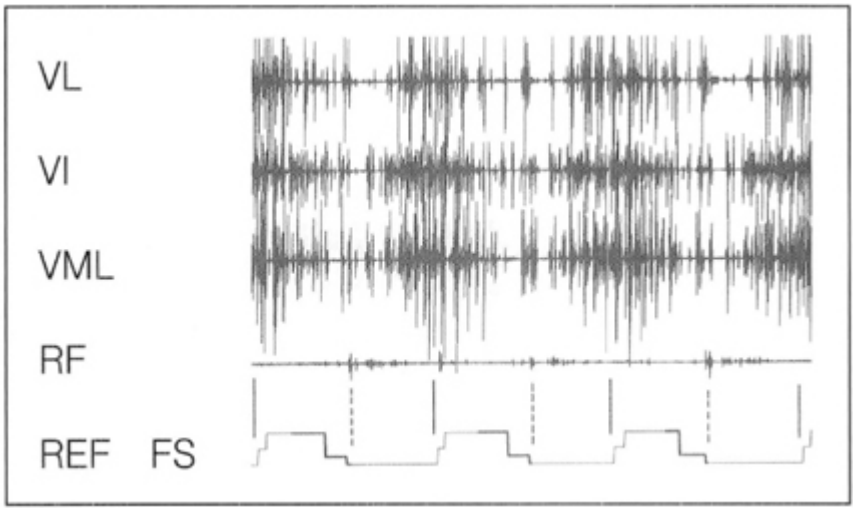

Figure 12-9. Continu-ous activity of the vastus lateralis (VL), vastus intermedius (VI), and vastus medialis longus (VML) inhibits knee flexion. Rectus femoris (RF) activity is not significant. REF FS = reference limb footswitches.

Hip Flexor Weakness

Knee flexion is lost in initial swing when inadequate hip flexion deprives the patient of the momentum normally used to flex the knee. This problem is accentuated by inadequate pre-swing function. The initiation of knee flexion is delayed and the arc of motion is incomplete (Figure 12-10). Slow walkers also need increased hip flexor effort to bend the knee. They rely on femoral advancement and tibial inertia for knee flexion. With weak hip flexors, the thigh stays vertical and the knee relatively extended.

Inappropriate Hamstring Activity

Prolonged or premature activity of the hamstrings during pre-swing has been associated with reduced knee flexion during early SLA.[5] The hamstrings restrain forward advancement of the thigh via their action at the hip. Passive knee flexion is reduced.

Impact of Pain on Limited Knee Flexion

Rapid motion of a knee with intrinsic joint pathology increases tissue tension and, thus, pain. This leads to limited knee flexion in 3 phases: loading response, pre-swing, and initial swing.

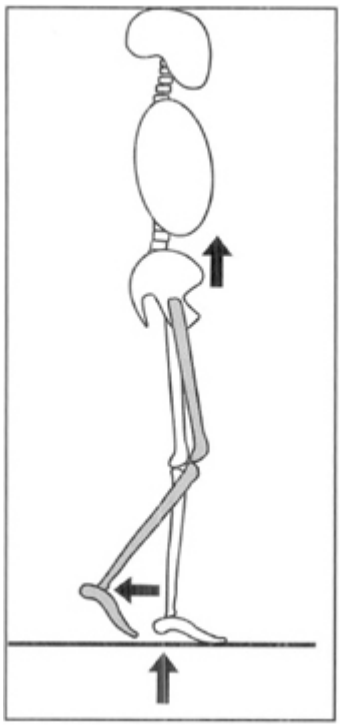

Figure 12-10. Hip flexor weakness causes limited knee flexion in initial swing. Limb clearance requires increased ipsilateral ankle DF and contralateral vaulting or a pelvic hike.

Loading response knee flexion is avoided by the same mechanisms used for quadriceps weakness. The purpose is to escape the shear force that accompanies joint motion. A second objective is to reduce the compressive force from a contracting quadriceps. Articular surface damage from arthritis, gross instability, and scarring following multi-ligamentous injury are the common causes. Patellar lesion are particularly limiting.

During pre-swing, intentional limitation of knee flexion is a mechanism used to escape the associated shear forces. This action is less complete as weight bearing is rapidly reduced. Continued failure to move the knee rapidly during initial swing results in an inadequate arc of flexion during walking, even though there is sufficient passive range by clinical examination. The effect is a stiff-legged gait pattern that may carry into mid swing.

Impact of Extension Contractures and Joint Fusion on Limited Knee Flexion

The knee flexion needed for swing may be lost through 2 mechanisms. There may be insufficient passive range from capsular scarring or quadriceps contracture (ie, less than 60°). More commonly, the passive range obtained by clinical examination is sufficient, but the scarred tissues cannot yield rapidly enough to meet the motion demands of pre-swing and initial swing. The functional requirement of 60° knee flexion within 0.2 seconds (pre-swing and initial swing combined) commonly exceeds the plasticity of scar tissue. Consequently, a knee that bends adequately under slow passive teasing still may display severe limitations during walking.

During loading response, knees fused in 15° to 20° of flexion will lack the normal arc of motion required for shock absorption despite being in the appropriate end position. While the fused position may provide the requisite stability for SLS, the knee flexion necessary for foot clearance during SLA will be blocked.

KNEE HYPEREXTENSION

Definition: The knee is positioned posterior to the anatomic neutral. This is also referred to as recurvatum.

Phases: IC, loading response, mid stance, terminal stance, pre-swing

Functional Significance: Knee hyperextension reduces the demand on a weak quadriceps. During WA, it also decreases the normal shock-absorbing mechanism and interferes with forward progression. During mid stance, terminal stance, and pre-swing, the deviation limits forward progression and preparation of the limb for SLA.

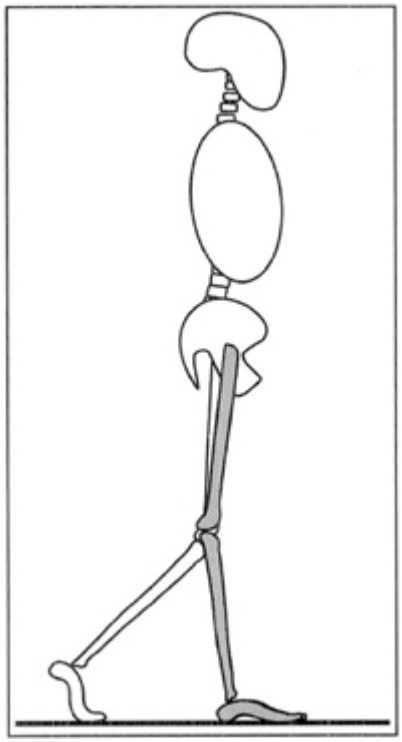

Figure 12-11. Knee hyperextension. Retraction of the tibia or the femur pulls the knee into recurvatum (backward curvature).

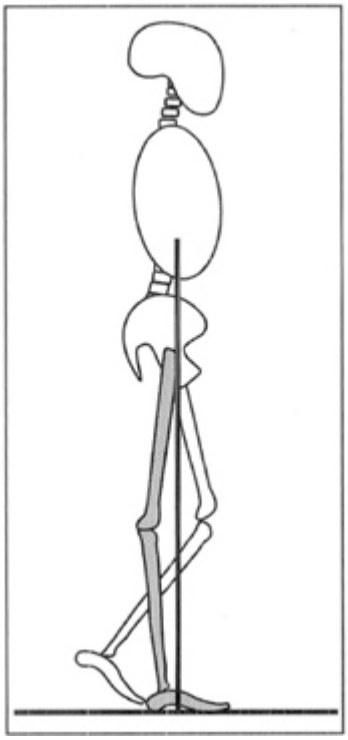

Figure 12-12. Knee hyperextension substitution for a weak quadriceps. Vector is anterior to the knee, providing extensor

moment.

Underlying Causes:

* Quadriceps weakness (loading response)
* Combined quadriceps and plantar flexor weakness (SLS)
* Quadriceps and plantar flexor spasticity
* Severe PF contracture

Hyperextension occurs when the knee has the mobility to angulate backwards (recurvatum). The onset of hyperextension can occur in any of the weight-bearing phases (Figure 12-11). It may be slow and passive or actively abrupt. Hyperextension is a position of relative knee stability as the body weight vector now falls anterior to the knee joint throughout the majority of stance (Figure 12-12).

Hyperextension often develops over time in response to chronic strain placed on the posterior capsule and ligaments that support the knee joint. The deforming mechanism is a combination of 2 events: tibial retraction with continued forward momentum of the proximal body. Excessive restraint of the rate of tibial advancement can arise due to either a PF contracture or over-activity of the calf muscles. As the proximal body advances more rapidly than the restrained tibia, a knee extensor force develops. The deformity progresses rapidly when spasticity of both the plantar flexors and quadriceps is present.

Alternatively, a forward trunk lean displaces the body weight vector anterior to the knee joint, eventually contributing to elongation of the posterior-supporting structures. This can be a voluntary movement for individuals who lack quadriceps stability.

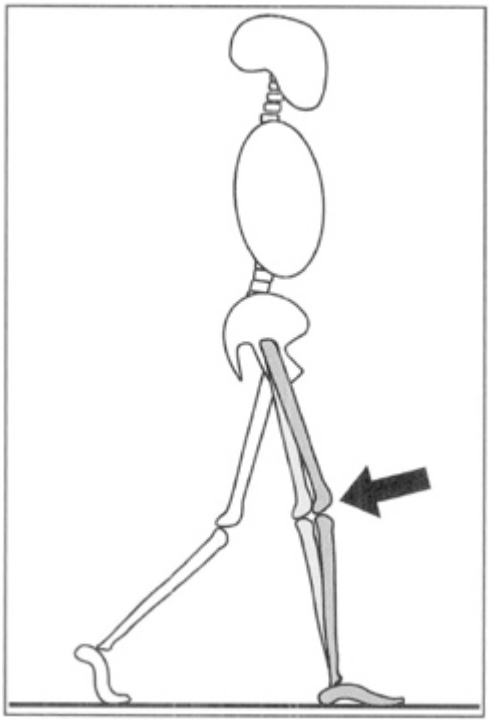

Figure 12-13. Extension thrust. Rapid posterior movement of the knee toward extension delays forward progression during loading response.

EXTENSOR THRUST

Definition: A forceful motion of the knee toward extension

Phases: Loading response, mid stance

Functional Significance: An extension thrust at the knee reduces the demand on a weak quadriceps. During WA, it also decreases the normal shock-absorbing mechanism and interferes with forward progression.

Underlying Causes:

* Quadriceps weakness (loading response)
* Quadriceps or plantar flexor spasticity (loading response, mid stance)
* PF contracture with a forefoot IC
* Proprioceptive deficits of the knee

An extension thrust is a dynamic, rapid action arising from an extensor force that moves the knee posteriorly (Figure 12-13). When the quadriceps are weak, a knee extension thrust ensures that the knee moves into extension during WA versus assuming the more demanding position of flexion. Often a forward trunk lean serves as the mechanism for realigning the body vector anterior to the knee joint to promote knee extension.

An extensor thrust often is the first reaction to limb loading in the presence of a PF contracture, PF spasticity, or quadriceps spasticity. When a severe PF contracture is present, the initial mode of ground contact is often with the forefoot. As the limb rapidly loads, the excess passive plantar flexor stiffness drives the tibia posteriorly, contributing to an extensor thrust at the knee. A similar pattern emerges in the presence of plantar flexor spasticity. As the limb loads, the stretch applied to the plantar flexors results in a rapid increase in calf muscle activity, driving the tibia posteriorly. When quadriceps spasticity is present, the initial stimulus is a quick stretch arising from the onset of knee flexion during loading response. The stretch accentuates activation of the vastii, and a knee extension thrust versus the normal knee flexion results. Timing varies with the severity of the contributing pathology.

When proprioception is impaired at the knee, a knee extension thrust may be used to ensure that the knee is in an extended and stable posture during stance. The forcefulness of the extension thrust may provide an added benefit of sensory feedback related to motion and position of the knee.

Both hyperextension and an extension thrust can lead to long-term penalties. Disabling fatigue of the stabilizing muscles may develop. Also, chronic strain can cause degeneration and pain of the ligaments supporting the hyperextended knee.

The next 2 deviations, excess knee flexion and excess contralateral flexion, share many causes in common. Any impairment that can cause excess knee flexion on the reference limb could, if present in the opposite limb, contribute to excess contralateral knee flexion.

Excess Knee Flexion

Definition: More-than-normal knee flexion for a particular phase

Phases: IC, loading response, mid stance, terminal stance, terminal swing

Functional Significance: During WA and SLS, excess knee flexion increases the demand on the quadriceps. In terminal swing, excess knee flexion limits step length and forward progression.

Underlying Causes:

* Plantar flexor weakness (SLS)
* Knee flexion contracture/joint effusion
* Compensation for a hip flexion contracture (SLS)
* Knee flexor over-activity or spasticity
* Hamstring contracture
* Primitive synergy movement patterns
* Knee pain

Excess flexion occurs under 2 situations. One is an exaggeration of a normal flexion arc. This is seen in loading response and mid swing. The other circumstance is a loss of normal extension. This occurs in mid stance, terminal stance, and terminal swing. Each situation represents a very different pathology.

Excessive knee flexion during loading response relates to a knee posture greater than 15° (Figure 12-14). Most of this flexion arc develops in loading response, but a few more degrees of knee flexion actually follows the added demands of SLS at the onset of mid stance. This timing presents slight confusion in the phasic interpretation of knee dysfunction. To differentiate the major purpose of mid stance (progressive knee extension), the term *excessive flexion* has been identified with the loading response phase.

During mid stance and terminal stance, the inability to fully extend the knee is a common gait error that compromises weight-bearing stability. During mid stance, the knee fails to move toward extension (Figure 12-15). The knee in terminal stance does not extend within

10° of neutral (Figure 12-16). Generally, these gait errors represent a continuation of the excessive flexion displayed in loading response.

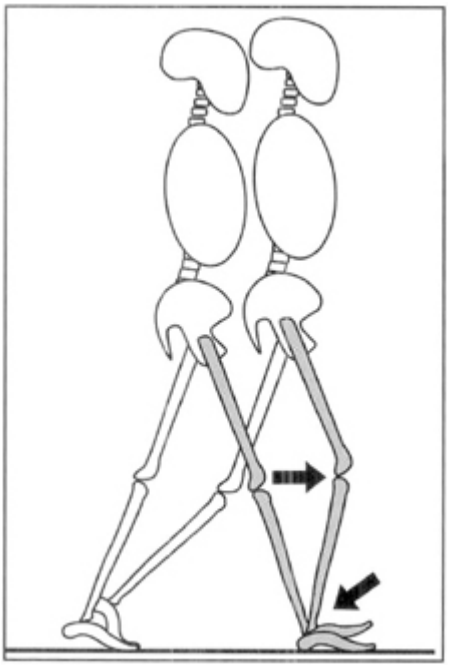

Figure 12-14. Excessive knee flexion in loading response. Note accompanying excessive ankle DF.

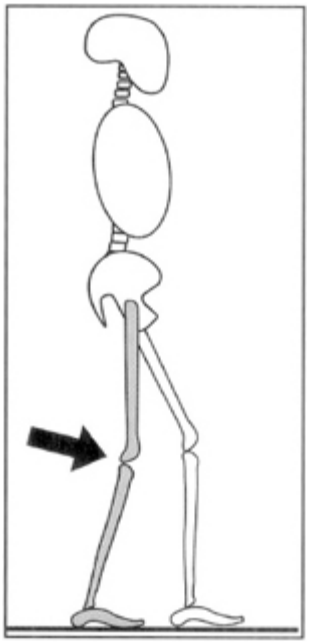

Figure 12-15. Excess knee flexion in mid stance accompanied by excessive ankle DF.

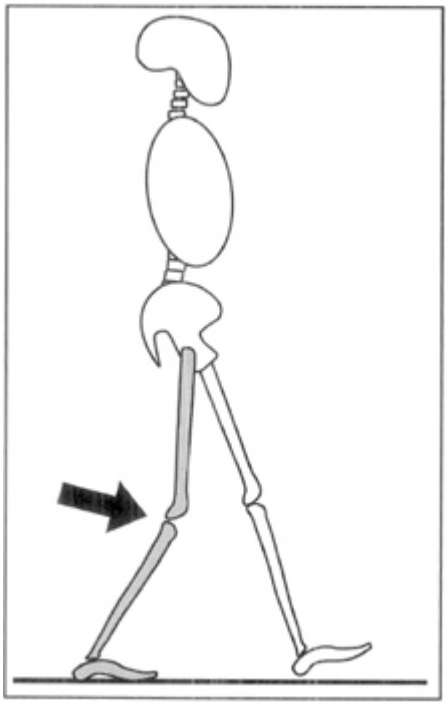

Figure 12-16. Excess knee flexion in terminal stance accompanied by excessive ankle DF and lack of a trailing limb posture.

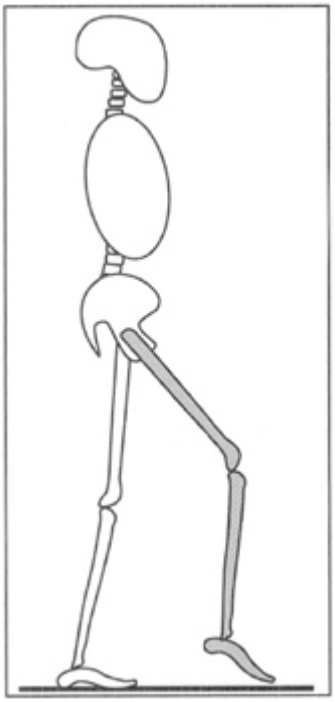

Figure 12-17. Excess knee flexion in mid swing to compensate for excessive ankle PF. Without compensation, the toe drags. Increased hip flexion to lift a plantar flexed foot for clearance also increases knee flexion.

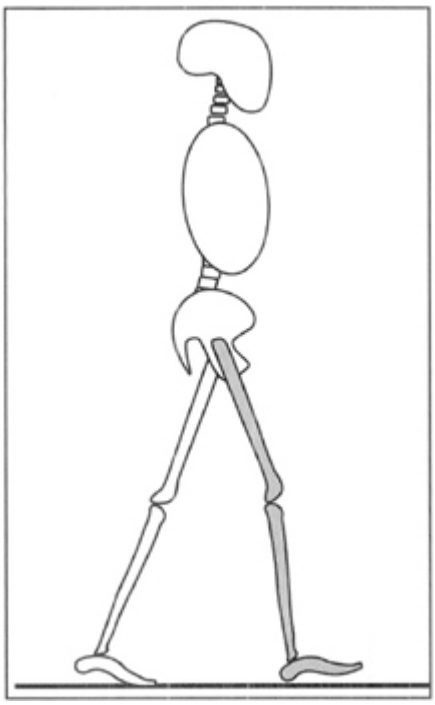

Figure 12-18. Inadequate knee extension in terminal swing shortens step length.

Excessive knee flexion during mid swing generally represents the secondary effect of increased hip flexion and gravity drawing the tibia to a vertical posture (Figure 12-17). It is not a clinical concern.

Inability to fully extend the knee by the end of terminal swing results in a shortened step length (Figure 12-18). This may be intentional to decrease the muscle demands associated with WA. Alternatively, patients with primitive control may lack terminal knee extension due to the inability to mix flexion and extension in the same limb.

There are numerous causes of excessive knee flexion. Some are referred from dysfunction at adjacent joints while others arise from the knee structures themselves.

Impact of Calf Weakness on Excess Knee Flexion

Inability of the soleus to effectively control the tibia is a major cause of inadequate knee extension in mid and terminal stance even

though it is often overlooked. Weakness may arise from paralytic conditions as well as situations that limit forefoot loading such as arthritis. Borderline weakness of the plantar flexors is difficult to detect with traditional manual muscle testing, hence excessive knee flexion may provide the first insight into the presence of a weak calf to the clinician. The lack of sufficient plantar flexor muscle strength allows the tibia to fall forward as the body vector advances. As a result, the tibia advances faster than the femur, causing continued knee flexion (Figure 12-19). The quadriceps, despite having adequate strength to support the flexed knee, cannot re-establish knee extension because the base from which it pulls (the tibia) is unstable. As the quadriceps draws the femur forward for knee extension, it also advances the body mass resting on the hip. This increases the demand torque at the ankle, which the weak soleus cannot resist. Hence, the whole limb rather than just the femur advances and knee extension remains inadequate.

Figure 12-19. Excessive knee flexion can follow excessive ankle DF when the soleus is too weak to stabilize the tibia.

Impact of Contractures on Excess Knee Flexion

As the resting position for a swollen knee is about 30°,[4] this is not an uncommon posture for a contracture (see Figure 10-6). Numerous pathologies may lead to this deformity, of which major trauma, arthritis, and knee surgery are the most likely causes. Knee flexion contractures introduce a constant limitation to extension. All phases of the GC except initial swing will be abnormal with a 30° flexion contracture. Partial recovery of the passive range to a 15° contracture will allow loading response and pre-swing to have normal posturing. Knee extension in terminal swing, IC, mid stance, and terminal stance will still be inadequate.

While the difference between 15° and 30° may not be obvious during the loading response, inadequate extension in the SLS phases will be visually apparent and can notably increase the muscular demand on the quadriceps. During terminal swing, the penalty for loss of extension is shortening of step length.

In contrast to the increased quadriceps demand resulting from a knee flexion contracture, knee extensor activity diminishes when the excess knee flexion arises from an intra-articular knee joint effusion.[11] The reduced force-producing capabilities of the quadriceps when the knee joint capsule is distended from effusion[3] may necessitate proximal compensations (eg, a forward trunk lean) to reduce the knee extensor demand.

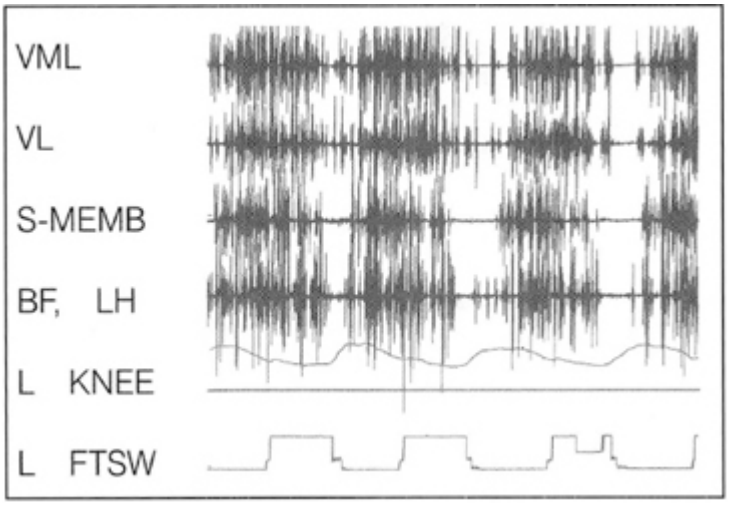

Figure 12-20. Simultaneous activation and cessation of the quadriceps (vastus medialis longus [VML], vastus lateralis [VL]) and hamstrings (semimembranosus [S-MEMB], biceps femoris long head [BF, LH]) are characteristic of the primitive extensor synergy at the knee. The quadriceps lacks the selective control to pull harder against the flexor force of the hamstrings and excessive knee flexion (L KNEE) persists throughout stance. The left footswitches (L FTSW) indicate a curtailed heel rocker.

A hip flexion contracture can also contribute to excess knee flexion during SLS if the severity of the contracture results in the distal end of the femur being tilted forward. Excess knee flexion will be required to accommodate the hip flexion contracture if the foot is to remain flat on the ground and forward progression is to occur.

Impact of Hamstring Over-Activity on Excess Knee Flexion

Patients with upper motor neuron lesions (cerebral palsy, stroke, spinal cord injury, traumatic brain damage) commonly display over-activity of the hamstrings. This generally is attributed to spasticity, but inappropriate hamstring activity also is a common part of the primitive mass extensor muscle synergy (Figure 12-20).[2] Excessive hamstring activity may be premature, prolonged, or even continuous. Hamstring over-activity generally begins in early mid swing and

continues through mid stance or longer (Figure 12-21). The patient's knee may remain excessively flexed throughout terminal swing and stance.

Over-activity of the spastic hamstrings may also result from the forward trunk lean required to advance the body when ankle DF is limited. The patient's forward posture increases the need for hip extensor support. This is an additional stimulus for prolonged hamstring activity in stance. Unfortunately, it also contributes to knee flexion (Figure 12-22). The forward trunk lean helps protect the quadriceps from over-demand.

Hamstring Contractures

During gait, the maximum ROM demand for the hamstrings occurs during terminal swing. At this point, the pelvis is tilted anteriorly 10°, the thigh is flexed 20°, and the knee has achieved near full extension. Severe hamstring tightness (eg, a straight leg raise of less than 40°) will limit normal terminal swing knee extension. The knee is more susceptible to hamstring tightness than the hip during terminal swing because the knee is attempting to attain a position of greater extension during this period while the hip is retreating from its peak position of 25° flexion prior to making contact with the ground. A posterior pelvic tilt may be used to increase the available knee extension motion when limb advancement is severely curtailed.

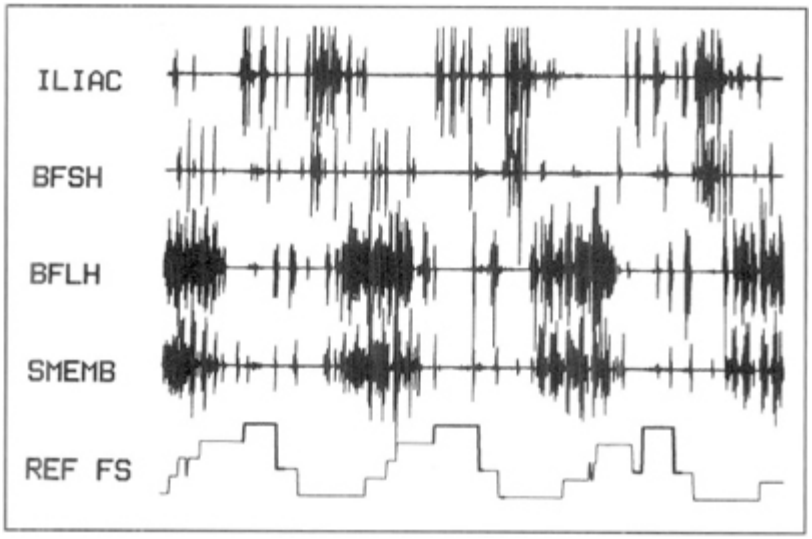

Figure 12-21. Hamstring spasticity causing inadequate knee extension in stance. Biceps femoris long head (BFLH) and semimembranosus (SMEMB) demonstrate premature activity during mid swing and prolonged activity until mid stance. Iliacus (ILIAC) demonstrates a dominant pattern during initial swing with semi-clonic activity during terminal stance, suggesting a spastic response to quick stretch. Biceps femoris short head (BFSH) shows an inconsistent and insignificant pattern of activity. The reference limb footswitch (REF FS) shows an inconsistent normal sequence.

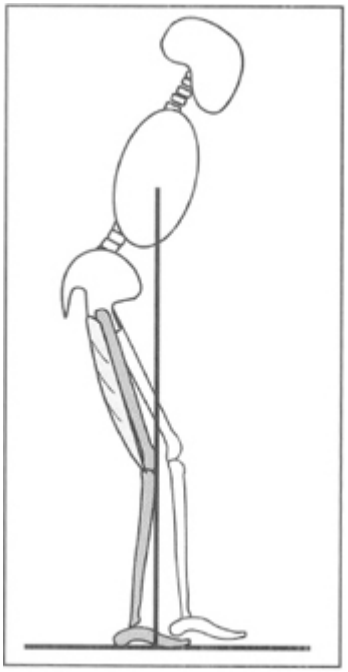

Figure 12-22. Prolonged hamstring activity in mid stance. Use of hamstrings to support flexed trunk also causes excessive knee flexion.

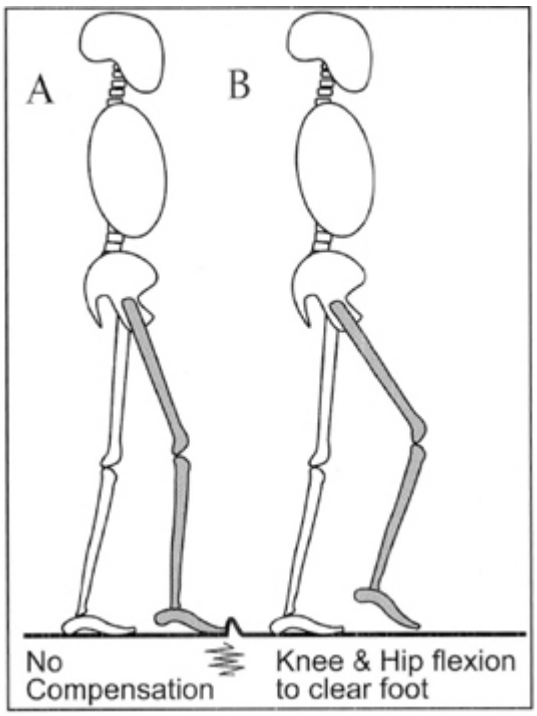

Figure 12-23. Mid swing excessive knee and hip flexion to compensate for a drop foot. (A) Toe drag without compensation. (B) Floor clearance by excessive hip flexion lifting limb.

Impact of Weakness of the Single Joint Hip Extensors on Excess Knee Flexion

Selective substitution of the hamstrings for weakness of the primary hip extensors (gluteus maximus and adductor magnus) in a person with normal motor control leads to a mild loss of knee extension in stance because of the hamstrings' insertion on the tibia. The knee remains flexed approximately 15° from loading response through terminal stance. A forward lean of the trunk is used to keep the body vector anterior to the knee if quadriceps weakness is also present. Knee stability is precarious as the secondary flexion of the knee by the hamstrings also increases the need for a forward lean.

Impact of Excess Ankle Plantar Flexion on Excess Knee Flexion

The distance between the hip joint and toe is increased by ankle PF. Whenever the swing limb must pass the stance limb, this relative lengthening must be accommodated by knee flexion if there is no anatomical shortening. Mid swing advancement of the limb will be obstructed by an ankle PF contracture or dorsiflexor paralysis. To ensure foot clearance of the floor, the limb is lifted (Figure 12-23). Hip flexion is the basic action while gravity, which holds the tibia vertical, flexes the knee. The visibility of the knee makes excessive knee flexion appear to be the obvious substitution, but this is only accomplished through the added hip flexion.

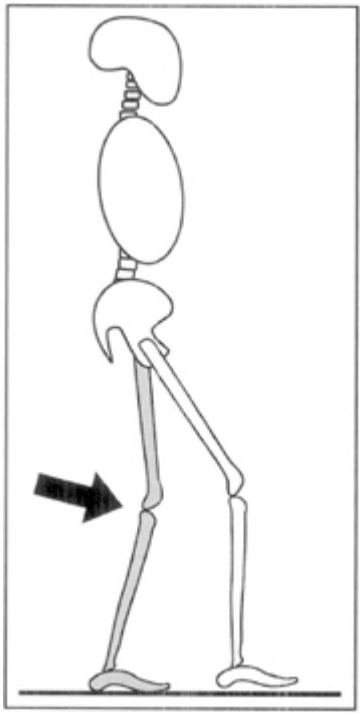

Figure 12-24. Excess contralateral knee flexion from any of the previously discussed causes will reduce pelvis height (gray limb). This leads to relative leg length discrepancy, which can increase the demand on the reference limb (white) and the need to compensate for foot clearance.

Excess Contralateral Knee Flexion

Definition: Greater-than-normal knee flexion of the contralateral limb

Phases: Contralateral limb stance phases (corresponding with reference limb swing)

Functional Significance: Greater-than-normal contralateral knee flexion lowers the body toward the ground, effectively lengthening the reference limb

Underlying Causes:

* Any factor that could cause excess stance phase knee flexion in the contralateral limb
* Intentional knee flexion to lower the reference limb to the ground

The contralateral deviation is worthy of note as it lowers the body toward the ground and may necessitate compensatory maneuvers on the reference limb to clear the limb (eg, increased hip flexion or circumduction). Excess contralateral flexion occurs when the contralateral limb is in greater flexion than it should be during reference limb stance (Figure 12-24). The causes are similar to those outlined for excess knee flexion of the reference limb. On occasion, excess contralateral flexion is a strategy used to lower the body toward the ground when the reference limb is anatomically shorter (eg, with a leg length discrepancy). This posture increases demand on the vastii of the contralateral limb and increases energy cost.

The final sagittal plane deviation, the “wobble,” involves alternating arcs of flexion and extension. Although not a common deviation, its detection points to impairments in proprioception or to the presence of clonus.

Table 12-2

Phasing of Coronal Plane Gait Deviations at the Knee

	IC	*LR*	*MSt*	*TSt*	*PSw*	*ISw*	*MSw*	*TSw*
Valgus	X	X	X	X				
Varus	X	X	X	X				

Key: X = phases affected by the designated pathology

WOBBLE

Definition: Rapid alternating flexion and extension of the knee occurring during a single phase

Phases: Loading response, mid stance, terminal stance

Functional Significance: A wobble decreases forward progression, increases energy cost, and decreases limb stability

Underlying Causes:

* Proprioceptive deficit
* Hypertonicity of the plantar flexors or quadriceps

The term *wobble* was adopted to identify small, alternating arcs of flexion and extension occurring during stance. It may represent a search for stability in a joint with impaired proprioception. Spastic clonus of either the plantar flexors or quadriceps also leads to a wobble.

CORONAL PLANE GAIT DEVIATIONS

The terms *abduction* (valgus) and *adduction* (varus) refer to lateral and medial angulations of the tibia at the knee in the coronal plane. A false impression of knee joint distortion can be gained when knee flexion and limb rotation are combined. This faulty interpretation is

very likely with observational analysis unless the presence of the confounding motions is recognized. Misinterpretation of knee angulation also can occur in single camera filming (or video). As both rotation and flexion occur in swing, the presence of coronal plane malalignments should only be diagnosed during the stance period of the GC (Table 12-2).

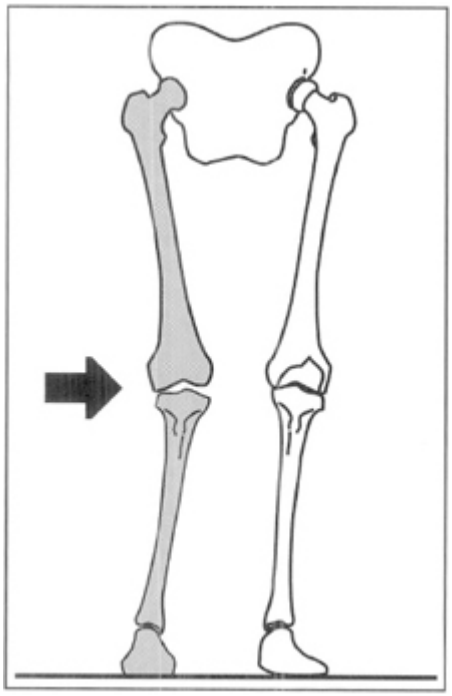

Figure 12-25. Knee valgus (excessive abduction). The knee is displaced medially from the line between the hip and ankle because the distal end of the tibia is tilted laterally.

EXCESSIVE ABDUCTION (VALGUS)

Definition: Excess lateral deviation of the distal tibia from the center of the knee

Phases: Stance phases

Functional Significance: When severe, it may limit stability, necessitate proximal or distal compensations, and contribute to pain

Underlying Causes:

* Joint or ligamentous instability
* Bony deformity
* Pain
* Ipsilateral abductor weakness
* Ipsilateral trunk lean

Excessive abduction (valgus) refers to excessive lateral deviation of the distal tibia from the center of the knee (Figure 12-25). The normal abduction of approximately 10° represents an angulation of the femur while the tibia is vertical. Pathological abduction at the knee causes the distal tibia to tilt laterally with corresponding lateral displacement of the foot. This clinically is called a *valgus deformity* or "*knock-knee*." During quiet standing, the distance between the feet will be greater that at the knees. *Pseudo-valgus* is a term used to describe combined internal hip rotation and knee flexion that can give a false impression of abduction during either stance or swing. It can arise from combined weakness of the gluteus medius and gluteus maximus.[6] An ill-fitted prosthesis for a trans-tibial amputation can contribute to valgus and pain.

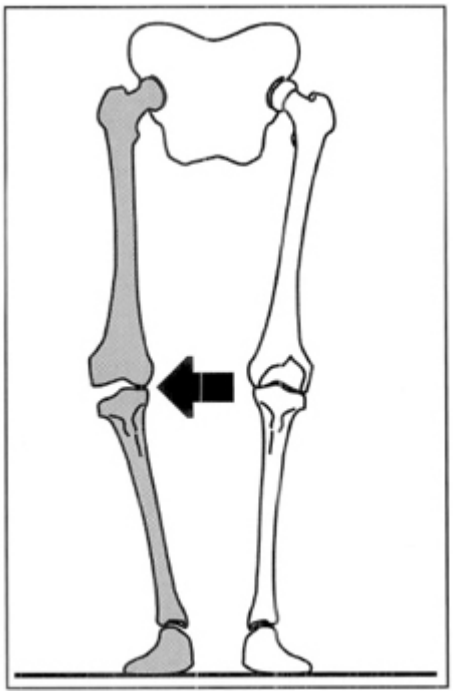

Figure 12-26. Knee varus (excessive adduction). The knee is displaced laterally from the line between the hip and ankle because

the distal end of the tibia is tilted medially.

Excessive Adduction (Varus)

Definition: Excess medial deviation of the distal tibia from the center of the knee

Phases: Stance phases

Functional Significance: When severe, it may limit stability, necessitate proximal or distal compensations, and contribute to pain

Underlying Causes:

* Joint or ligamentous instability
* Bony deformity
* Degenerative joint changes
* Pain

Excessive adduction (varus) of the knee is displayed by a medial tilt of the tibia and medial displacement of the foot relative to the knee (Figure 12-26). Femoral alignment also is altered as the hip abducts to accommodate the foot displacement. Rather than the deforming, medial angulation within the knee causing the foot to cross the midline for stance, the entire limb is moved laterally by hip abduction. This also widens the distance between the knees. Clinically, this deformity is called *varus* or "*bow-leg*." Quiet standing now shows the knees to be farther apart than the feet. A false impression of knee varus may result from the combination of flexion and external rotation.

Both varus and valgus can result from static or dynamic influences. Static deformities will be present on manual examination. Dynamic angulations represent the effects of altered body position and ligament laxity. Patients often display a mixture of the 2 mechanisms. Prosthetic malalignment could also contribute to varus, valgus, and pain.

Static Factors

Intrinsic congenital or developmental deformities are childhood mechanisms. Trauma can induce a static malalignment of the knee at any age. For example, excessive strain to either the medial or lateral collateral ligaments can contribute to ligamentous laxity and a valgus or varus joint deformity.

Dynamic Factors

The position of the knee at rest is increased by the dynamics of walking. Over time, the abnormal stresses contribute to degenerative joint changes. Osteoarthrosis, rheumatoid arthritis, and the knee joint changes arising from years of walking with a paralyzed gluteus medius provide examples of the influence of dynamic forces over time on coronal gait deviations.

Osteoarthrosis allows the knee to yield to the persistent medial alignment of the body weight vector throughout stance. This creates a greater load on the medal tibial plateau. The osteoarthritic knee reacts to the unequal loads with degenerative changes and increasing knee deformity. Progressive malalignment toward knee varus results. Patients can partially unload the medial knee by using a lateral trunk lean or external rotation of the limb.[9]

Rheumatoid arthritis tends to produce knee valgus. The causative mechanisms appear to be either the lateral trunk lean used to unload a painful hip or a valgus foot deformity.

A gluteus medius paralytic gait pattern may result in knee valgus. In growing children, the repetitious lateral displacement of the trunk to stabilize the hip with abductor muscle weakness (the gluteus medius limp) displaces the vector at the knee and may lead to knee valgus. Bone growth responds to the forces it experiences.

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Chapter 13

Hip Gait Deviations

The multidirectional mobility of the hip makes this joint sensitive to dysfunction in all 3 planes. A further complexity to assessing the effects of hip pathology is its role as the junction between the lower limb and trunk. Abnormal hip function may be displayed by malalignment of either the thigh or pelvis (and indirectly the trunk). Pelvic motion may accompany the displacement of the thigh, remain stationary, or move in the opposite direction depending on the mobility of its articulation with the trunk. Thus, in the assessment of walking, thigh motion analysis should be separated from that of the pelvis. Observation of the thigh relative to vertical identifies what the limb is doing. The functional patterns of both segments, however, are influenced by the interplay between postural demand and hip joint mechanics (mobility and the actions of its controlling muscles).

The potential gait errors in the sagittal plane include excess flexion, limited flexion, or a past retract. Deviations in the other planes are excessive adduction, abduction, and transverse rotation (internal or external).

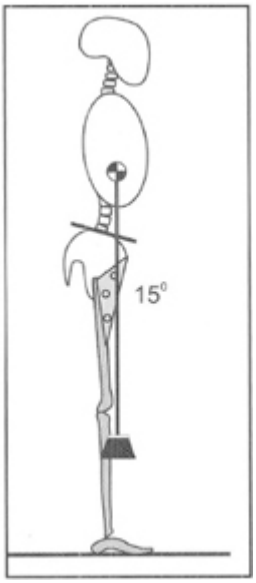

Figure 13-1. Excess hip flexion in mid stance. Without compensation, the pelvis and trunk are tilted forward and demand on the hip extensors increases due to the forward alignment of the body weight vector.

EXCESSIVE SAGITTAL PLANE MOTION

EXCESS FLEXION

Definition: More-than-normal hip (thigh) flexion for a particular phase

Phases: IC, loading response, mid stance, terminal stance, mid swing

Functional Significance: During stance, excess hip flexion increases the demand on the hip extensors and quadriceps unless compensations at the trunk reduce the demand. During mid swing, excess hip flexion is often used to assist with foot clearance when ankle PF is excessive.

Underlying Causes:

* Hip flexion contracture

* IT band contracture
* Hip flexor spasticity
* Compensation for excess knee flexion and ankle DF (stance)
* Hip pain (SLS)

Compensation for excess ankle PF in mid swing

During WA, excess hip flexion (>30°) increases the demand on the hip extensors as body weight rapidly drops onto the outstretched limb. Greater hip flexion during this period can also increase the friction requirements between the foot and the floor surface, making a slip event more likely.[2]

In mid stance, excessive hip flexion can modify the alignments of either the pelvis or thigh. This introduces 3 postural errors in adjacent body segments: forward trunk lean, lumbar spine lordosis, and a flexed knee with increased DF.

Forward tilt of the pelvis is one mechanism that can be used to accommodate limited ROM between the pelvis and the thigh. The forward tilt of the pelvis allows the thigh to advance to a vertical position over the lower leg as knee extension is completed in mid stance (ie, thigh vertical) (Figure 13-1). The forward trunk also places the body weight vector anterior to the hip joint. This increases the demand on the hip extensor muscles.

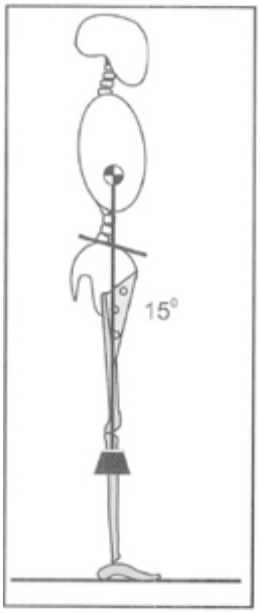

Figure 13-2. A 15° hip flexion contracture is readily compensated by lumbar lordosis to place the body vector over the supporting foot.

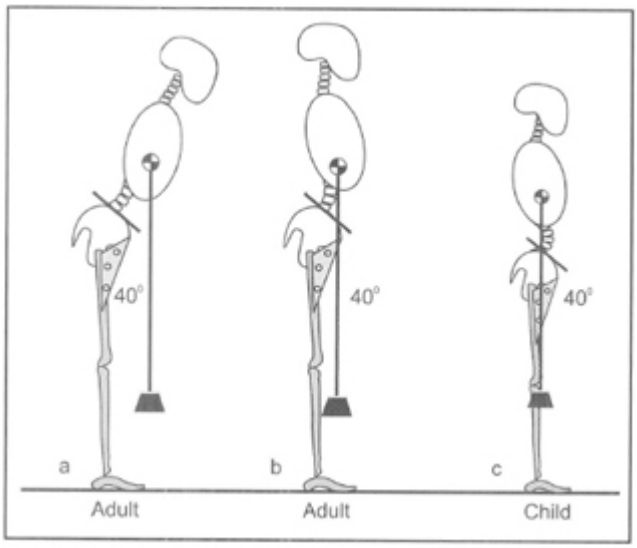

Figure 13-3. A severe (40°) hip flexion contracture. (A) Failure to compensate for the contracture leads to high hip extensor demand due to the anterior location of COG. (B) Adults lack spine mobility to compensate fully, thus COG remains anterior to area of foot support. (C) Children's growth malleability allows the spine to develop necessary lordosis for postural compensation.

The least stressful means of reducing the resulting flexor leverage is to allow the pelvis to tilt forward and use lumbar lordosis to keep the trunk erect. Hip flexion of 15° is easily accommodated by the spine unless it is abnormally stiff (Figure 13-2), but greater loss of hip extension begins to strain spine mobility. Each degree of anterior pelvic tilt (symphysis down) moves the base of the lumbar spine (lumbosacral joint) proportionally more anterior to the hip joint (Figure 13-3A). To realign the body vector over the hip joint (Figure 13-3B), the spine must increase its arch to carry the trunk mass back an equivalent distance. Failure to provide sufficient lumbar lordosis leaves the body vector anterior to the hip joint, and a corresponding greater degree of extensor muscle action (hip and back) is required. In general, children develop more lordosis than adults as spinal growth absorbs the postural stresses (Wolff's law) (Figure 13-3C).

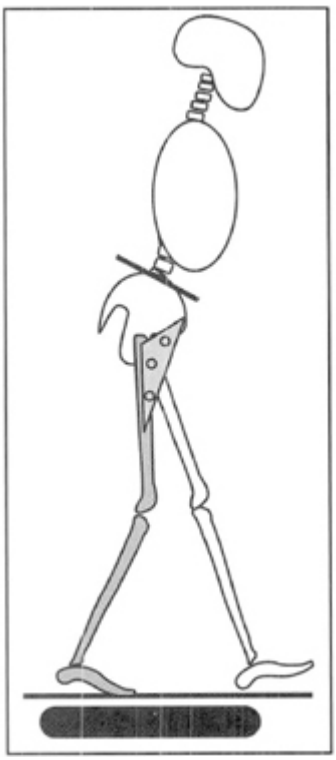

Figure 13-4. Knee flexion equal to the fixed hip flexion can help right both the pelvis and trunk. An early heel rise and/or increased dorsiflexion are required to maintain foot-floor contact.

Flexing the knee tilts the thigh back and allows the pelvis to retain its normal alignment despite the fixed hip flexion (Figure 13-4). Hence, a crouched posture is an alternate means of accommodating to inadequate hip extension in mid stance. This is very inefficient, however, as the flexed knee must be stabilized by increased quadriceps control. Also, excessive ankle DF or an early heel rise onto the forefoot is required to accommodate the tilted tibia. Body progression is significantly reduced.

During terminal stance, the trailing alignment of the thigh (ie, hip hyperextension) magnifies the functional limitations of excess hip flexion. Thus, at the end of SLS, the patient generally exhibits 2 functional deficits: anterior pelvic tilt and loss of a trailing thigh (see Figure 13-4). Anterior tilting of the pelvis (symphysis down) with its associated lumbar lordosis is the first change. As the limits of spine mobility are challenged, the trailing position of the thigh lessens. Knee flexion may be increased to reduce the extensor strain on a hip

with inadequate range. The lack of hip extension also shortens the step of the other limb.

Pre-swing is a transitional situation. The weight-bearing challenge is continually diminishing as body weight is transferred to the opposite limb. The beginning posture most commonly represents a continuation of excess hip flexion from the previous stance phases. Occasionally, there is rapid advancement of the thigh when weight transfer to the other foot unlocks the limb and releases flexors that previously had been held under tension. In either situation, hip flexion for swing is initiated prematurely (Figure 13-5).

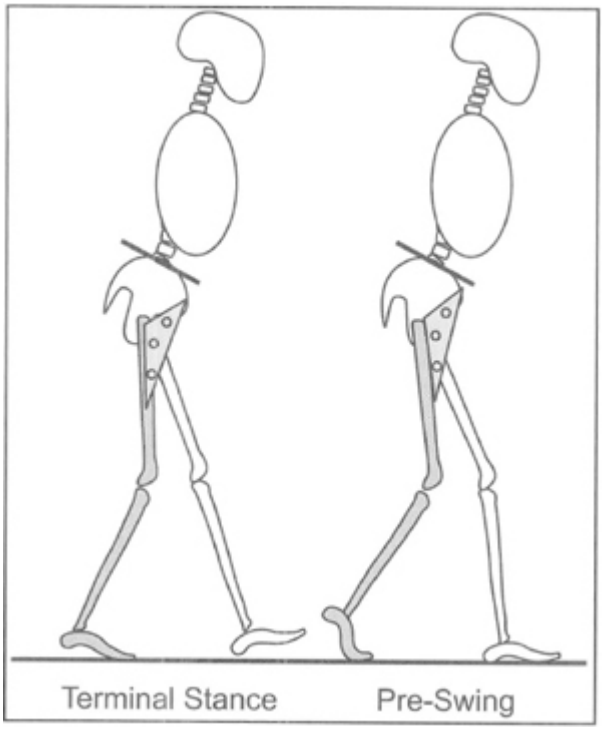

Figure 13-5. In pre-swing, unloading of the limb allows the thigh to advance rapidly, leading to premature swing.

During swing, the fixed hip flexion would have to exceed 35° (normal 25° thigh flexion and 10° anterior pelvic tilt) to be excessive. Most often, this limitation is reflected as increased pelvic tilt rather than an altered thigh position. Excessive elevation of the thigh in mid swing, however, is a common substitution for excessive ankle PF (Figure 13-6).

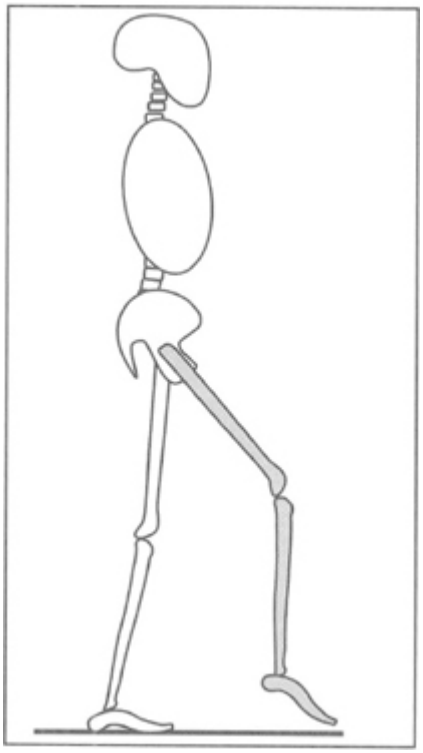

Figure 13-6. Excessive hip flexion is used voluntarily in mid swing to provide floor clearance by a plantar flexed ankle.

There are 5 common pathologies that restrict the mobility of the anterior hip joint tissues. Each can cause excessive hip flexion. These are a flexion contracture, anterior IT band contracture, spasticity of the hip flexors, pain, and hip joint arthrodesis. Excess hip flexion during mid swing can also reflect the need to compensate for distal pathology such as an excessively plantar flexed ankle. Often differentiating the cause of the limited motion depends on dynamic EMG rather than motion analysis.

Hip Flexion Contractures

Shortening of the tissues of the anterior joint capsule or flexor muscles is the most common limitation of full hip extension (see Figures 13-1 and 13-4). As the contracture introduces a fixed hip position, its functional significance varies with the normal angle used during walking. Terminal stance presents the most challenging phase, as full thigh extension plays a key role in achieving a trailing limb posture (see Figure 13-4). In contrast, the thigh is normally

positioned in 20° to 25° of flexion from mid swing until loading response, permitting contractures of this magnitude to be masked within the normal posture of the hip.

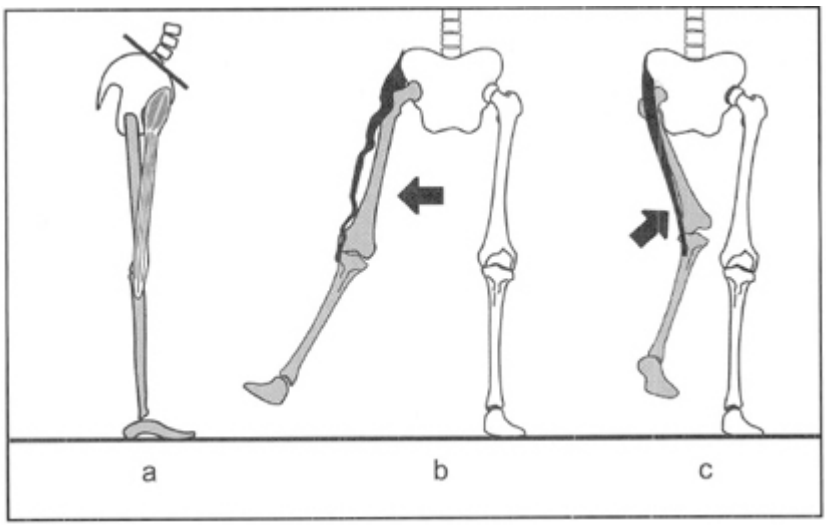

Figure 13-7. Influence of posture on IT band contracture. (A) Standing posture with leg in normal adduction. (B) Supine with the limb abducted, IT band is slack, and hip extends fully. (C) Supine with thigh adducted, IT band is tightened, and the hip pulls into flexion.

Iliotibial Band Contractures

A variant of a hip flexion contracture is a tight IT band. As the name implies, the band extends from the lateral aspect of the iliac crest to the anterior-lateral aspect of the tibia. During walking, the patient exhibits a greater limitation of hip extension than when lying supine (Figure 13-7). The difference is the relative adduction that occurs with weight bearing, which increases IT band tension and introduces an equivalent forward tilt of the pelvis. In contrast, lying supine allows the hip to posture in subtle abduction and external rotation, thus reducing tension on the IT band.

The clinical diagnosis is made with a supine lying test. With the hip widely abducted, the flexed hip can be fully extended. When the

hip is neutrally aligned, hip extension is limited by the contracture in the IT band (see Figures 13-7B and 13-7C). The supine test is preferred to the standard Ober test in clients who have walked with this pathology for a long time. These individuals learn to compensate for the tight IT band with excessive pelvic rotation. The supine test allows for greater control of the pelvis.

Hip Flexor Spasticity

Stretch stimulates spastic muscles to contract. During walking, the spastic hip flexors react when the muscles' free range is exceeded. As 8 muscles cross the hip anteriorly and any or all may be spastic, the timing and magnitude of hip extension limitation varies with the individual. There also is much overlap of associated adduction, abduction, and rotation. Dynamic EMG is the only means of identifying the offending muscles (Figure 13-8).

Similarly, stretch caused by the dangling limb can stimulate a spastic response during the swing phases of walking in patients with upper motor neuron lesions. This would be superimposed on other compensatory maneuvers (selective or synergistic movement). Flexion in excess of 40°, however, is unusual unless there is an associated contracture.

Pain

Arthritis and other joint pathology that cause swelling within the hip joint introduce a flexed posture. Intra-articular pressure is least when the hip is flexed 30° to 60° (see Figure 10-7).[4] Hence, this position is assumed reflexively. The actual degree of flexion varies with the intensity of the joint pathology. SLS is reduced as progression increases the tension on the joint capsule, leading to greater pain.

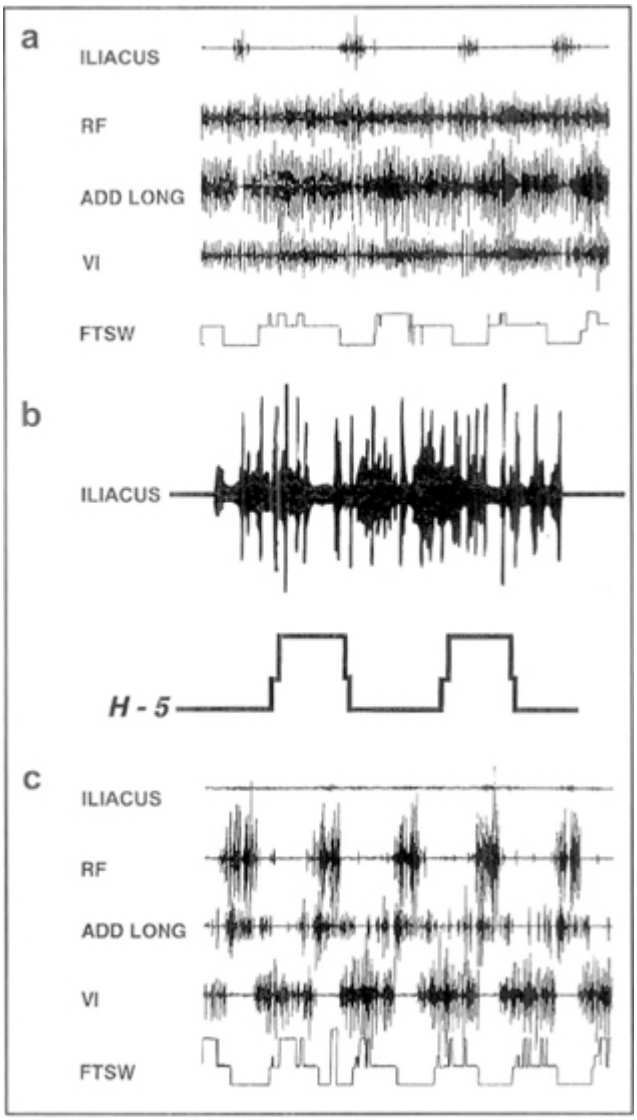

Figure 13-8. Spastic hip flexors contribute to excess hip flexion in stance. (A) Rectus femoris (RF) and adductor longus (ADD LONG) show continuous EMG while iliacus (ILIACUS) timing is normal. (B) Continuous activity of a spastic iliacus (ILIACUS). (C) Useful swing phasing of rectus femoris (RF) and adductor longus (ADD LONG) as a substitution for the inactive iliacus (ILIACUS). Vastus intermedius (VI) has extended action through stance. Footswitches (FTSW) show stance instability.

Arthrodesis

Loss of joint motion from either surgical arthrodesis or spontaneous ankylosis is an infrequent occurrence in this era of total joint replacement. Customarily, to minimize the lordotic stress of walking with a fused hip, the surgical fusion is fixed in a position of flexion between 15° and 30°.[3] In contrast, the spontaneous fusion arising over time from the ankylosis of hip joint pathology is more likely to be between 30° to 60° of flexion,[5] consistent with a position of minimal intra-articular pressure (see Figure 10-7).

The times within the GC that are affected are determined by the angle of joint fixation. Characteristic compensations by the reference limb include increased backward rotation and anterior tilt of the pelvis during late stance to assist with step length, as well as sustained knee flexion throughout stance to accommodate the flexed hip posture.[5] Sound limb hip flexion is increased throughout the GC in part due to the presence of greater anterior pelvic tilt.[5] Depending on the magnitude of the flexed posture, the compensations stress the lumbar spine, ipsilateral knee, and contralateral hip.

Impact of Voluntary Flexion on Excess Hip Flexion

Deliberate flexion of the thigh beyond 30° relative to vertical in mid swing gains floor clearance when the foot is plantar flexed (see Figure 13-6). The added flexion might carry over into terminal swing but would not persist as the limb approached the floor.

Limited Flexion

Definition: Less-than-normal hip (thigh) flexion for a particular phase

Phases: IC, loading response, initial swing, mid swing, terminal swing

Functional Significance: During WA, limited hip flexion may interfere with normal knee flexion and ankle PF. In swing,

limited hip flexion interferes with forward progression of the limb and shortens step length. Foot clearance also may be disrupted.

Underlying Causes:

* Hip flexor weakness
* Hamstring spasticity or over-activity
* Synergistic (patterned) movement
* Hip pain
* Intentional to decrease demand on the primary hip extensors during WA

Resulting from foot drag

Initial floor contact with the thigh positioned in less than 20° flexion reduces the demand on the single joint hip extensors. During initial swing, failure to flex the thigh to 15° (hip 25°) reduces limb advancement. A secondary effect is limited knee flexion as the thigh momentum needed to initiate this action is lacking (see Figure 12-10). This, in turn, contributes to toe drag and ankle PF. As a dragging toe can also inhibit hip flexion, function of the knee and ankle in other gait phases is used to elucidate the cause of the observed limitation at the hip. Little additional flexor muscle action is available in the other phases to increase hip flexion. In mid swing, failure to achieve 25° of thigh flexion will hinder limb clearance. In terminal swing, step length will be shortened if hip flexion remains limited. The lack of adequate hip flexion generally relates to the lack of active or selective muscle control.

Hip Flexor Weakness

Reduced strength or inability to activate the hip flexor muscles may result in a loss of speed or step length.[1] Functional failure of these muscles indicates a major physical impairment as walking requires little of the flexor muscles. With normal motor control, grade 2+ (poor plus) muscle strength is sufficient for an average gait.

Upper motor neuron lesions commonly make the patient dependant on the mass flexor synergy for limb advancement.

Normal initial swing acceleration is lacking. Hence, the limb flexes slowly in initial swing. With primitive patterned control, maximum hip flexion is reached in late mid swing, similar to the normal timing of peak hip flexion.

Weakness of the Primary Hip Extensors

The peak demand on the primary hip extensors (gluteus maximus, adductor magnus) occurs during WA. The hip stabilization requirements of loading response are lessened when impact with the ground occurs with the limb in a more vertical position as the force vector is now more closely aligned to the hip joint center. Thus, one method for reducing the demand on weak single joint hip extensors is to intentionally decrease hip flexion in late swing and impact the ground with the hip positioned in less-than-normal flexion. Alternatively, persons with selective control will substitute with a posterior lean of the trunk to realign the vector posterior to the hip joint. This is not an available compensation if quadriceps weakness is present, however, due to the associated increased demand on the vastii.

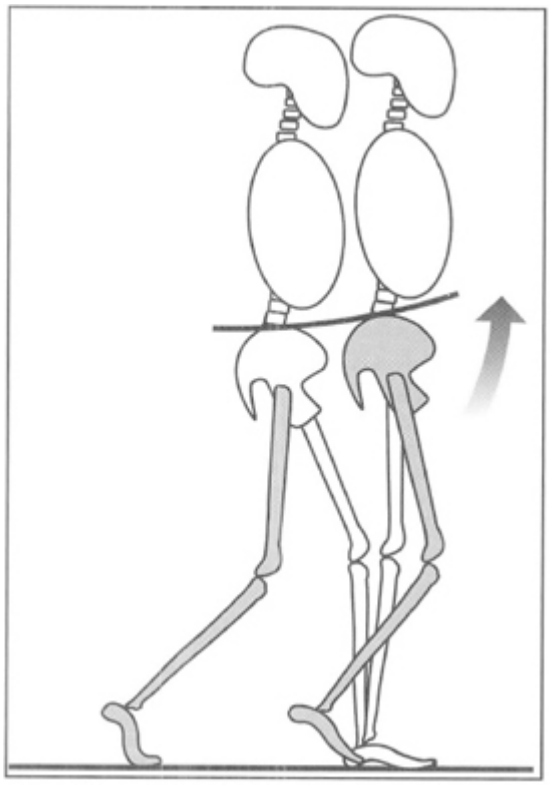

Figure 13-9. Posterior pelvic tilt. Voluntary posterior pelvic tilt to advance the thigh when hip flexion is inadequate.

Hip Extensor Hypertonicity

Increased activation of the spastic hamstring muscles (semimembranosus, semitendinosus, BFLH) may limit step length. The stimulus for this increased activation most commonly occurs in mid and terminal swing.

Hip Joint Arthrodesis

Flexion of the hip during swing is dictated by the fixed position of the joint. Only if the hip is fused in a position less than 35° flexion (25° thigh flexion) would a swing phase have inadequate posturing. When the hip is fused, initiating thigh advancement in pre-swing is delayed until the limb is unloaded.

Substitutive Actions for Limited Hip Flexion

Several substitutions are possible for advancing the limb when primary hip flexion is inadequate. Posterior tilt of the pelvis (symphysis up) uses the abdominal muscles to advance the thigh (Figure 13-9). Circumduction also is common. This combines hiking and forward rotation of the pelvis with abduction of the hip. Advancing the limb in this manner utilizes considerable energy as so much trunk mass must be moved.

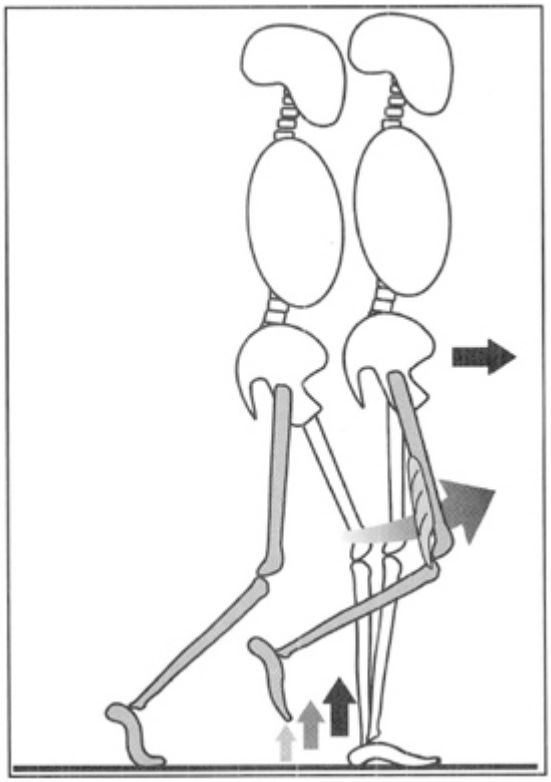

Figure 13-10. Voluntary rapid knee flexion helps flex the flaccid hip by changing the limb's COG.

Voluntary excessive knee flexion is an indirect means of flexing the hip (Figure 13-10). The posterior alignment of shank and foot weight introduces passive thigh advancement to balance total limb weight under the point of suspension (the hip joint). A modest but useful amount of hip flexion results.

Additional substitutions used to ensure floor clearance by an unflexed limb include contralateral vaulting and lateral lean of the trunk to the opposite side.

PAST RETRACT

Definition: An observable forward and then backward movement of the thigh during terminal swing

Phase: Terminal swing

Functional Significance: As a voluntary mechanism, the past retract serves as a means for ensuring full knee extension in terminal swing when quadriceps activity is insufficient. An involuntary past retract occurs when the hip is part of the primitive extensor pattern. The resulting posterior translation of the thigh can impede forward progression and step length.

Underlying Causes:

* Quadriceps weakness
* Synergistic (patterned) movement
* Impaired proprioception at the ankle or knee

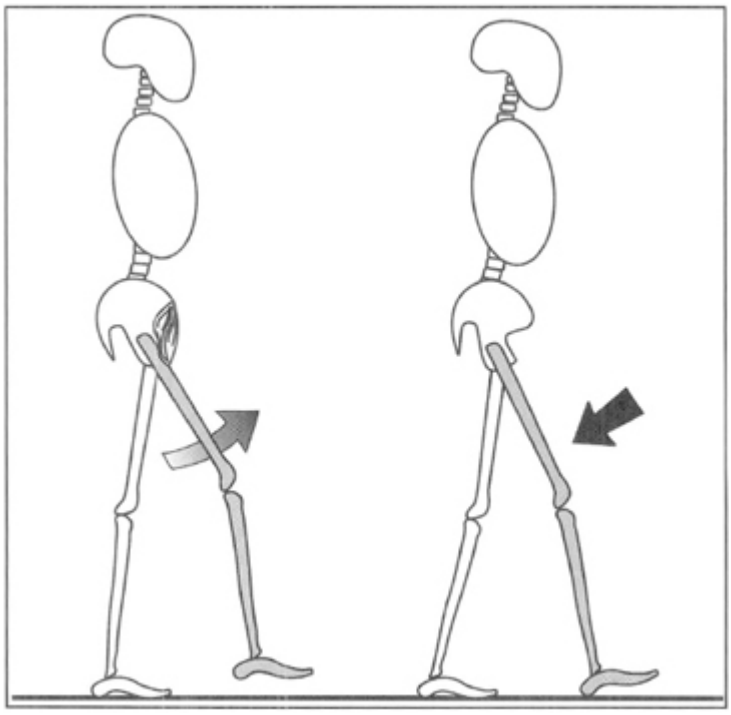

Figure 13-11. The past retract maneuver: Voluntary excessive hip flexion and its release in terminal swing to rapidly extend the flaccid knee by using tibial inertia.

In terminal swing, the hip is flexed and then notably moves toward extension. There are 2 subtle variations of this action. Mid swing may be a period of excessive hip flexion with the terminal swing extension returning the thigh to the normal 20° posture. If flexion during mid swing does not exceed the normal range, then the subsequent extension in terminal swing will result in a reduced posture of flexion at IC.

Quadriceps Weakness

Past retract hip motion is used by persons with a paralyzed quadriceps and normal neural control (poliomyelitis) to extend the knee. Rapid hip flexion advances both the thigh and tibia. Quick, active retraction of the femur allows inertia to continue advancing the tibia (Figure 13-11). In this way, the limb is prepared for IC. The action may be overt or very subtle.

Synergistic Movement and Hypertonicity

Dominance of the primitive patterns in patients with central nervous system disorders also can introduce a past retract motion pattern. In mid swing, the flexor pattern provides limb advancement and floor clearance. Preparation for stance in terminal swing initiates the extensor pattern. When the hip extensors are included in the primitive pattern, hip extension may occur as the knee is straightened. Now, the retraction of a previously flexed hip represents the inability to mix hip flexion and knee extension.

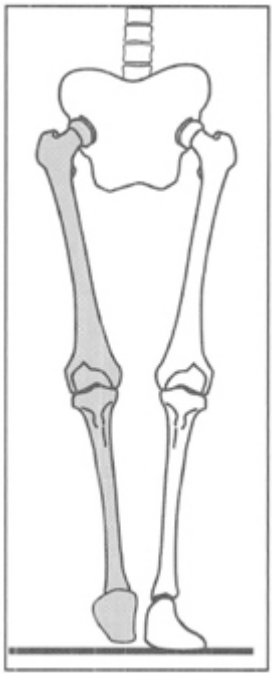

Figure 13-12. Excessive hip adduction in swing limb causing a scissor gait. The whole limb moves medially (thigh and foot).

Hypertonicity of the hamstrings can also result in a past retract. The rapid stretch applied to the muscles during the transition from mid to terminal swing, results in a vigorous burst of activity that reverses the motion of the thigh from flexion to extension.

EXCESSIVE CORONAL PLANE MOTION

Deviation of the thigh from its normal alignment may be either lateral or medial. The use of instrumented analysis has identified minor arcs of normal motion beyond the neutral alignment. Functionally, significant gait deviations represent excessive arcs. To accommodate these different situations, the terms *excessive adduction* and *excessive abduction* are used.

EXCESS ADDUCTION

Definition: Greater-than-normal adduction for a particular phase

Phases: Can occur in any phase

Functional Significance: During stance, excess adduction interferes with stability, while it can interfere with foot clearance and limb advancement in swing

Underlying Causes:

* Abductor weakness with contralateral pelvic drop
* Substitution of adductors for weak hip flexors
* Adductor hypertonicity or contracture
* Leg length discrepancy

A contralateral drop of the pelvis increases hip adduction during the weight-bearing period of walking. This posture begins during loading response and persists through SLS. It corrects in pre-swing as body weight is transferred to the opposite limb.

In swing, excessive hip adduction relates to medial alignment of the whole limb. This begins in initial swing as the hip is flexed and progresses during swing (Figure 13-12). When it is sufficiently severe to cause the swing limb to cross the stance limb, the patient is said to have a “scissor gait.” The result of excessive adduction in swing is a narrow base of support. With a severe scissor gait, progression can be blocked. Floor contact with the foot on the opposite side of the midline obstructs forward swing of the other limb. In initial swing, the foot can catch on the stance limb and advancement is obstructed.

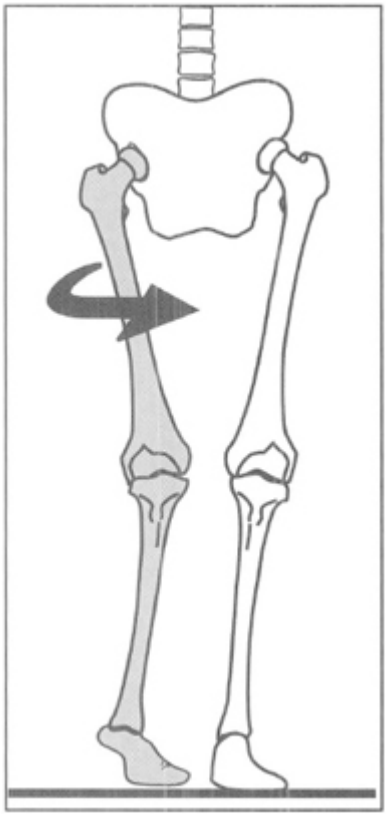

Figure 13-13. Pseudoadduction. Combined hip flexion and internal rotation excessively adducts the thigh (knee), but the foot remains lateral.

The dynamic causes of coronal plane deviations relate to muscle weakness, spasticity, or compensatory substitutions. Static malalignment also can result in an inappropriate hip position during gait. Because obliquity of the pelvis causes excessive adduction of one hip and excessive abduction of the other hip, mobility and muscular control of both sides must be considered in seeking the cause of coronal plane deviations.

Pseudoadduction

Excessive adduction often is confused with combined internal rotation and knee flexion (pseudoadduction) (Figure 13-13). This combination of limb postures directs the knee inward, even to the extent of overlap with the other limb. The 2 situations are differentiated by the relative closeness of the feet. The feet are

separated with pseudoadduction. Conversely, they approximate each other when true adduction exists.

The cause of pseudoadduction is combined gluteus medius and gluteus maximus weakness.[6] The upper gluteus maximus is a synergist of the gluteus medius. The gluteus maximus also provides external rotation as well as extension. Loading the limb with inadequate gluteal support allows the femur to rotate medially and adduct. This has been identified in athletes as a source of retro-patellar knee pain.[6] The customary practice is to emphasize synergistic training of the hamstrings and quadriceps. It has been assumed that there is an overemphasis of the hamstrings as an antagonist to the quadriceps, which allows the hamstrings to provide the hip extension and essentially replace the gluteus maximus. The same situation may exist in the client with cerebral palsy with spastic hamstrings. If adductor over-activity cannot be confirmed, then weakness of the gluteus medius and gluteus maximus should be considered as a cause for the pseudoadduction.

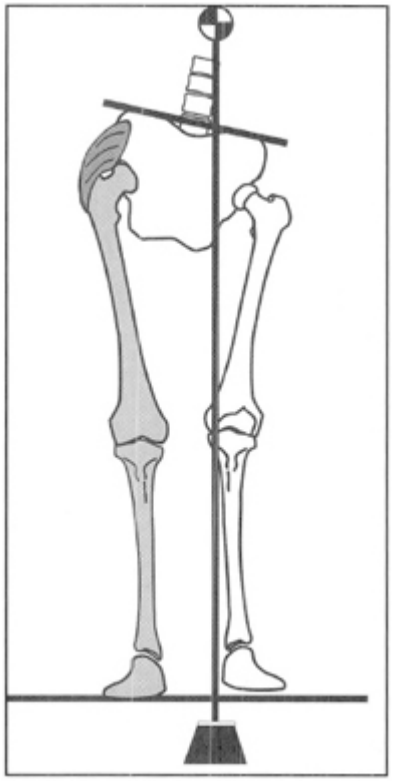

Figure 13-14. Excessive hip adduction caused by weak hip abductors (gluteus medius) allows a contralateral pelvic drop as the

swing limb is lifted.

Abductor Weakness

Lifting the contralateral foot for swing removes the support for that side of the pelvis while body weight is medial to the supporting hip. This creates a strong external adduction torque on the ipsilateral stance limb that must be stabilized by the hip abductors. Hip abductor muscles (ie, the gluteus medius-minimus complex) with less than grade 3 plus (3+) strength cannot prevent the pelvis and trunk from falling to the opposite side (ie, a contralateral drop) (Figure 13-14). This lateral fall begins with the rapid transfer of body weight onto the stance limb in loading response. The pelvic drop continues through the weight-bearing period as there is no mechanism to raise it until the other foot again contacts the floor in pre-swing.

Adductors as Hip Flexors

Substitution of the adductor muscles (longus, brevis, or gracilis) for a weak or absent iliacus muscle (the primary hip flexor) leads to medial displacement of the thigh (excessive adduction) in swing (Figure 13-15). Spastic activation of the adductor muscles by stretch of the dangling limb creates the same situation.

If the hip is fully extended at the onset of adductor muscle action, advancement of the thigh will be accompanied by external rotation. Having a flexed position at the start of the adductor muscle action introduces internal rotation.

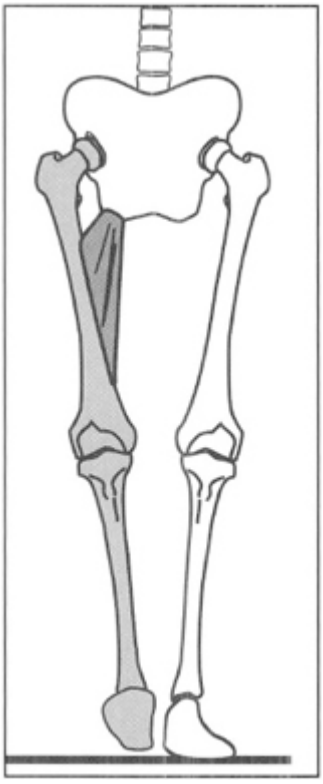

Figure 13-15. Excessive hip adduction is caused by the hip adductor muscles compensating for the weak primary hip flexors.

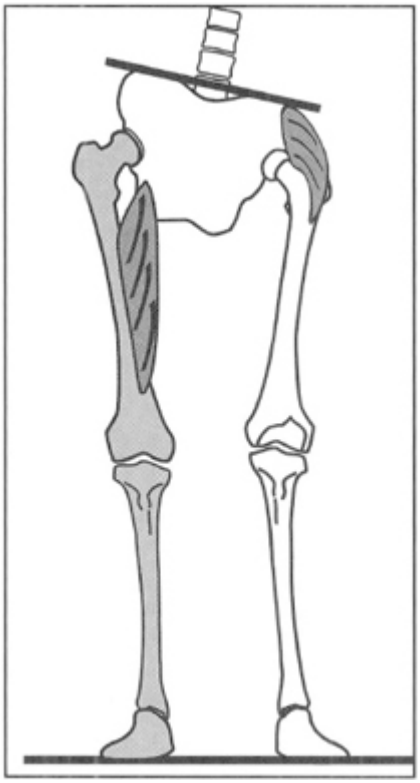

Figure 13-16. Excess hip adduction caused by an ipsilateral adductor muscle contracture or spasticity. The secondary effect is a contralateral pelvic drop. The same posture can be caused by a contralateral hip abduction contracture.

Adduction Contracture or Spasticity

Static posturing of the hip by fibrous tissue tightening causes a continuous deviation throughout the GC (Figure 13-16). Very commonly, an adduction contracture is associated with internal rotation and flexion. Spasticity can mimic contracture when the patient is standing but be mild or absent when the patient is supine.

EXCESS ABDUCTION

Definition: Greater-than-normal abduction for a particular phase

Phases: Can occur in any phase

Functional Significance: During stance, it widens the base of support. During swing, it can assist with foot clearance by decreasing the relative length of the limb.

Underlying Causes:

* Abductor contracture
* Leg length discrepancy
* Scoliosis with pelvic obliquity
* Pain associated with the medial brim of a knee-ankle-foot orthosis (KAFO) or transfemoral prosthesis

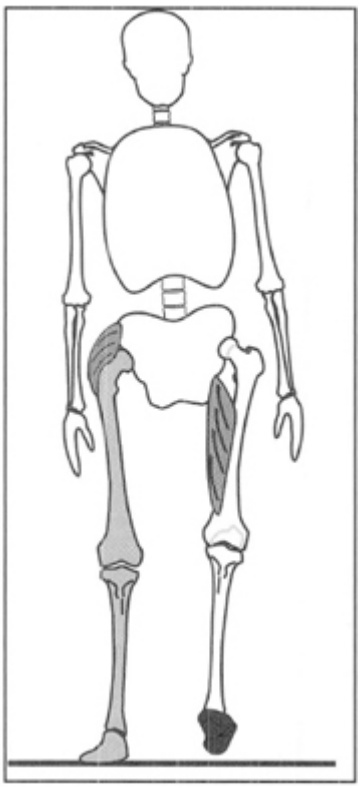

Figure 13-17. Excessive hip abduction caused by an abduction contracture displaces the limb laterally (wide-based gait). An ipsilateral trunk lean during stance moves the trunk mass more in line with the center of the foot. The ipsilateral pelvic drop reduces lateral displacement of the leg.

Lateral displacement of the thigh during stance presents a wide-based gait. This form of excessive hip abduction increases stance stability but also requires greater effort during walking to move the body from one limb to the other. In swing, floor clearance is made easier.

Abduction Contracture

Shortening of the abductor musculature or capsule displaces the femur laterally. The patient's gait generally is a mixture of wide base (foot to the side) and an ipsilateral pelvic drop (Figure 13-17). This relatively lengthens the leg, making the initiation of swing difficult. Abductor spasticity could create the same situation but this is rare.

Conversely, grossly inadequate adductor tone can result in an excessively abducted limb.

A tight IT band causes hip abduction with extension, but neutral alignment is possible when the hip is flexed (see Figure 13-7). Hence, patients generally show a mixture of excessive flexion, abduction, and pelvic tilt.

Voluntary Abduction

Patients with good control of their trunk muscles commonly substitute abduction for the lack of adequate hip flexion motion as the means of advancing the limb in swing (Figure 13-18). Generally hip abduction is combined with pelvic rotation and hiking to complete the motion complex commonly called *circumduction.* Ipsilateral abduction as part of a trunk lean also is used to help the other limb clear the floor.

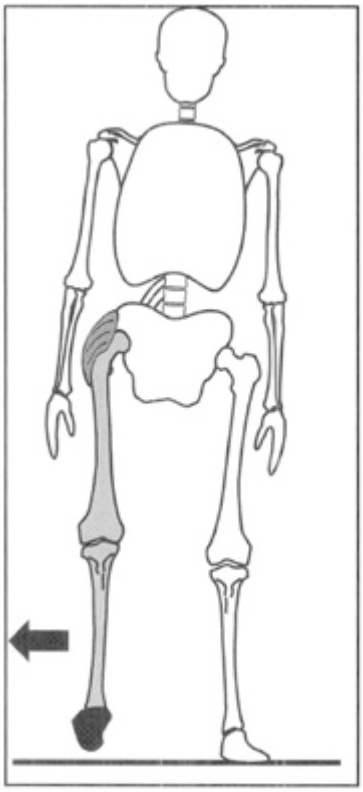

Figure 13-18. Excessive hip abduction by voluntary displacement provides floor clearance in swing as a substitution for inadequate

knee flexion.

Abduction in stance is used to widen the base of support. This is a frequent substitution for impaired balance.

Leg Length Discrepancy

When the reference limb is shorter than the opposite limb, an ipsilateral pelvic drop is used during stance to accommodate the lack of leg length. This puts the hip into excessive abduction. This is not a significant finding unless the leg length discrepancy is severe.

When the reference limb is longer than the opposite limb, hip abduction may be used to assist with foot clearance during swing of the reference limb. Alternatively, a pelvic hike or increased hip flexion may also be used to prevent the reference foot from dragging.

Pain

Individuals with medial groin pain secondary to an ill-fitting KAFO or a transfemoral prosthesis often walk in excess abduction during stance. This posture lessens compression on sensitive tissues (eg, the proximal adductor tendons) but is not a good long-term solution.

Scoliosis With Pelvic Obliquity

Pelvic obliquity secondary to scoliosis leads to excessive abduction in the hip on the low side. Conversely, the hip on the high side will be in excessive adduction. The oblique pelvis also can represent an accommodation to fixed abduction or adduction within the hip joint.

Contralateral Hip Adduction Contracture

The pelvis is tilted down as the contralateral contracted limb assumes a vertical alignment for weight bearing. This creates an ipsilateral pelvic drop and excess hip abduction for the reference limb, which also results in relative lengthening (see Figure 13-16). If there is adequate quadriceps and hip extensor control, the patient substitutes with knee flexion to shorten the reference limb.

EXCESSIVE TRANSVERSE PLANE ROTATION

Any visible transverse rotation of the limb represents excessive motion as the normal arc of 5° (10° total displacement) is obscured by the anterior-posterior changes in limb alignment. While the arcs of transverse hip rotation in normal gait are too small to be apparent by observational analysis, there is a sense of fluidity in the body motions. Excessive rotation of the thigh can arise from too much motion at the hip, pelvis, or trunk. Hence, the originating site of the excessive rotation must be determined as well as the magnitude of the limb displacement.[7]

EXCESS EXTERNAL ROTATION

Definition: Greater-than-normal external rotation for a particular phase

Phases: Can occur in any phase

Functional Significance: Excessive external rotation results in the limb being in a toe-out position, provides a widened base of support, aids progression over the forefoot rocker, and assists with foot clearance during swing. Excessive external rotation may also increase stress on ligaments of the hip or knee during stance.

Underlying Causes:

* External rotation contracture

* Intentional to avoid compression on the arthritic joint by assuming a loose-packed position
* Gluteus maximus over-activity
* Compensation for PF contracture in stance to facilitate progression
* Compensation for the reference limb being too long in swing

External hip rotation is often accompanied by a laterally facing patella. Close observation of the ipsilateral pelvis, femoral condyles, patella, and distal malleoli assists in discerning whether the rotation between the femur and pelvis is truly excessive or whether the posture is actually arising at an adjacent location (eg, excessive backward rotation of the pelvis). Excess external rotation at the hip arises from a number of factors, including over-activity of muscles that are external rotators as well as intentional posturing to compensate for limitations at other joints (eg, a hallux rigidus).

External Rotation Contracture

Individuals with transfemoral amputations may develop excessive external rotation if not properly positioned postoperatively. Tightness of the IT band or the gluteus maximus can also lead to excessive external rotation.

Gluteus Maximus Over-Activity

Normally, the hamstrings serve as the primary muscular force for decelerating the leg in terminal swing. Excessive activity of the gluteus maximus during this period can cause rapid external rotation of the hip.

Intentional to Reduce Painful Hip Joint Compression

External rotation reduces compressive forces within the hip joint.[4] This posture can be useful when pain or joint effusion is present.

Excessive Ankle Plantar Flexion

In SLS, excessive external rotation may accompany marked equinovarus if the ankle PF threatens to block forward tibial advancement. The external rotation shortens the forefoot lever and eases advancement across the foot. This appears to be a useful means of circumventing the barrier to progression that the lack of ankle DF imposes.

Compensation for a Long Leg During Swing

External rotation accompanies abduction as a means of assisting with foot clearance when the swing limb is relatively too long. These 2 motions are part of the collective term *circumduction.*

EXCESS INTERNAL ROTATION

Definition: Greater-than-normal internal rotation for a particular phase

Phases: Can occur in any phase

Functional Significance: Results in toe-in position and can increase stress on the lateral joint during stance. Associated limb lengthening makes toe clearance more difficult during swing.

Underlying Causes:

* Contracture or spasticity of the internal rotators
* Femoral anteversion
* Deliberate action to increase knee stability during stance when the quadriceps are weak

The patella often faces medially when the hip is excessively internally rotated. As with excessive external rotation, close observation of the adjacent joints is required to confirm the anatomic origin of excess rotation. Excess internal rotation at the hip occurs for a variety of reasons, including inappropriately timed muscle activity, contractures, and compensation for quadriceps weakness.

Medial Hamstring Over-Activity

The semimembranosus and semitendinosus lie both posterior and medial to the hip joint, making these muscles natural internal rotators. Over-activity as a result of spasticity or strong involvement in a primitive pattern accentuates the rotatory effect of these muscles. Because the hamstring muscles are flexors at one joint (knee) and extensors at the other (hip), their action can accompany either the flexor or extensor primitive locomotor patterns.

Adductor Over-Activity

Flexion of the hip by the adductors is accompanied by internal rotation if the hip's resting position included some flexion. Internal rather than the expected external rotation occurs because the flexed position displaces the insertion on the femur anterior to the vector between the hip and knee (Figure 13-19).

Anterior Abductor Over-Activity

The tensor fascia lata and anterior portion of the gluteus medius produce internal rotation. Use of these muscles to assist hip flexion will result in excessive rotation of the hip.

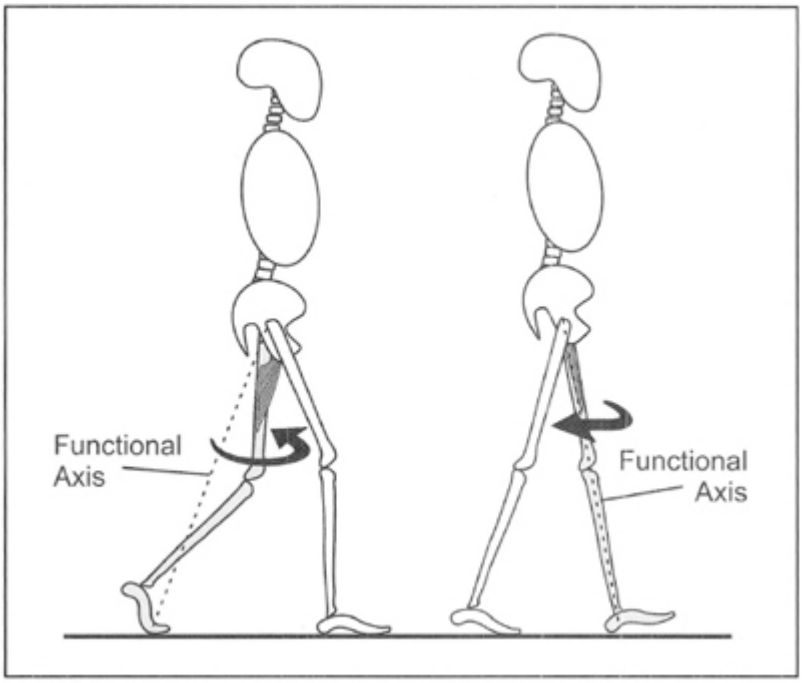

Figure 13-19. The adductor longus inserts on the posterior portion of the femur. The muscle's rotational contribution is influenced by the location of its insertion and the limb's functional axis. With the hip extended, the muscle serves as an external rotator. With the hip flexed, the adductor longus serves as an internal rotator.

Quadriceps Weakness

Internal rotation of the thigh utilizes the lateral knee ligament and IT band to resist the sagittal thrust of limb loading that, otherwise, would flex the knee. This is a voluntary substitution for a very weak (or absent) quadriceps when there is no available hyperextension at the knee.

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Chapter 14

Trunk and Pelvis Gait Deviations

The average change in the position of the pelvis is only 5°, and the trunk normally maintains a neutrally aligned erect posture. Visible deviations of the pelvis or trunk from neutral represent abnormal function.

PELVIS

Excessive pelvic motion can occur in any of the 3 planes of motion. Inadequate pelvic motion is seen as stiffness.

SAGITTAL PLANE PELVIC DEVIATIONS

Gait errors in this plane are identified as forms of pelvic tilt. The direction of the motion has been described by 2 commonly used sets of terms: *anterior and posterior* or *upward and downward*. Definition of these terms has varied according to the anatomical site selected as the apex of the motion (ie, the symphysis or sacrum). To circumvent this confusion, the more obvious terms of *symphysis up* and *symphysis down* have been substituted. As these phrases are euphemistically awkward, their best use may be to clarify the definitions of anterior tilt (symphysis down) and posterior tilt (symphysis up).

Anterior Tilt (Symphysis Down)

Definition: Tilting of the pelvis in the sagittal plane so that the pubic symphysis is pointed downward beyond the normal gait posture (10° anterior pelvic tilt).[2] The anatomical landmarks used to define the tilt of the pelvis in the sagittal plane are the anterior superior iliac spines (ASIS) and the posterior superior iliac spines (PSIS). During normal gait, a line connecting the PSIS to the ASIS tilts 10° relative to horizontal.

Phases: Any phase

Functional Significance: During terminal stance, increased anterior pelvic tilt may contribute to a better trailing limb posture if hip extension is limited. Excess anterior pelvic tilt, when accompanied by increased lumbar lordosis, can lead to low back pain.

Underlying Causes:

* Weak hip extensors
* Hip flexion contracture or spasticity
* Abdominal muscle weakness

Phasic timing of the abnormal posturing varies with the cause. A tilt of 30° is not uncommon.

Weak Hip Extensors

In loading response, the symphysis moves down as a reaction to the anterior alignment of the CG relative to the hip joint when the extensor muscles lack the strength to restrain the pelvis. This posture may continue throughout stance or revert to the neutral gait alignment (10° anterior pelvic tilt) when the demands on the hip extensors are relieved by the trailing limb position in terminal stance. Surgical release or over-lengthening of spastic hamstrings to relieve a crouch gait may cause hip extensor weakness, especially when spasticity of the hip flexors is overlooked.

Hip Flexion Contracture or Spasticity

The pelvis may be drawn into an anterior tilt in any of the stance phases depending on the severity of the deformity. With contractures of 30°, the postural change starts in mid stance as the limb becomes vertical and increases in terminal stance and pre-swing when the thigh assumes a trailing posture (Figure 14-1). Lesser deformities start later. Contractures or joint fusions that exceed 40° may introduce a symphysis-down posture at IC and become prominent by the end of loading response.[1] Spasticity can be partially differentiated from contracture by supine clinical testing. Dynamic EMG is needed during gait. This distinction assists in surgical planning and prediction of long-term outcome.

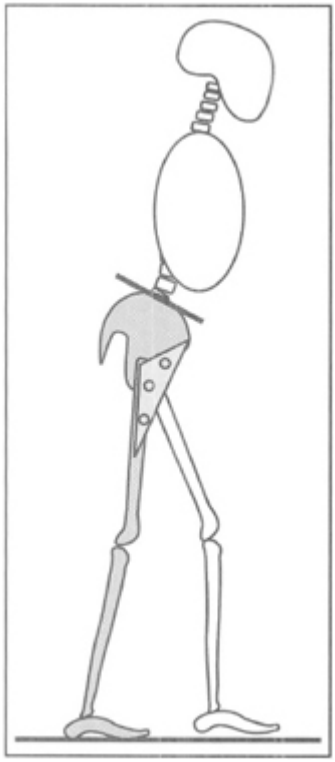

Figure 14-1. Anterior pelvic tilt (symphysis down) resulting from a hip flexion contracture (or spasticity).

Abdominal Muscle Weakness

Insufficiency of the abdominal muscles can result in an increased anterior pelvic tilt at any point throughout the GC. The periods of greatest susceptibility, however, are terminal stance and terminal swing based on muscular demands.[3]

Posterior Tilt (Symphysis Up)

Definition: Tilting of the pelvis in the sagittal plane so that the pubic symphysis points upward beyond the normal gait posture (ie, 10° anterior pelvic tilt)

Phases: Any phase

Functional Significance: Posterior pelvic tilt is a sign of tight hamstrings or can be used as a method of substitution for weak hip flexors

Underlying Causes:

* Hip flexor weakness
* Tightness of the hamstrings
* Low back pain or limited extension range of the lumbar spine
* Hip extensor weakness

Tilting the pelvis posteriorly to create a symphysis-up posture is an infrequent event and must be differentiated from correction of a previous anterior tilt.

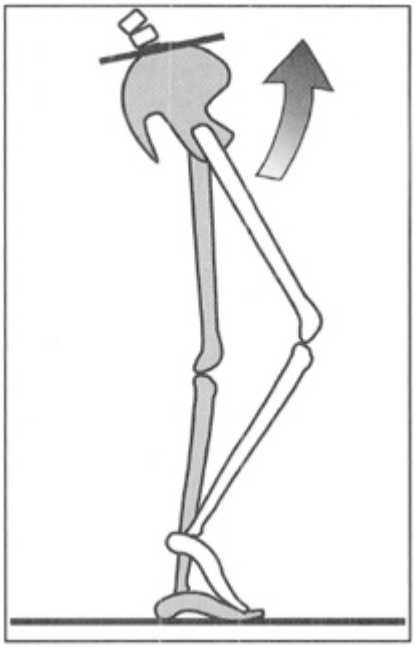

Figure 14-2. Posterior pelvic tilt (symphysis up). The trunk leans back. The swing limb thigh is advanced if the hip remains at neutral. With the thigh vertical, the stance limb hip moves into relative hyperextension.

Hip Flexor Weakness

During early swing, a posterior tilt of the pelvis can be used to augment advancement of the thigh if the hip flexors (iliacus, sartorius, gracilis) are weak (Figure 14-2). Persons who have a transfemoral amputation may use this strategy to assist with prosthetic limb advancement if the remaining hip flexor muscles lack sufficient strength.

Hamstring Tightness

In terminal swing, tension of the hamstrings can contribute to a posterior tilt of the pelvis. This is typically accompanied by excessive flexion of the knee. The posterior pelvic tilt will continue until early WA, when normal knee flexion reduces tension on the hamstring muscles.

Low Back Pain or Reduced Range of Motion

Pain in the low back is frequently accompanied by an appearance of stiffness in the lumbar region and a flattening of the normal lordotic curvature. Normal extension range of the lumbar spine may be lacking.

CORONAL PLANE PELVIC DEVIATIONS

Two clinical terms are used to define abnormal pelvic motion in the coronal plane: hip hike and pelvic drop. Hip hike (more correctly called *pelvic hike*) indicates lateral elevation of the pelvis above the neutral axis. Pelvic drop, conversely, implies descent of the pelvis. This is differentiated into contralateral drop and ipsilateral drop. For both deviations, it is important to isolate a primary motion error from correction of prior malpositioning.

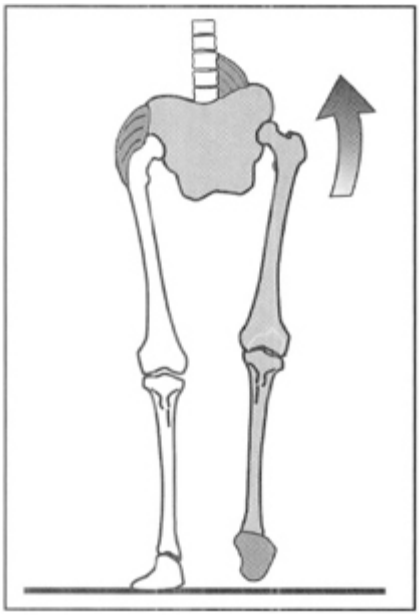

Figure 14-3. Ipsilateral pelvic hike. The reference side of the pelvis is elevated. This is a deliberate action that is coordinated with the

swing limb.

Pelvic Hike

Definition: Elevation of one side of the pelvis above the neutral horizontal plane

Phases: Initial swing, mid swing, terminal swing

Functional Significance: Assists with foot clearance of the swing limb. May increase energy cost required to clear the limb.

Underlying Causes:

* Limited knee flexion in initial swing or hip flexion during mid swing
* Excess ankle PF during mid swing

The clinical term *pelvic hike* refers to excessive elevation of the ipsilateral side of the pelvis. This is a swing phase event (Figure 14-3). It is a deliberate action that begins in initial swing, continues through mid swing, and then corrects in terminal swing. The purpose is to assist foot clearance when either hip or knee flexion is inadequate. The presence of excessive ankle PF in mid swing combined with inadequate hip or knee flexion is another common cause of pelvic hiking.

Mild (5°) ipsilateral elevation of the pelvis in stance is a normal recording with instrumented gait analysis as the middle of the pelvis is the point of reference and it drops at the onset of limb loading. Contralateral pelvic hike occurs as the other limb experiences the same functions.

Contralateral Pelvic Drop

Definition: Contralateral iliac crest is lower than the ipsilateral iliac crest

Phases: Loading response, mid stance, terminal stance

Functional Significance: May reduce the stability of the stance limb and increase the relative length of the opposite limb

Underlying Causes:

* Ipsilateral hip abductor weakness
* Ipsilateral hip adductor contracture or spasticity
* Contralateral hip abductor contracture

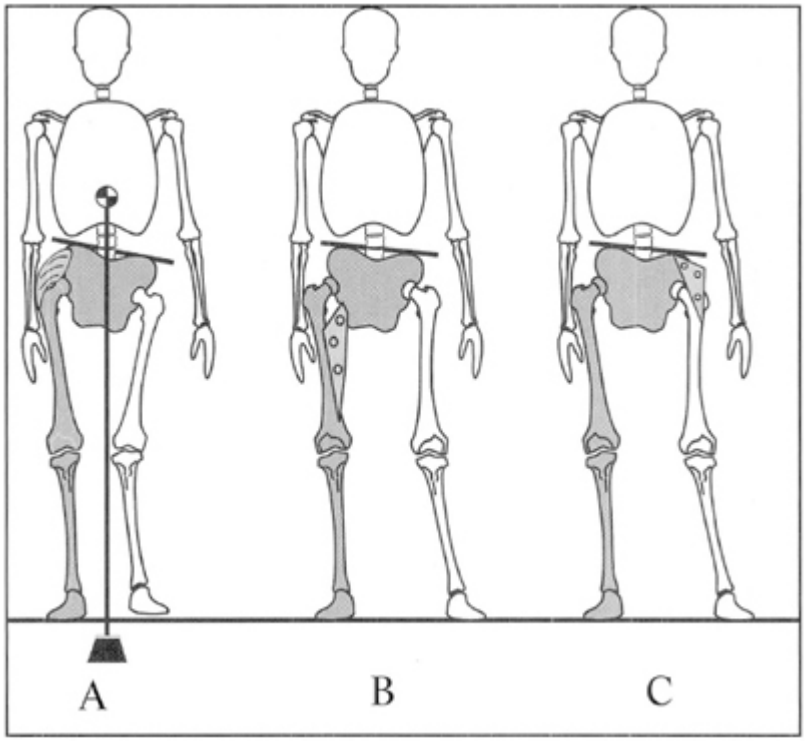

Figure 14-4. Common causes of contralateral pelvic drop. (A) Weakness of the ipsilateral hip abductor. (B) Ipsilateral adductor contracture (or spasticity). (C) Contralateral abduction contracture.

Descent of the opposite side of the pelvis occurs in stance. It begins as body weight is dropped onto the limb in loading response and persists through terminal stance. With instrumented motion analysis, contralateral pelvic drop is identified as ipsilateral pelvic elevation. This occurs because the midline reference point drops while the stance limb's hip joint maintains its height.

Hip Abductor Muscle Weakness

Abductor muscle strength less than grade 3+ creates an unstable pelvis in stance. Unloading the opposite limb in preparation for swing removes the support for that side of the pelvis. If the ipsilateral hip abductors are unable to meet the challenge of stabilizing the pelvis in the frontal plane, a contralateral pelvic drop will occur due to the medial alignment of body weight relative to the supporting hip joint (Figure 14-4A). This action begins in loading response due to the rapid transfer of body weight to the stance limb. With greater incompetency of the abductors, the contralateral pelvic drop is accompanied by an ipsilateral trunk lean to preserve stance stability (Figure 14-5). Weakness of the hip abductor muscles can be masked by a tight IT band.

Hip Adductor Contracture or Spasticity and Contralateral Hip Abductor Contracture

During mid stance, the contralateral pelvis is drawn down as the femur assumes a relatively vertical posture (Figure 14-4B). Generally, there is associated hip flexion and internal rotation. The same pattern of pelvic obliquity seen with tight hip adductors can be created by excessive abduction in the opposite hip (Figure 14-4C). This makes the ipsilateral limb relatively short.

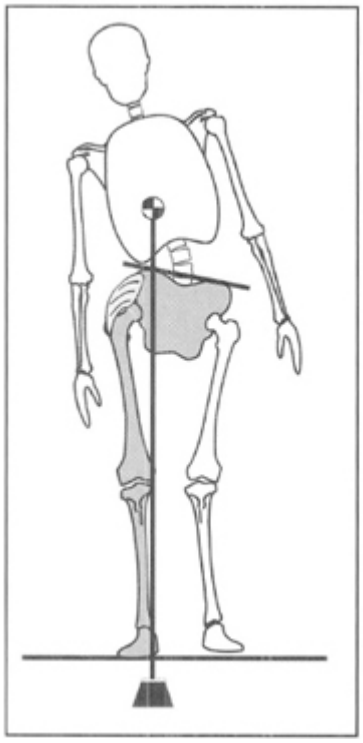

Figure 14-5. When profound hip abductor weakness is present, standing balance can be re-established by compensating with trunk lean to move the COG toward the stance limb.

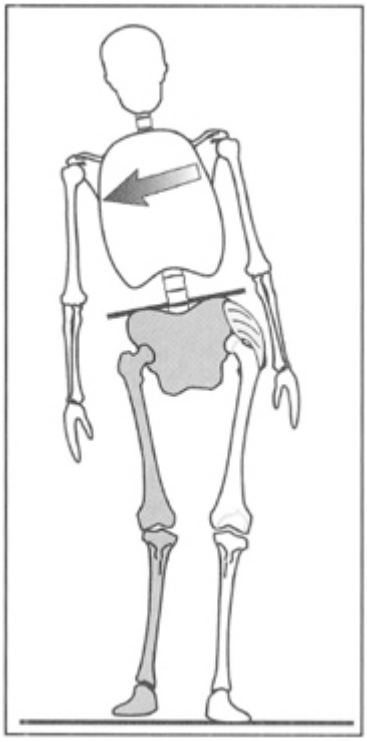

Figure 14-6. Ipsilateral pelvic drop accompanies the swing limb when contralateral hip abductors are weak.

IPSILATERAL PELVIC DROP

Definition: Ipsilateral iliac crest is lower than the contralateral iliac crest

Phases: All phases

Functional Significance: During swing, an ipsilateral drop increases the relative length of the reference limb. During stance, prolonged walking with an ipsilateral drop may lead to back pain.

Underlying Causes:

*Contralateral hip abductor weakness

* Ipsilateral calf muscle weakness

* Scoliosis

* Short leg

Dropping of the ipsilateral side of the pelvis occurs most frequently in swing (Figure 14-6). It often reflects contralateral pathology.

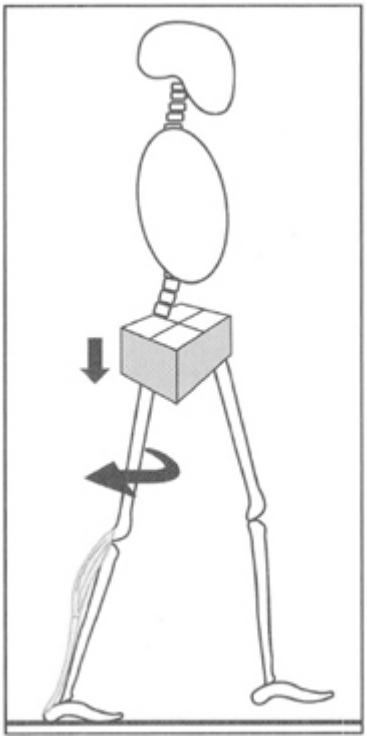

Figure 14-7. Ipsilateral pelvic drop and posterior pelvic rotation. The cause is a weak soleus on the ipsilateral stance limb, leading to an absent heel rise and relative shortening of the stance limb.

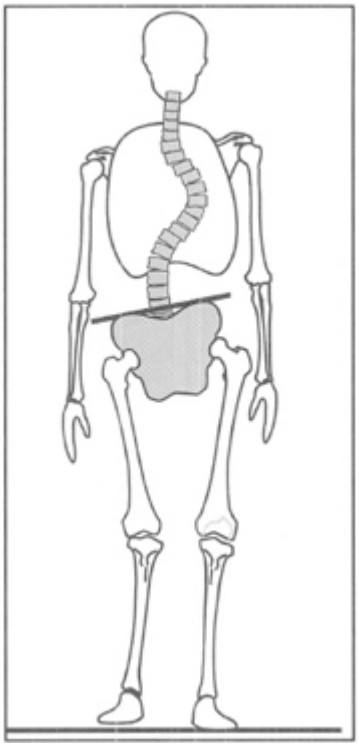

Figure 14-8. Pelvic drop as part of scoliosis curve.

Contralateral Hip Abductor Weakness

During pre-swing, the rapid transfer of weight from the trailing limb to the leading limb (which is in loading response) places a high demand on the lead limb's abductor muscles. When the muscle force is insufficient to stabilize the pelvis in the frontal plane, an ipsilateral drop occurs. The pelvis drops during pre-swing and this often persists through terminal swing (see Figure 14-6).

Ipsilateral Calf Muscle Weakness

In terminal stance, insufficient calf muscle strength for a weight-bearing heel rise results in relative shortening of the limb as it assumes a trailing position. Limb length is gained by dropping the pelvis on that side. This is accompanied by excessive backward rotation of the pelvis (Figure 14-7).

Scoliosis

Deformity in the spine can lead to a static malalignment of the pelvis and present as either a contralateral or ipsilateral drop. Deviations of the pelvis may be oblique as well as lie in the coronal plane (Figure 14-8).

TRANSVERSE PLANE PELVIC DEVIATIONS

Rotation of the pelvis may be excessive or lacking. Also the direction of the gait error may be forward or backward. Whenever rotation of the pelvis is visible, it is excessive. By instrumentation, this would be greater than 5°. In contrast, when the linkage between

the leg and trunk appears stiff, this is often a sign of limited pelvic rotation.

Excessive Forward Pelvic Rotation

Definition: Greater-than-normal forward pelvic rotation in the transverse plane

Phases: IC, loading response, mid stance, mid swing, terminal swing

Functional Significance: Increases step length during terminal swing

Underlying Causes:

* Intentional to advance the limb during terminal swing
* Excess backward pelvic rotation of the contralateral limb

The pelvis may have a fixed forward alignment or move in company with the swing limb. Rapid pelvic forward rotation in initial swing and continuing into mid swing is a means of advancing the limb when the hip flexors are incompetent.

Excessive Backward Pelvic Rotation

Definition: Greater-than-normal backward pelvic rotation in the transverse plane

Phases: Mid stance, terminal stance, pre-swing, initial swing, mid swing

Functional Significance: Improves trailing limb posture

Underlying Causes:

* Compensatory for weak calf with no heel-off
* Compensatory maneuver to increase the trailing limb posture in the presence of a hip flexion contracture

The pelvis may have either a fixed backward alignment or dynamic backward rotation may be increased.

Calf Muscle Weakness

Dynamic backward rotation occurs in terminal stance. It is a fairly abrupt motion that accompanies persistent heel contact in a person who has a moderately good gait velocity. The cause is calf muscle weakness (see Figure 14-7). The lack of heel rise makes the limb relatively short. Length is gained by a combined backward and downward rotation of the pelvis.

Hip Flexion Contracture

When the ipsilateral hip flexors are tight or the hip has been fused in flexion, excess backward rotation of the pelvis can be used as a compensatory maneuver to increase the trailing limb posture during terminal stance.[1] The forward reach of the contralateral limb also will be lengthened because excess forward rotation of the contralateral pelvis occurs at the same time as excess ipsilateral backward pelvic rotation.

LIMITED PELVIC ROTATION (FORWARD or BACKWARD)

Definition: Less-than-normal pelvic rotation in the transverse plane for a given phase

Phases: Terminal stance, terminal swing

Functional Significance: Reduces step length

Underlying Causes:

* Impaired motor control of the trunk and pelvic muscles
* Surgical fusion
* Back pain

Insufficient pelvic motion is seen in patients with spastic rigidity of the spine. Surgical fusion is a less frequent cause. Low back pain (lumbar region) is a third cause. Visually, the patients appear stiff.

TRUNK

A direct correlation between trunk (spine or abdominal) muscle strength and posture has not been recorded. Clinical experience, however, indicates there is no significant deviation from normal until the muscle weakness is marked, generally less than grade 3 (fair). This is consistent with the small amounts of muscular activity needed to control the minor postural deviations induced by the mechanics of walking.[3] When muscle strength is insufficient, the postural deviation is constant. This also is true for spastic muscle imbalance or skeletal abnormalities causing scoliosis, kyphosis, or lordosis. The phasic changes in trunk alignment recorded during walking represent postural adaptations to inadequate mobility or faulty muscle control at the hip, knee, or ankle.

A deviation in trunk alignment from the neutral upright posture is classified as a trunk lean. The direction may be backwards, forwards, ipsilateral, or contralateral. Also, the trunk may rotate toward or away from the reference limb.

SAGITTAL PLANE TRUNK DEVIATIONS

Backward Lean

Definition: Posterior alignment of the shoulder girdle relative to the pelvic girdle

Phases: All phases

Functional Significance: Contributes to hip stability in stance and assists with advancing the thigh during swing

Underlying Causes:

* Compensation for weak hip extensors during stance
* Compensation for weak hip flexors during swing

Displacement of the trunk posterior to the vertical axis uses the weight of the trunk as a substitution for inadequate hip muscle strength. The mode of compensating for weak hip extensors and hip flexors differs.

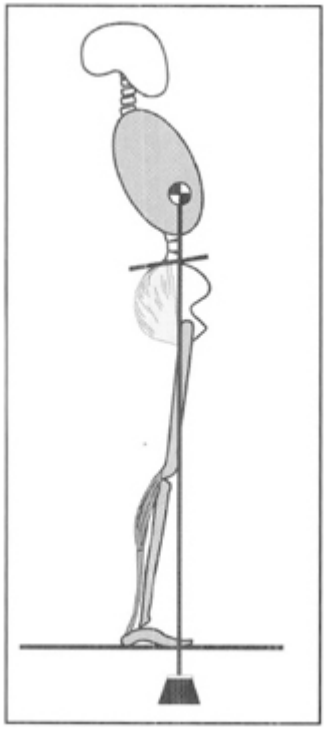

Figure 14-9. Backward trunk lean displaces the COG behind the hip joint and substitutes for weak hip extensors. The soleus stabilizes the tibia. Hip hyperextension and ankle dorsiflexion are needed to maintain COG over the foot.

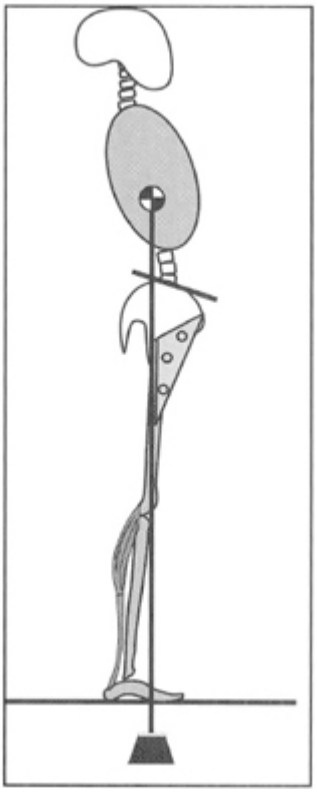

Figure 14-10. Backward trunk lean and lordosis accommodate a rigid hip flexion contracture. The pelvis is tilted anteriorly.

Weak Hip Extensors

In stance, a backward lean of the trunk substitutes for weak hip extensors by placing the body vector behind the hip joint axis (Figure 14-9). The need for such a substitution begins with IC and continues throughout stance, ending in pre-swing. In anticipation, patients commonly assume the posture at IC. Patients with bilateral hip extensor weakness maintain a backward lean of the trunk during the entire stride.

The severity of lumbar lordosis associated with the backward lean is related to the degree of hip flexion contracture present (Figure 14-10). A flexed hip displaces the base of the spine (lumbosacral joint) anterior to the hip joints, thereby necessitating greater backward

lean of the thoracic trunk segment. The head maintains a vertical posture.

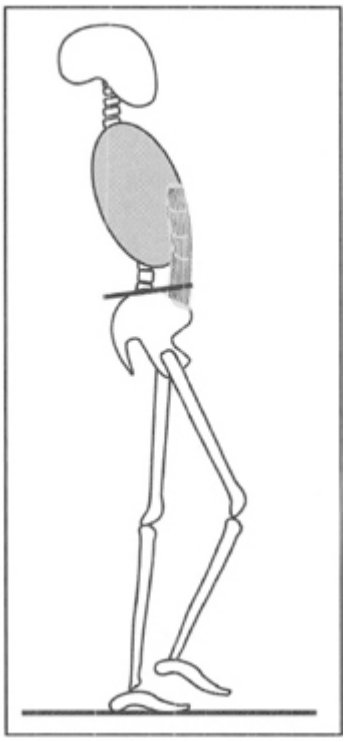

Figure 14-11. Backward trunk lean, increased abdominal muscle activity, and posterior tilt of the pelvis assist thigh advancement when the hip flexors are weak.

Inadequate Control of the Hip Flexors

During swing, a backward lean of the trunk may be used to assist limb advancement when hip flexor range or muscle control is inadequate. As upward rotation of the pelvis (posterior tilt) is a more direct substitution, using the trunk implies a need for further force to substitute for the inadequate hip function (Figure 14-11). The cause for involving the trunk could either be lumbar spine immobility or poor control of the abdominal muscles. In the latter instance, backward lean of the trunk uses the abdominal muscle mass as a dynamic

strap. Backward lean of the trunk in swing also can indicate contralateral hip extensor weakness.

Forward Lean

Definition: Anterior alignment of the shoulder girdle relative to the pelvic girdle

Phases: All phases

Functional Significance: Increases energy cost and demand on hip and trunk extensor muscles. In stance, a forward trunk lean may contribute to forward progression and improved knee stability.

Underlying Causes:

* Ankle PF (contracture/spasticity)
* Quadriceps weakness
* Hip extensor weakness
* Hip flexion contracture
* Use of assistive device (eg, walker)

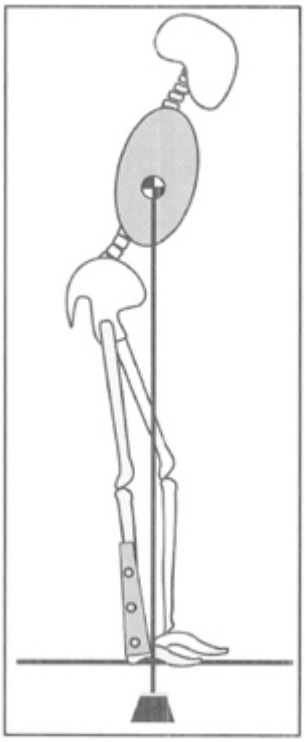

Figure 14-12. Forward trunk compensates for a rigid plantar flexion contracture by positioning the COG over the supporting foot.

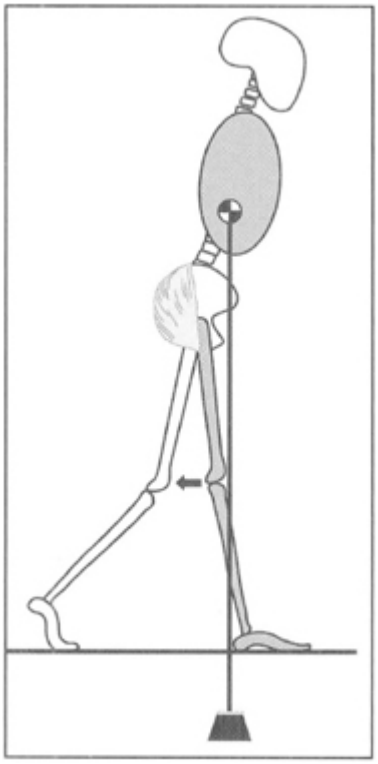

Figure 14-13. Forward trunk lean compensates for quadriceps weakness by moving the vector anterior to the knee if the ankle is stabilized.

Positioning the trunk forward of vertical moves the body vector anteriorly (Figure 14-12). The basic indications are to preserve weight-bearing balance and to stabilize the knee. Pathology at the ankle, knee, or hip most commonly creates the need for anterior displacement of the vector by forward lean of the trunk. Other possible causes are a lack of spine mobility or abdominal muscle weakness. The cause of forward trunk lean changes with the gait phase.

Excess Ankle Plantar Flexion

Persistent ankle PF into mid stance, terminal stance, or pre-swing combined with continued heel contact requires forward lean of the

trunk to place the body weight vector over the area of foot support (see Figure 14-12). The cause may be a contracture or spasticity of the soleus/gastrocnemius musculature.

Vastii Weakness

Insufficiency of the quadriceps at the knee stimulates a forward trunk lean as a compensatory alignment. This posture provides a passive knee extensor force by placing the body vector anterior to the knee (Figure 14-13). Forward lean begins in loading response and persists through the rest of stance until body weight is transferred to the other limb in pre-swing. This substitutive posture can be very subtle if quadriceps insufficiency is the only impairment. Forward trunk lean is increased if there is an associated knee flexion contracture.

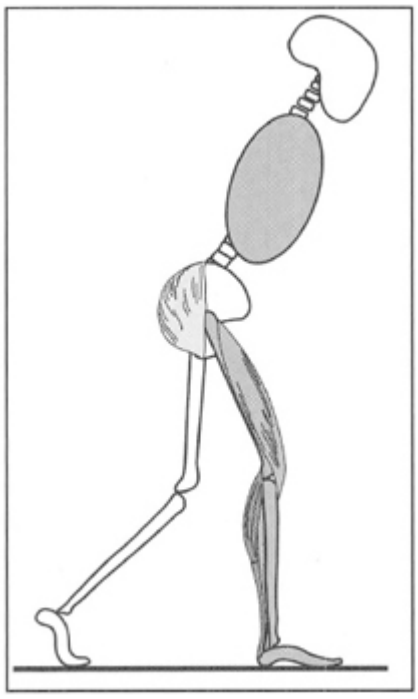

Figure 14-14. The trunk follows the anterior tilt of the pelvis when hip extensor strength is insufficient to meet the demands of loading response. Increased hip flexion improves the moment arm and force production of the hip extensors. Without the quadriceps and calf muscles to stabilize the limb, the leg will collapse.

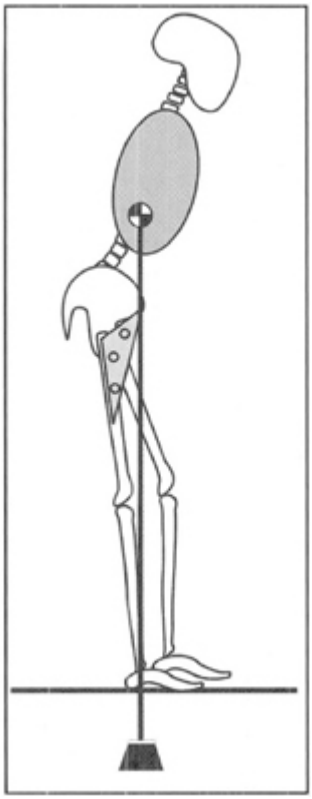

Figure 14-15. Forward trunk lean follows the anterior pelvic tilt created by a rigid hip flexion contracture. Ankle plantar flexion allows the COG to remain over the foot.

Hip Extensor Insufficiency

Hip extensor weakness of a moderate degree initiates forward trunk lean in loading response and early mid stance. As body weight is dropped onto the limb, the weak extensors allow the pelvis to fall forward and the trunk follows when either spine mobility or control is insufficient to provide compensatory lumbar lordosis. Postural lengthening of the hip extensor muscles provides the strength needed to stabilize this forward position (Figure 14-14). This represents an extensor lag of the hip muscles. An upper extremity assistive device may be required to support the trunk. As the limb advances beyond vertical in late mid stance, passive extension of the thigh removes the stimulus for a forward lean.

Hip Flexion Contracture

A hip flexion contracture without a compensatory lordosis results in a forward lean of the trunk during mid stance, terminal stance, and pre-swing (Figure 14-15). Of the 3 phases, terminal stance is the period most likely to be impacted by a hip flexion contracture as this is the phase when the thigh is normally most extended.

Assistive Device

Bearing weight through an upper extremity assistive device such as a walker or bilateral crutches can result in a forward trunk lean, particularly if the equipment is set too low.

Phasing of Forward Trunk Lean

Loading response has 2 causes for a forward trunk lean. The most common stimulus is quadriceps muscle inadequacy of any magnitude as this is the phase of highest demand. Deliberate reduction of quadriceps action to protect the anterior cruciate ligament represents a more subtle use of a forward trunk lean.

During mid stance and terminal stance, excess ankle PF becomes a third potential cause of forward trunk lean. Inhibition of tibial advancement necessitates forward trunk lean to progress body weight over the rigid ankle. With the onset of pre-swing, the transfer of body weight to the other limb terminates the mechanisms inducing forward trunk lean. Consequently, unless there are similar problems in the contralateral limb, the trunk resumes its upright posture.

CORONAL PLANE PELVIC DEVIATIONS

The trunk may passively fall or be actively displaced toward the same (ipsilateral) or opposite (contralateral) side. One also may refer

to the direction of trunk displacement as right and left, but this is not recommended as the functional significance is obscured. Lateral deviation of the trunk in either direction also may occur in either stance or swing, although most causes relate to standing stability.

Ipsilateral Trunk Lean

Definition: Movement of the trunk toward the reference (ipsilateral) limb

Phases: All phases

Functional Significance: During stance, ipsilateral trunk lean decreases demand on the limb's hip abductors; however, energy cost may be increased and forward momentum slowed. During swing, an ipsilateral trunk lean is detrimental to balance.

Underlying Causes:

* Weak ipsilateral hip abductors (stance)
* Ipsilateral hip adduction contracture (stance)
* Tight IT band (stance)
* Scoliosis
* Impaired body image (swing)

Moving the trunk toward the stance limb is a deliberate action. In contrast ipsilateral trunk lean in swing is incompatible with stability. Thus, it occurs only with the attempted first step. Lateral fall of the trunk toward the limb being lifted for swing represents failure to accommodate for the lack of limb support.

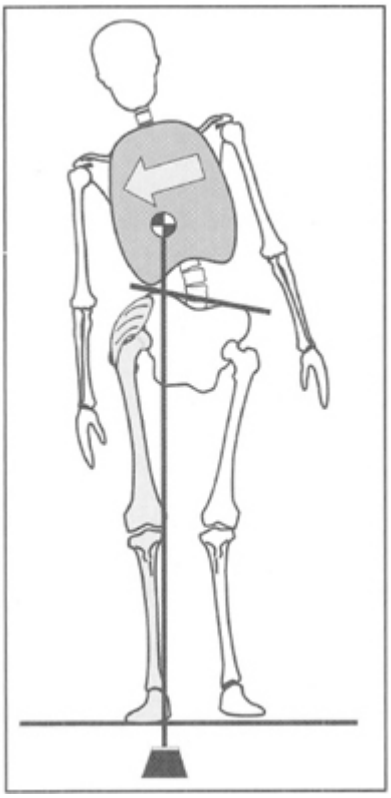

Figure 14-16. Ipsilateral trunk lean moves the COG closer to the stance limb and reduces the demand on weak hip abductors.

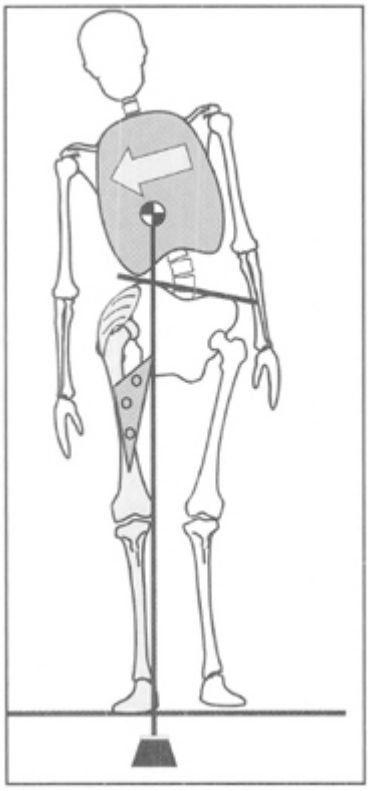

Figure 14-17. Ipsilateral trunk lean to preserve the COG alignment when a hip adduction contracture causes a contralateral pelvic drop.

Weak Hip Abductors

Shifting the trunk toward the supporting limb in stance is a useful substitute for inadequate hip abductors. This is a deliberate action that reduces the abductor moment required to stabilize the trunk mass (Figure 14-16). Body weight is moved toward the center of the hip joint. Substitutive ipsilateral trunk lean begins with IC and persists through terminal stance. The amount of lateral lean varies with the weakness of the hip musculature.

Contractures

Two types of contractures can cause ipsilateral trunk lean. Adductor muscle contracture has the same effect as weak hip abductors. By pulling the pelvis down, the tight adductors move the body's COG away from the stance limb. To correct this imbalance, the trunk leans toward the stance limb (Figure 14-17).

Contracture of the lateral hip structures, most commonly a tight IT band, moves the stance limb away from midline. Now the trunk leans to that side to place the COG closer to the area of support. As the IT band also has a flexor component, the resulting trunk posture tends to be a mixture of forward and lateral lean (Figure 14-18).

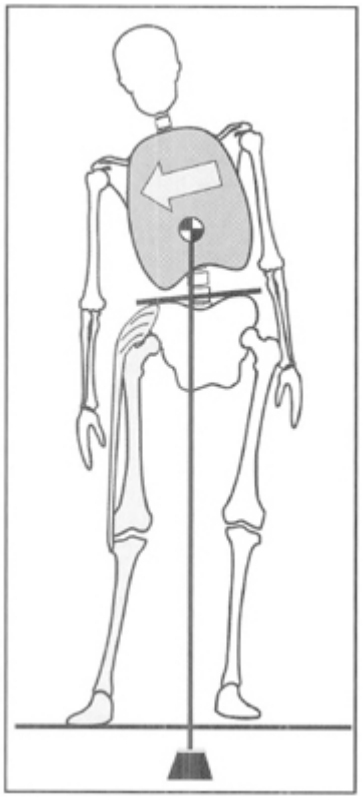

Figure 14-18. Ipsilateral trunk lean moves the COG closer to the area of support when a hip abduction contracture or tight IT band displaces the foot laterally (wide-based stance).

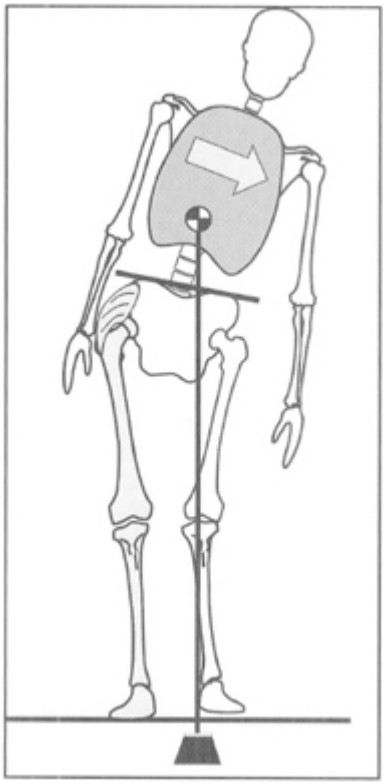

Figure 14-19. Patients with impaired body image fail to recognize that their swing limb is not supporting that side of the body. Thus, they fail to compensate with a lean of the trunk.

Scoliosis

Spinal curves that displace the upper trunk lateral to the midline create a static form of lateral trunk lean. The lean can be either ipsilateral or contralateral.

Impaired Body Image

In swing, fall of the trunk toward the limb being lifted can occur when the patient fails to recognize that half of his or her body is unsupported (Figure 14-19). Hemiplegic patients with an impaired body image may be unaware of the location of their body weight line. Having no sense of instability, they make no effort to shift their body weight over the stance limb. The resulting imbalance is incompatible with walking.

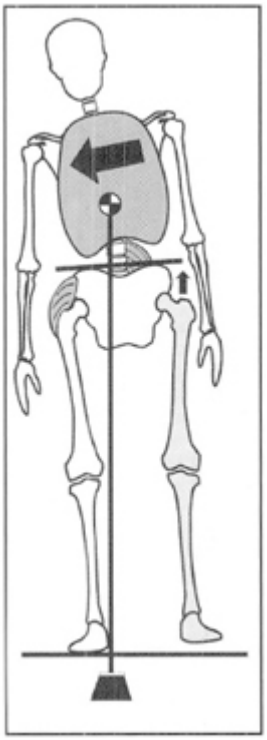

Figure 14-20. Contralateral trunk lean in swing as a voluntary act to assist pelvic hiking for foot clearance.

CONTRALATERAL TRUNK LEAN

Definition: Movement of the trunk toward the opposite limb

Phases: All phases

Functional Significance: During swing, a contralateral trunk lean may reduce demand on the opposite limb's hip abductors. During stance, a contralateral trunk lean is a posture of instability.

Underlying Causes:

* Maneuver to compensate for limited swing phase hip or knee flexion or ankle DF
* Contralateral hip abductor weakness (swing)
* Tight contralateral IT band (swing)
* Scoliosis
* Impaired body image (stance)

A contralateral trunk lean may arise from either an ipsilateral or contralateral problem. Any factor that has been identified as a cause of an ipsilateral trunk lean can result in a contralateral trunk lean if it affects the opposite limb. In stance, contralateral trunk lean represents failure to compensate for a situation that threatens balance. Contralateral trunk lean in swing is a deliberate substitutive action.

Compensation for Inadequate Hip Flexion, Knee Flexion, or Ankle Dorsiflexion During Swing

Inadequate hip flexion for swing stimulates leaning to the opposite side as a means of raising the foot to clear the floor (Figure 14-20). The cause can be either a lack of joint mobility or hip flexor muscle weakness. Both situations would cause the lateral trunk lean to begin in initial swing and persist through mid swing. Inadequate joint mobility also introduces abnormal trunk posturing in terminal stance, while hip flexor weakness does not require a substitutive action until the onset of swing.

TRANSVERSE PLANE TRUNK DEVIATIONS

Excessive and out-of-phase rotation of the trunk are identifiable gait errors but have little functional significance other than an increase in energy cost. Most commonly the deficit is dynamic. Rotatory scoliosis is a static cause of transverse plane malignant of the trunk.

EXCESSIVE TRUNK ROTATION

Definition: Rotation greater than neutral on the reference side

Phases: All phases

Functional Significance: Increased energy cost

Underlying Causes:

* Synergistic movement of the pelvis and trunk
* Use of an assistive device
* Arm swing synergy

Synergy With the Pelvis

The trunk may accompany the pelvis as it follows the swing limb rather than providing the normal counter balance in the opposite direction. This results in excessive (and out-of-phase) forward rotation of the ipsilateral side of the trunk during mid and terminal swing. If the response is bilateral, the trunk will move into excessive backward rotation by terminal stance (contralateral terminal swing).

Synergy With an Assistive Device

Trunk rotation may accompany the walking aid. In persons relying on a single walking aide, the trunk may follow the crutch or cane. The pattern of rotation varies depending on the location of the walking aid. When the support is ipsilateral, as for a painful knee, the cane (or crutch) follows the limb. This results in contrary phasing with excessive forward rotation at IC and peak backward rotation in pre-swing. Contralateral use of the walking aid, as is the practice with hip pathology, results in normal phasing of the rotation but with an increased range. Now at IC, the trunk displays excessive backward rotation. Then there is excessive forward rotation by terminal stance.

Arm Swing Synergy

Normal phasing of trunk rotation occurs but it can become excessive as exaggerated arm motion is used to assist balance. The

pattern of motion is similar to that of single contralateral cane use.

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Section IV

Clinical Considerations

Chapter 15

Examples of Pathologic Gait

The purpose of this fourth section is to provide a clinical bridge between the isolated components of walking and the management of pathology. While the unique attributes of each client's clinical picture prevent generalization of conclusions to all patients with a given diagnosis, the framework for interpreting gait abnormalities and informing clinical decision making can be widely applied.

As observation is the basic clinical technique for gait analysis, key gait patterns are described first by this technique. Then the relevant and available laboratory data are used to clarify and quantify the gait deviations seen. Motion has been recorded with a three-dimensional, automated multiple video camera system. Muscle action was documented using dynamic EMG and fine wire indwelling electrodes unless otherwise indicated.

DEFORMITY

The ability to walk can be significantly impaired by an acquired contracture or congenital deformity of the joints of the lower extremity that obstructs forward progression over the stance foot or clearance of the ground in swing. Patients who are neurologically intact have the ability to substitute by motions at other joints, as their position sense (proprioception) and selective control are intact. The substitutive effort, however, increases the energy cost of walking.

CONTRACTURES

Contractures are created by stiffening of the fibrous tissue encompassing the joint (ie, the capsule) or the fibrous sheathes within the muscles.[137] The cause may be either prolonged immobilization or scar formation as part of the healing process. Trauma accelerates the development of a contracture as both immobilization and fibrous tissue deposition are involved.[58]

The basic components of fibrous connective tissue are collagen fibers for strength and proteoglycan for mobility.[149] Collagen fibers, while highly flexible, have minimal ability to stretch. Thus, extension is determined by their resting length and changes in alignment. Both qualities are compromised by the lack of motion. Proteoglycan is a highly flexible gel that lubricates and separates the collagen fibers.[150] Its water content and chemical composition are dependent on the physiological effects of motion.[2-4,150] In 1951, Twitchell noted the onset of joint stiffness in the hemiparetic limb within 4 days following a stroke, well before there was any evidence of muscle tone.[138] The addition of operative scarring was found to cause irrecoverable loss of motion following surgical repair of elbow fractures immobilized more than 2 weeks.[56] Subtalar joint mobility was restricted in 50% of limbs with tibial shaft fractures treated by cast immobilization.[85] Experimental studies of rigid knee immobilization displayed measurable chemical changes in proteoglycan within 4 weeks.[1]

Differences in contracture density result in 2 functional patterns: elastic and rigid. While both display excessive resistance to manual examination, they differ in their response during the GC. Elastic contractures, arising from inactivity, often yield under the force of body weight. As a result, the impairment is less in terminal stance than in earlier phases. Contractures enhanced by the scarring of trauma or surgery are characteristically dense and rigid. Being able to resist body weight, the deformity persists throughout the GC. These functional differences are particularly evident at the ankle.

The primary functional impairment imposed by the loss of joint mobility from contracture is restraint of progression. During stance, patients either are delayed or inhibited from advancing over the supporting foot. In swing, inhibition of floor clearance or reach is the

deterrent to normal progression. When patients are neurologically intact, as is true for the examples selected, then they will know the joint's exact position (ie, proprioception is intact) and will have the selective control to substitute.

Ankle Plantar Flexion Contracture

A common cause of an ankle contracture is plaster cast immobilization of fractures involving the shank, ankle, or hindfoot. Regardless of the level of injury, the cast is molded snugly around the ankle and foot. This impedes motion of both the subtalar joint and ankle. The fibrous tissue within the plantar flexor muscles and/or the capsule of the ankle and subtalar joint become unacceptably stiff in patients with an injury requiring more than 6 weeks of nonweight-bearing immobilization for healing (R. Watkins, oral communication, 1978). In addition, each week of inactivity allows the muscles to progressively weaken.[74]

Unless special care is taken, the ankle's position will be 15° of PF because that is the joint's natural resting position.[38] To achieve a trailing limb posture and full step length, 10° DF is used in terminal stance to advance body weight to the anterior portion of the MTP joint. The difference between normal mobility and a 15° PF contracture is a 25° functional deformity.

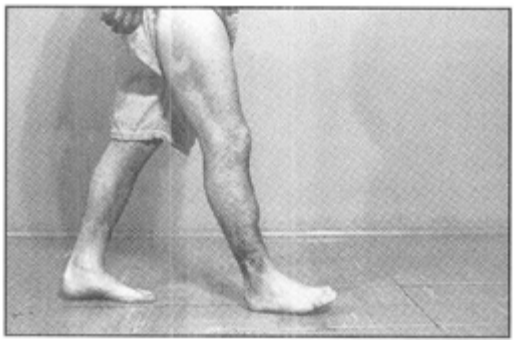

Figure 15-1. Patient A has an ankle PF contracture. (A) IC with low heel strike.

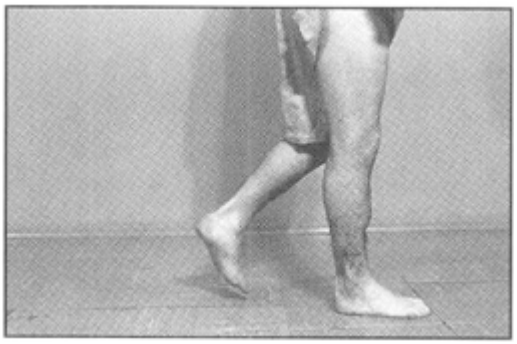

Figure 15-1. (B) Mid stance posture showing excessive ankle PF.

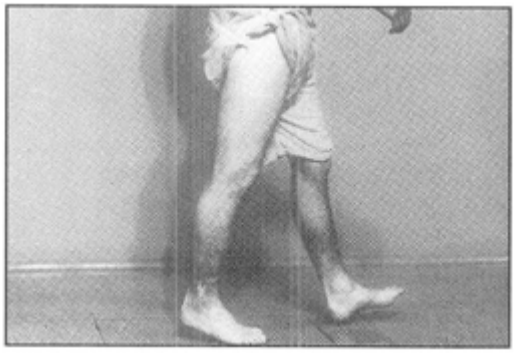

Figure 15-1. (C) Terminal stance displays excessive heel rise.

Patient A: Ankle Plantar Flexion Contracture (Figure 15-1)

This man was a healthy, vigorous construction worker before his injury. His diagnosis was delayed healing of a complicated tibial shaft fracture that necessitated prolonged nonweight-bearing immobilization in a cast. Physical findings included rigid contractures at the ankle (15° PF) and subtalar (neutral) joints. Grossly, his gait appeared normal but a bit slow. In addition, he could not run and complained of forefoot pain.

IC was made with a “low heel strike.” Despite the 15° of PF, the heel was the first portion of the foot to reach the floor because the foot had been sufficiently tilted upward by the normal 25° hip flexion and full knee extension (Figure 15-1A). However, the short distance between the forefoot and the floor led to premature floor contact by the forefoot and early foot flat weight bearing. Without close observation, this deviation would have been easily overlooked. The

abbreviated heel rocker could barely stimulate any knee flexion for loading response shock absorption.

At the onset of mid stance, the tibia was angled more posterior than normal and tibial advancement was inhibited by the lack of ankle rocker mobility (Figure 15-1B). Advancement of the body mass over the forefoot had to function against a longer moment arm as the ankle did not yield into the normal 5° of DF. The added effort would increase metatarsal head pressure against the floor. Guarding of the forefoot from excessive pressure was implied by the plantar flexed position of the clawed toes. In terminal stance, the excessive heel rise (Figure 15-1C) showed that this patient had the ability to roll onto his forefoot because he was healthy, strong, and vigorous. Rolling onto the forefoot without the usual ankle DF range required considerable propulsive energy by the trunk and hip extensors. The high heel rise, while not a conspicuous deviation, was indicative of great metatarsal head strain from prolonged weight bearing and obstructed progression.

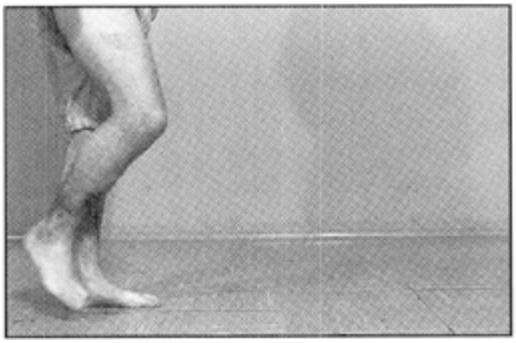

Figure 15-1. (D) Initial swing limb alignment obscures the excessive PF.

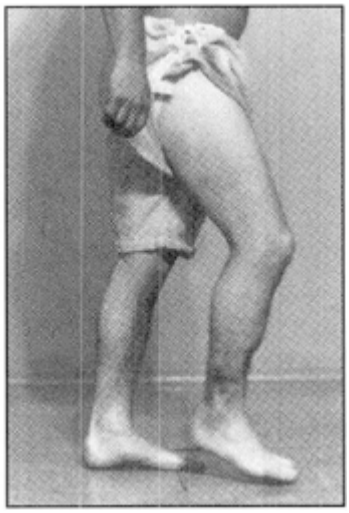

Figure 15-1. (E) Mid swing use of excessive hip and knee flexion to avoid toe drag from excess PF.

The natural toe-down (equinus) posture of the foot in pre-swing and initial swing masked the patient's contracture (Figure 15-1D). The potential delay in knee flexion was overcome by selective action of the hip and knee flexor muscles. In mid swing, however, the vertical tibia exposed his fixed ankle PF contracture (Figure 15-1E). Toe drag was avoided by the substitution of excessive hip and knee flexion to lift the plantar flexed foot higher than normal. This conspicuous mid swing gait deviation, however, usually has little functional significance, as increasing hip flexion is an easy task for most patients.

Thus, this rigid 15° PF contracture introduced 4 major functional penalties related to premature loading of the forefoot. First, early forefoot contact minimized the heel rocker stimulus for knee flexion needed for shock absorption. Premature loading of the forefoot increased the duration of pressure on the metatarsal heads with corresponding tissue strain. Loss of the ankle rocker delayed advancement of the tibia until the vector arrived at the forefoot and required the weight of the body to be lifted as it rotated forward over the forefoot rocker. Lastly, the tibial lag created a hyperextension torque at the knee that locked the joint. To permit swing, the limb had to be unloaded during the double support period of pre-swing. As running does not have a double support period, this vigorous man

could not run and was impaired by the excessive forefoot weight bearing and pressure.

In conclusion, the only conspicuous gait deviation, mid swing ankle PF, had minimal functional significance, as increasing hip flexion is an easy task for most patients. Conversely, the patient's disabling gait deviations were such subtle stance phase events that they were identifiable only by quantified biomechanical analysis.

Hip Flexion (Burns)

Contractures at the hip severely limit forward progression even when neurological control is normal. A secondary reaction is the development of knee flexion contractures due to the constant posturing of the femur. Step length is significantly shortened by the inability to extend the limbs in terminal stance and terminal swing. Weight-bearing muscular effort also is increased.

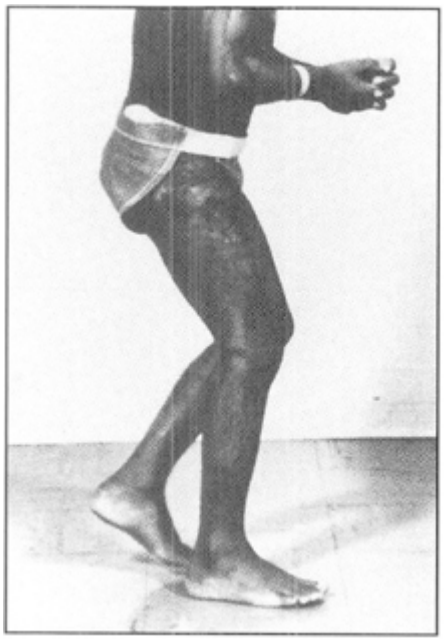

Figure 15-2. Patient B has hip flexion contractures secondary to burns. (A) Hip and knee flexion deformities have placed the pelvis behind the supporting foot as excessive ankle DF is not available.

Figure 15-2. (B) Trailing limb posture is lost, leading to markedly shortened step length.

Patient B: Hip Flexion Contractures Secondary to Burns (Figure 15-2)

Fibrous rigidity resulted from the lengthy immobilization needed to regain adequate skin coverage following burns to both lower extremities. The 30° flexion contractures at the knees and hips reflected the natural resting positions of these joints (see Figures 10-6 and 10-7).[38] Ankle DF to approximately 5° had been preserved.

Advancement of the body over the foot in mid stance was severely restricted. While the tibia reached a vertical position, fixed knee flexion restrained the thigh. To accommodate the excessive knee flexion, the ankle would need to excessively dorsiflex. As this mobility was lacking, the junction between the pelvis and thigh remained behind the supporting foot (Figure 15-2A). Excessive hip flexion and forward lean of the trunk were required to place the body vector over the supporting foot for weight-bearing balance. The flexed knee posture also increased the demand on the quadriceps.

Inability to extend the hip and knee in terminal stance prevented effective advancement of the body weight ahead of the supporting foot. The flexed hip did not allow the limb to assume a trailing position and step length was severely shortened (Figure 15-2B). In

terminal swing, the patient lacked knee extension for significant forward reach of the limb.

Because the patient had a bilateral disability, he had minimal ability to substitute. Shortened stride lengths necessitated a corresponding increase in step rate to accomplish the desired walking distance.

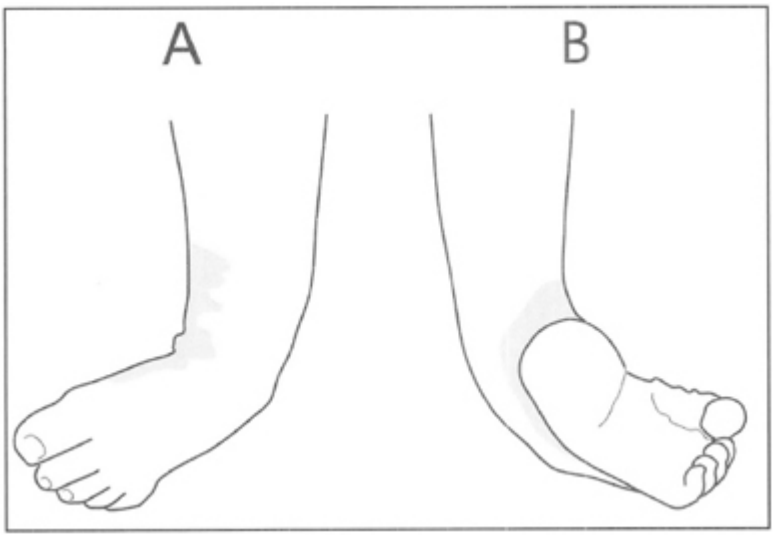

Figure 15-3. Typical deformities of clubfoot include (A) adduction and inversion of forefoot and (B) ankle equinus and midfoot cavus. (Adapted from Ponseti I, Smoley E. Congenital club foot: The results of treatment. *J Bone Joint Surg Am*. 1963;45-A(2):261-275, 344.)

STRUCTURAL DEFORMITY: CLUBFOOT

The congenital clubfoot, first described by Hippocrates (400 BC), is an ancient deformity that continues to appear today.[119] At birth, the newborn's deformed foot is twisted in all three planes (adduction, inversion, and equinus; Figure 15-3).[70,111,119] Historically, the method of correction has varied with the severity of the malformation and the techniques available.

The first therapeutic system was the gentle stretching and protective bandaging described in the writings of Hippocrates.[119] Information was scant during subsequent centuries. Museum displays, such as Pare's boot (1564) and special wrenches from the

19th century, indicate that more forceful procedures had been adopted in the hope of more rapid improvement.[119] Simple surgery, such as an Achilles tendon lengthening to reduce equinus, was introduced in the late 18th century.[119] The discovery of anesthesia by Morton allowed more forceful manipulations and a wide variety of operative procedures.[119] While the appearance of the foot generally was improved, stiffness of the foot became a significant problem.[17] In 1939, Kite appealed for the return to gentle manipulation and stressed that there was an essential order of corrective maneuvers (ie, correction of forefoot adduction must precede attention to the equinus).[70] Ponseti identified a compromise that shortened the therapeutic program.[111,112] Beginning with an essential series of manipulations and long-leg casts (average 7.6) over 5 to 12 weeks, most cases (79%) had an Achilles tenotomy to correct the residual equinus. Occasionally, lateral transfer of the TA was added to correct residual inversion.[113] Then Denis Browne splints[133] were worn for approximately 24 months to prevent recurrence. Success in achieving a normal-appearing, functional, painless foot was reported as 90%.[111,112]

Significance of Stretch Force

"Gentle manipulation" has been substantiated by both clinical and scientific criteria. Ligaments and tendons are formed by multiple, strong collagen fibers supported by a proteoglycan matrix.[141,149] The viscoelastic quality of ligaments or tendons can be graphed as a stress-strain curve (Figure 15-4).[149] At the onset of the stretch (called the "toe"), the curve is shallow and the strain (length) increases faster than the stress (force). Once the collagen molecules are stable, the strain curve becomes linear. The slope of the linear region identifies the elastic stiffness of the tissue. When the increased tension starts to exceed the strength of the fibers, the linear pattern begins to fragment and at the point of failure, tension drops. Clinical experience has demonstrated that the tension in the toe region of the stress-strain curve is the therapeutic level of

stretch. The clinical rules are to stretch the tissue until it is taut and then yield about 5° before setting the hold position of the cast. Under this level of tension, the taut fibers “creep” (ie, the viscous molecules yield) and the tissue lengthens (Figure 15-5).[141] An attempt to sustain the stretch at higher tension levels caused swelling, stiffness, and failure to lengthen.

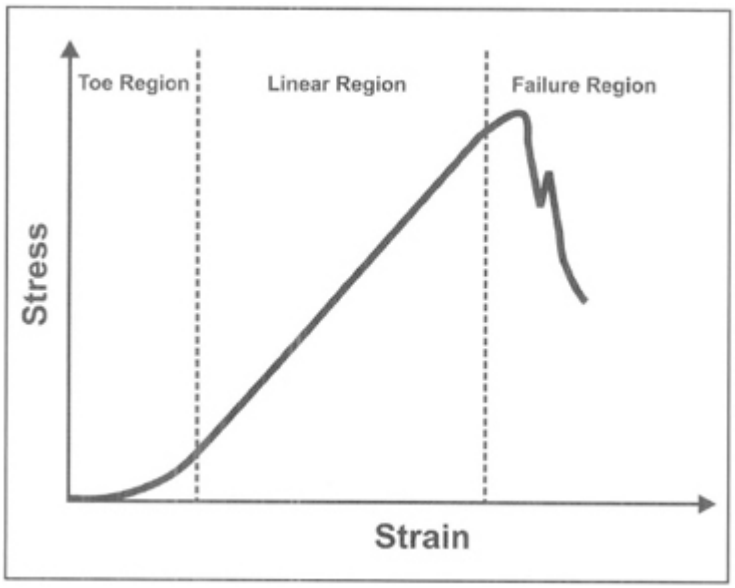

Figure 15-4. Stress-strain curve demonstrating nonlinear re-sponse to a stretching force of a ligament or tendon. (Adapted from Woo SL-Y, An K-N, Frank C, et al. Anatomy, Biology and Biomechanics of Tendon and Ligament. In: Buckwalter J, Einhorn T, Simon S, eds. *Orthopaedic Basic Science; Biology and Biomechanics of the Musculoskeletal System*. 2nd ed. American Academy of Orthopaedic Surgeons; 2000:581-616.)

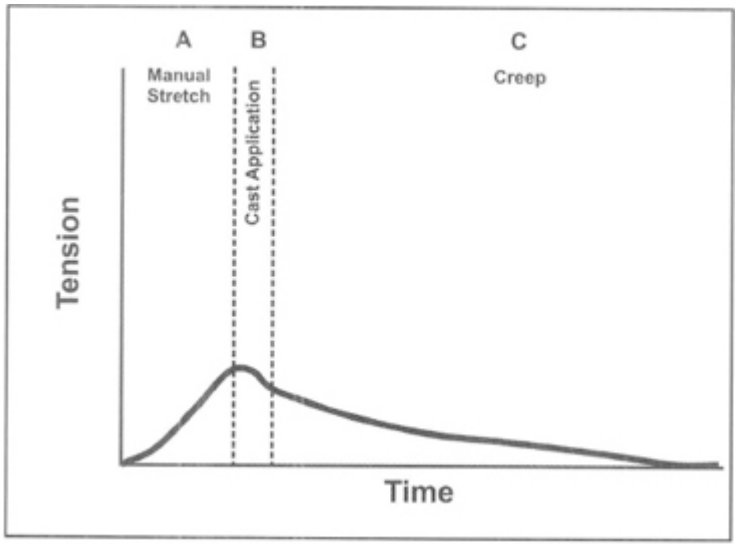

Figure 15-5. Creep response to sustained low-level stretch force during casting. (A) Manual stretch until joint is taut. (B) Reduction in tension by allowing joint to yield 5°. (C) Collagen fibers creep in response to sustained low-level stretch. (Adapted from Woo SL-Y, An K-N, Frank C, et al. Anatomy, Biology and Biomechanics of Tendon and Ligament. In: Buck-walter J, Einhorn T, Simon S, eds. *Orthopaedic Basic Science; Biology and Biomechanics of the Musculoskeletal System*. 2nd ed. American Academy of Orthopaedic Surgeons; 2000:581-616.)

Surgical Management of Clubfoot

Anatomical dissections of clubfeet in stillborn infants identified severe tendon displacement and medial displacement of the anterior head of the talus.[40,57,63,111,112,128] Muscle insertions remained intact and evidence of paralytic episodes was rare.[40,57,63,128]

Surgical correction of equinus by an Achilles tendon lengthening was the most frequent recommendation. To also reduce adduction or inversion, the choices of procedures were posterior, posterior-medial, and comprehensive releases. Displacement of foot structures, however, could blur the normal planes between the 4 plantar layers of muscles and tendons. Postoperative stiffness has been a common complication.[8,36,52,113]

Gait Analysis in the Management of Clubfoot

Recent replacement of the historical descriptive grades, such as excellent, good, or fair, by instrumented gait analysis has allowed quantified comparison of different approaches. A 2008 comparison of the Ponseti approach (gentle manipulations, serial casting, and limited surgical intervention) versus a French program of more intense stretching and a home follow-up program[36] identified no differences between gait speed, stride length, and cadence. Mild differences, however, in kinematic parameters were identified. Excess DF (ie, >15°) was present in 48% of the Ponseti clients compared to 12% in the physical therapy program. Additionally, this deviation was more frequent following an Achilles tenotomy. Equinus gait was more common following the physical therapy versus the Ponseti intervention (15% versus 1%, respectively). Foot drop (ie, >9° PF during the final quarter of swing) also dominated in the physical therapy program (19% versus 4%). Instrumented gait analysis can help refine decision making related to surgical interventions.

Patient C: Child with Bilateral Clubfeet

A 4.5-year-old boy with bilateral clubfeet and marked residual deformity following previous bilateral clubfoot release surgery at age 1 year was evaluated clinically (Tables 15-1, 15-2, and 15-3) and by observational and instrumented gait analysis. The observed abnormalities were cavo-varus feet during both stance and swing with marked adduction of the forefeet and high longitudinal arches, which were essentially nonweight bearing under the medial foot column.

Table 15-1

Selective Manual Muscle Testing Grades (5-Point Scale) of Child With Bilateral Clubfoot Deformities

Muscle Group	*Strength*	
	Left	Right
Hip flexion	4	4
Hip extension	4	>3
Hip abduction	4	>4
Knee flexion	4	4
Knee extension	4+	4+
DF	4+	4+
PF	4-	>3
Ankle inversion	4	4-
Ankle eversion	4	>4

Table 15-2

Select Passive Range of Motion Findings of Child With Bilateral Clubfoot Deformities

Muscle Group	*Range of Motion*	
	Left	Right
Knee extension	20° hyperextension	20° hyperextension
DF, knee flexed	5° inverted 5° neutral	5° inverted 5° neutral
DF, knee extended	0° inverted 0° neutral	0° inverted 0° neutral
PF	15°	15°
Forefoot inversion	40°	40°
Forefoot eversion	0°	0°
Femoral anteversion	42°	45°
Transmalleolar axis	0°	0°
Hindfoot-thigh axis	5° internal	5° internal
Thigh-foot axis	35° internal	30° internal

Table 15-3

Stride Characteristics of Child With Bilateral Clubfoot Deformities

Variable	
Velocity (m/min)	59 (91% N)
Cadence	177 (115% N)
Step length (m)	Left = 0.34 (56% N) Right = 0.32 (52% N)
Double limb stance (% GC)	24 (120% N)
Single limb stance (% GC)	Left = 37 (93% N) Right = 39 (98% N)

During gait, excessive intoeing (Figure 15-6A) was compensated for by excess external hip rotation (Figure 15-6B). Increased hip abduction in swing was necessary for foot clearance (Figure 15-6C). Five of the functional factors related to over correcting the initial equinus. The onset of calf muscle activity was delayed until terminal stance on the left and mid stance on the right. This was consistent with the excessive DF documented for the left ankle and the bilateral delay in heel rise. The resulting shortened step length (~55% normal) was accommodated by a fast cadence (115% normal). Other signs were indicative of a lack of forefoot correction. These included excessive forefoot inversion, an obvious deviation. Premature activation of the anterior tibialis contributed to the inversion force. Premature and prolonged activity of the peroneus longus, which normally depresses the first metatarsal, was also documented. The presence of good to fair hip and knee strength preserved an effective velocity, but inverted feet introduced a compensatory leg alignment.

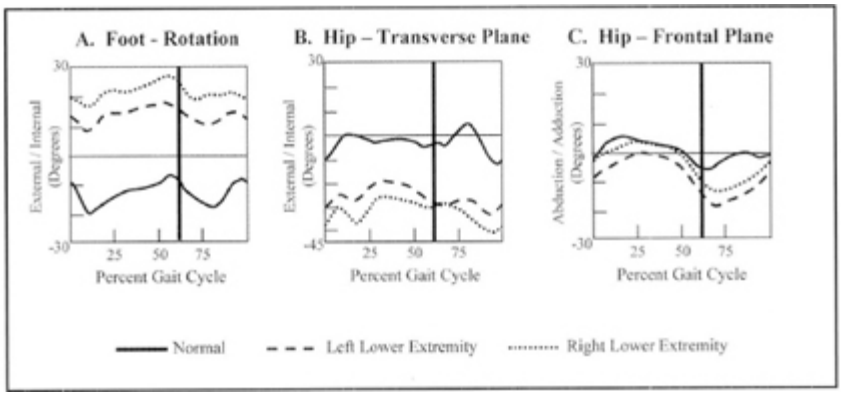

Figure 15-6. Patient C has bilateral clubfoot deformity. Motion analysis revealed (A) excessive intoeing during stance and swing, (B) external hip rotation during cycle, and (C) hip abduction during swing to assist foot clearance. (Data provided courtesy of Children's Hospital, Los Angeles.)

It was apparent that the initial surgeon chose to approach primarily the conspicuous equinus or perhaps was unfamiliar with the interdependence of the clubfoot deformities. The proposed corrective surgical plan appeared appropriate for the deviations.

SUMMARY

Acquired contractures and congenital deformities can substantially compromise walking. Disruptions to stance stability, forward progression, or limb clearance slow gait and increase energy expenditure. While therapeutic interventions, such as stretching and surgery, are frequently used to restore function, care should be exercised.

WEAKNESS

The functional significance of weakness depends on the area and extent of muscle involvement and the patient's ability to substitute. The ability to substitute is dependent on the balance of muscle strength within the limb and between the 2 limbs. Thus, bilateral impairment significantly reduces the patient's function. Muscle weakness also can lead to deformity.

QUADRICEPS INSUFFICIENCY (POLIOMYELITIS)

The quadriceps muscle is essential for shock absorption, weight-bearing stability and forward progression during gait. Quadriceps

function can be lost by direct trauma to the muscle, femoral nerve damage or various paralytic diseases. Poliomyelitis was selected as the example of muscle weakness because the disability caused by the polio virus is solely a motor deficit. Patients compensate for the abrupt reduction in muscle function so well that observers commonly fail to identify a disability.

Acute Poliomyelitis

During the first half of the 20th century, poliomyelitis epidemics of increasing severity were a major cause of quadriceps dysfunction. The polio virus paralyzed the muscle by invading lower motor neurons cells located in the spinal cord.[15] The resulting disability often was severe, yet polio survivors with complete quadriceps paralysis were able to walk with a stable knee by selectively substituting action of other extensor muscles. The skill of developing an alternate source of knee extension control was possible because all the components of normal muscle control, except a connection to the motor units of muscle fibers, were intact.[5]

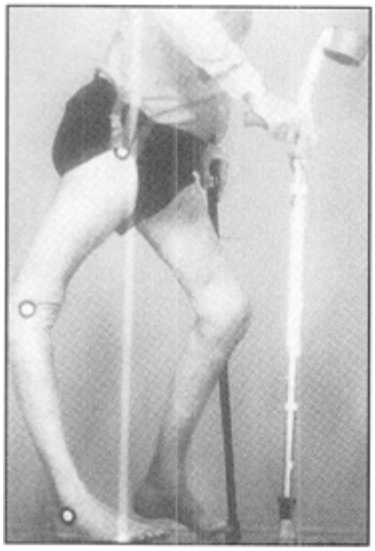

Figure 15-7. Knee hyperextension stabilizes the stance limb for a patient with quadriceps paralysis arising from poliomyelitis.

As the muscles affected by the acute poliomyelitis virus began to recover, walking was made possible by the use of a knee locking brace (knee-ankle-foot orthosis, KAFO) to protect and substitute for the weakened quadriceps muscle.[104] By the time children became teenagers, many opted to discard their braces to feel and look more normal. They had learned to substitute subtle postural adaptations at the ankle and hip to prevent the knee from becoming unstable during stance. These techniques often realigned the knee axis posterior to sagittal body vector, imposing a stretching force on the posterior knee structures with each step. Children's ligaments were particularly sensitive to stretch during their growth period. For some, the excessive forces over time led to severe knee hyperextension, pain and increased disability as adults (Figure 15-7). Attempts to surgically repair overstretched ligaments at the knee failed because standing stability still required 10° to 15° of knee hyperextension and this position put the repair at risk.[107]

Introduction of the Salk vaccine in 1955 was very effective at preventing new cases of acute poliomyelitis in all but 4 Asian countries.[67] However, the residual polio impairment persisted. Today, polio survivors are experiencing additional disability from the late effects of stressing muscles impaired by polio.[53,55,71,104,106]

The Late Effects of Poliomyelitis

Approximately 25 years after the acute episode, polio-survivors who had considered themselves cured, began seeking medical care for their fatigue, new muscle weakness and pain.[53,55,71,104,106] Initially this was assumed to be a return of the polio virus. Later it was related to a failure of enlarged motor units that had initially formed to reinnervate and "heal" paretic muscles.[54,84] The new lower motor neuron cells nourished as many as four times their normal number of muscle fibers, leading to apparent functional failure due to overuse.[130] Two patterns of dysfunction were identified.[79,84,136]

The post-polio syndrome identified a complex sequence of pathology. It began with severe acute paralysis and was followed by an excellent recovery of function. Some survivors participated in

professional sports. Then years later the patients developed a rapid loss of function. These patients were reluctant to consider overload damage as the cause as there was a strong belief that exercise had cured the acute paralysis. The overuse theory was rejected despite scientific confirmation.[130]

Post-polio sequela was the term for the other patients with a mild to moderate acute course and a residual weakness involving just a few muscles. These polio survivors not infrequently displayed deformities developed during the acute illness. Patients in all groups who initially recovered borderline quadriceps strength often reverted to using hyperextension for later knee stability.

Patient D: Post-Polio (Figure 15-8)

The gait ability of patients with late post-polio often depended on their ability to substitute for weakened quadriceps.[101] If their ankle plantar flexor muscles were still graded 4 (good), patients were able to use the hyperextension thrust as a substitute for quadriceps weakness. The “low heel strike” (angle between the foot and floor) reduced the entire heel rocker (Figure 15-8A). Loading response lacked tibial advancement. Thigh retraction prevented knee flexion (Figure 15-8B). Anterior joint pain was sometimes an added disability. Knee extension was maintained through mid stance (Figure 15-8C). The knee was sufficiently locked by passive alignment for the patient to tolerate a heel rise at the end of terminal stance (Figure 15-8D). Momentum from the velocity of the opposite limb rolled the body weight onto the forefoot despite the lack of DF beyond neutral. The resulting forefoot rocker action in terminal stance preserved significant stride length; however, pre-swing knee flexion was limited (Figure 15-8E). Near-normal knee flexion was achieved in initial swing, but thigh advancement at the hip was limited (Figure 15-8F).

The substitutive motions at the ankle were confirmed by the electrogoniometer recordings (Figure 15-8G). Initiating 10° ankle PF at the onset of terminal swing prepared the limb for stance. DF for progression in mid and terminal stance occurred slowly, reaching

neutral in the middle of single stance. Maximum ankle position in terminal stance was less than 5° DF. The minor amount of hyperextension (2° to 3°) during loading response indicated inadequate dynamic stability.

The footswitch record displayed a normal floor contact sequence, although the timing of each support mode varied among the steps. This inconsistency implied some joint control insecurity.

Dynamic EMG recordings confirmed that the patient accomplished this effective gait without relying on her easily fatigued quadriceps (Figure 15-8H). Preparation for stance began with premature gluteus maximus activity in terminal swing to retract the femur so tibial momentum could assist knee extension. During WA, continuing gluteus maximus action stabilized the knee. Simultaneously, premature and intense soleus action restrained the tibia and became the primary source of knee extensor stability as its action continued through terminal stance. Knee flexion for initial swing began with a strong burst of semimembranosus action. This timing indicated that direct knee flexion for toe clearance was more critical than the loss of step length, which would arise from the hip extension action of this 2-joint muscle. In mid swing, the need to start extending the knee necessitated semimembranosus cessation. The hamstrings began hip deceleration in preparation for stance.

Hip Abductor Weakness

Gluteus medius muscle weakness occurs in many types of pathology. Ipsilateral trunk lean in the frontal plane stabilizes the hip. The mobility developed by patients who experienced acute poliomyelitis in childhood allows the greatest freedom for substitution. Complete hip abductor muscle paralysis can be fully accommodated by shifting the trunk laterally until the body vector lies over the hip joint (Figure 15-9). A secondary effect of this posture is valgus of the knee as the displaced weight line is lateral to the knee.

Figure 15-8. Patient D has quadriceps weakness secondary to post polio. (A) IC with a low heel strike.

Figure 15-8. (B) Loading response with mild knee hyperextension.

Figure 15-8. (C) Mid stance with mild knee hyperextension.

Figure 15-8. (D) Terminal stance DF and heel rise are limited.

Figure 15-8. (E) Pre-swing lacking knee flexion.

Figure 15-8. (F) Initial swing with normal knee flexion but limited hip flexion.

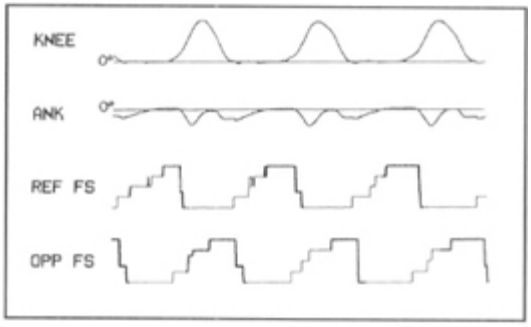

Figure 15-8. (G) Electrogoniometric recording of knee and ankle. ANK = ankle; REF FS = footswitch trace for the reference limb; OPP FS = footswitch trace of opposite limb; baseline = swing; elevated segments = stance. Height of footswitch steps indicates area of contact, length of step identifies duration of that mode of contact.

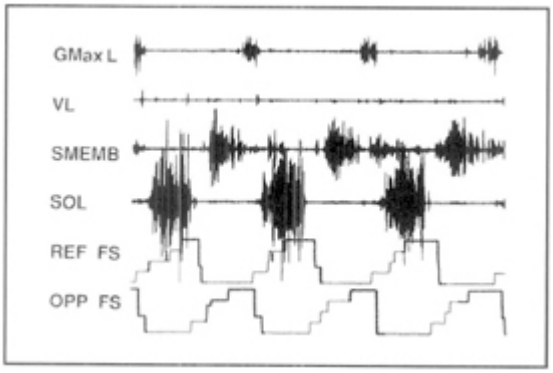

Figure 15-8. (H) Dynamic EMG (raw data). GMax L = gluteus maximus, lower; VL = vastus lateralis; SMEMB = semimembranosus; SOL = soleus.

Figure 15-9. Lateral trunk lean compensates for hip abductor paralysis in a patient with poliomyelitis. The body vector (vertical line) passes through the hip joint.

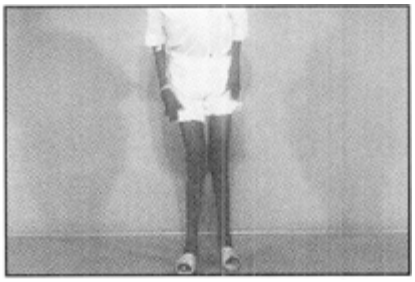

Figure 15-10. Lateral trunk lean compensates for gluteus medius inadequacy arising from rheumatoid arthritis.

Adult-onset hip abductor inhibition, such as occurs with rheumatoid arthritis, allows a lesser degree of lateral trunk lean. The knee experiences a smaller valgus thrust (Figure 15-10).

Soleus and Hip Extensor Weakness (Myelodysplasia)

As a congenital form of spinal cord injury, myelodysplastic paralysis is complicated by sensory loss in addition to the muscle weakness. A high potential for contractures is another complication of the disability. Myelodysplasia also subjects the joints to abnormal weight-bearing postures from the onset of walking. These multiple factors significantly reduce the patient's ability to substitute.

The severity of the gait deviations increases with each higher level of paralysis. Sequentially, impaired control of the limb involves the foot musculature, hip extensors, knee extensors (quadriceps), and hip flexors.

Patient E: Myelodysplasia (Figure 15-11)

This patient had a low lumbar lesion (L4 intact), and thus retained strong quadriceps for knee stability. However, walking function was seriously compromised by the lack of ankle plantar flexor, hip extensor, and hip abductor musculature. IC was made with a flexed knee and flat foot for several biomechanical reasons (Figure 15-11A). Hip flexion was excessive due to the inability of weak (or absent) hamstrings to adequately restrain the thigh in terminal swing. Lack of a forefoot rocker on the other limb led to inadequate advancement of the body mass. It was necessary to keep the foot within a weight-accepting range. To meet those limitations, the patient voluntarily relied on his strong quadriceps. The lack of dorsiflexor musculature allowed the foot to drop into mild PF. Hence, preparation for WA was compromised.

Loading the limb increased both knee and hip flexion while ankle PF only slightly decreased (Figure 15-11B). This limb posture represented slight collapse because the paralyzed gluteus maximus could not stabilize the femur. Any substitutive hip extensor action by the hamstrings merely added to the knee flexion. The markedly

flexed posture at the knee presented a very strong demand on the quadriceps to preserve weight-bearing stability.

Figure 15-11. Patient E has soleus and hip extensor weakness due to L4 myelodysplasia. (A) IC by the forefoot with excessive knee and hip flexion.

Figure 15-11. (B) Loading response showing excess hip and knee flexion, the ankle PF.

Figure 15-11. (C) Mid stance has excessive ankle DF and continued knee and hip flexion.

Figure 15-11. (D) Terminal stance displays extreme DF and additional knee flexion. The heel remains on the ground.

Figure 15-11. (E) Pre-swing has continued excess DF and the heel fails to rise. Excess backward pelvic rotation in the previous phase allowed for a greater step length.

Figure 15-11. (F) Mid swing requires excessive hip flexion to lift the plantar flexed foot to clear the floor.

Mid stance advancement of the body onto the supporting limb led to excessive ankle DF and persistent knee flexion (Figure 15-11C). The lack of tibial control at the ankle moved the knee joint more anterior to the body vector, increasing the demand on the quadriceps. The patient could not use a forward lean to reduce the

strain as the lean would have increased both the DF demand at the ankle and introduced an extensor moment at the hip and spine. Increased action by the quadriceps was the only source of knee stability because it was the sole weight-bearing muscle group. Terminal stance involved further advancement of body weight over the foot while the trunk remained erect to provide passive hip extensor stability (Figure 15-11D). Ankle DF and knee flexion were further increased. Stride length was gained by using total body rotation to add pelvic width to the step length between the 2 feet (Figure 15-11E). During pre-swing, heel contact continued until the limb was lifted for swing. In mid swing, excessive hip flexion was required to accommodate both the drop foot and the lower pelvic position created by the contralateral knee flexion (Figure 15-11F).

Tibialis Anterior Weakness (Spinal Cord Injury, Cauda Equina)

Fractures within the lumbar vertebrae are below the body of the spinal cord, which usually ends at L1. Beyond this level, the lumbar and sacral nerve roots, called the *cauda equina*, descend in the spinal canal until they reach their exit site. Injuries to these roots cause lower motor neuron (flaccid) paralysis. The functional impairment varies among patients according to the completeness of root damage.

Cauda equina differs from poliomyelitis by the addition of impaired proprioception. Often the residual sensation does not correlate with the level of motor impairment as the anterior location of the vertebral fracture may spare the posterior rami.

Patient F: Cauda Equina Spinal Cord Injury (Figure 15-12)

A mid lumbar neurological lesion caused complete paralysis of the TA and other ankle muscles as well as the knee flexors, hip

extensors, and abductors. The quadriceps muscle group was graded 3+ for strength. Sensation was intact at the knees but impaired at the feet.

Terminal swing preparation of the limb for stance was incomplete because there was marked ankle PF (Figure 15-12A). IC occurred with the forefoot. Tibial advancement (DF) in mid and terminal stance revealed the passive nature of the terminal swing equinus (ie, TA paralysis rather than a contracture; Figure 15-12B). There was, however, a useful degree of ankle PF contracture to provide weight-bearing limb stability by slight knee hyperextension. Passive tension of the contracture restrained the tibia at 5° DF while the femur rolled forward and hyperextended the knee. Alignment of the body weight line (trunk center to midfoot) provided an external extensor torque to supplement the weak quadriceps. Continuous weight bearing through the arms substituted for the paralyzed hip extensor and abductor muscles. A functional compromise between progression and stability was gained by the PF contracture stretching to only 5° DF with the advancement of the body vector.

Pre-swing preparation of the limb for swing was a deliberate process that included transferring a major portion of body weight onto the crutches through the arms (Figure 15-12C). Elevation of the shoulders was a sign of intense weight bearing. Passive pre-swing knee flexion was absent. Initial swing was delayed by a passive drop foot (absent TA) and the lack of active knee flexion. Advancement of the limb for swing depended on direct hip flexor action. Mid swing floor clearance required excessive hip flexion to accommodate the passive foot drop (see Figure 15-12B, left leg). Gait would be significantly improved by a DF assist orthosis.

Tibialis Posterior Weakness From Tendon Dysfunction

Posterior tibialis tendon dysfunction, once considered idiopathic, now is well correlated to overuse stress imposed by tendon anatomy and the mechanics of walking. As the TP muscle descends from its tibial origin, the tendon wraps snugly around the medial malleolus to

reach its insertion sites on the midfoot. Just below the bend, the tendon is flattened[89,90] and hypovascular.[41] During walking, the abrupt arc of hind foot eversion initiated by heel contact is actively opposed by the TP muscle. It is logical to assume that the abrupt, repetitive loading could create an interval of stress concentrated in the region where the tendon closely approximates the malleolus. Pathological studies show that tendon degeneration in this area and distally can be profound.[89,90] The functional consequences range from disruption in the linear organization of the collagen bundles, which reduces the tendon's strength, to frank tendon rupture.

Figure 15-12. Patient F has anterior tibialis weakness due to a cauda equina spinal cord injury. (A) Terminal swing with excess ankle PF and inadequate knee extension.

Figure 15-12. (B) Mid stance shows mild ankle PF and knee hyperextension. The contralateral (left) hip is excessively flexed to ensure clearance of drop foot.

Figure 15-12. (C) Pre-swing with a severe foot drop and no knee flexion to prepare for floor clearance.

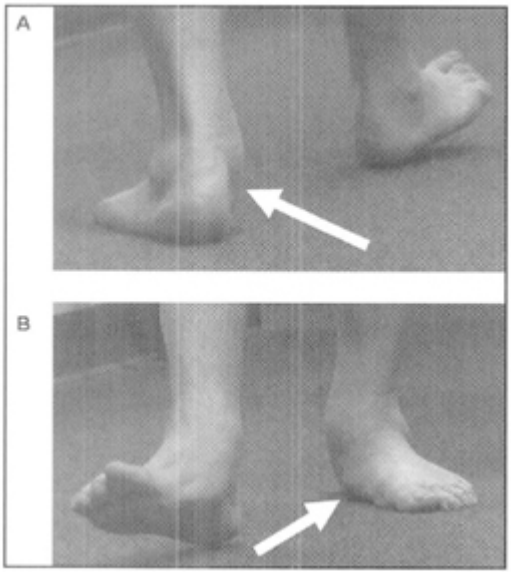

Figure 15-13. Patient G with posterior tibialis tendon dysfunction. (A) Weakened (2/5) posterior tibialis musculotendinous unit unable to prevent excessive eversion (lateral tilt of heel). (B) Medial arch flattens as body weight progresses across the foot.

Patient G: Posterior Tibialis Tendon Dysfunction (Figure 15-13)

Lateral tilt of the heel, identified by a posterior view of the foot (Figure 15-13A), was the most prominent sign of excessive eversion (ie, greater than 8° beyond vertical). The weakened TP musculotendinous unit (grade 2 manual muscle test) was unable to

support the loaded foot. Flattening of the medial arch as weight bearing progressed onto the forefoot accompanied heel eversion (Figure 15-13B).

As the underlying etiology of this condition is theorized to be tendon degeneration (tendinosis),[89,90] rehabilitation efforts in the early stages often focus on mechanically supporting the flattened arch to prevent further tendon lengthening and deformity.[24,73,75,144] Exercises to strengthen the weakened TP musculotendinous complex also have been strongly recommended to prevent further degeneration.[12,76-78,144]

SUMMARY

Reduced walking velocity often accompanies lower extremity weakness. This can either be intentional to reduce the muscle demands associated with walking or a result of an inability to achieve postures necessary for maximal step length (eg, calf weakness limiting the ability to achieve heel-off and a normal trailing limb posture). When sensation is intact, individuals with weakness often alter their movements in order to preserve stability and progression. Over time, however, the abnormal forces during stance can lead to further limb deformity (eg, the profound knee hyperextension found in individuals with longstanding post-polio or the flattening of the arch that occurs with posterior tibialis tendon dysfunction). When strengthening of weakened muscles is not realistic, then external support should be considered in order to prevent long-term joint disorders and pain.

PAIN

Pain is often underappreciated as a source of disability and gait dysfunction. While compensatory movement patterns may lessen pain, the altered mechanics can increase demands on the surrounding joint tissues and ligaments, thus furthering the potential for disability.

Osteoarthritis

Osteoarthritis is the most common clinical problem in orthopaedics.[81] The pathology is pervasive, progressive degeneration of the hyaline cartilage that covers the articular surface of the joints. The precise cause is unknown, but 3 contributing factors have been identified. Excessive repetitive impact force is the primary cause.[116] This is compounded by the limited capacity of articular cartilage to repair itself following damage.[82] Longevity, the third key factor, highlights the relationship between increased years of life and the overall number of impact events.[81] The incidence of osteoarthritis in young adults is low (<5% in those under age 44 years). Less than 30% of adults between 45 and 64 years of age have evidence of osteoarthritis. These percentages contrast sharply with the finding that more than 60% of the individuals over 65 years of age are disabled by osteoarthritis. A degenerated knee, however, is not inevitable, as a notable number of the elderly have pristine joints throughout life.[81,82]

Clinical histories of sports injuries, heavy manual labor, falls, and other accidents identify episodes of excessive loading during weight bearing. Joint instability from neuropathic arthropathy is another source of cartilage injury.[81,82] Excessive body weight and rapid loading of the strong limb to protect a weak or injured contralateral limb can also aggravate the situation.[22,61,81,82]

Biomechanical Factors Contributing to Osteoarthritis

Instrumented gait analysis has provided insights into biomechanical factors influencing the development of osteoarthritis. During each GC, the knee experiences 3 intervals with unique combinations of motion and weight bearing that can contribute significantly to the development of osteoarthritis. IC provides an interval of abrupt impact at the onset of each step (50% to 100%

body weight within 1% to 2% of the GC).[126,146] This is immediately followed by an interval of shock-absorbing knee flexion during loading response. Terminal stance is a longer but demanding phase of sustained single stance with an increasing moment (115% body weight for 15% GC). Radin, by animal experimentation, differentiated the response of articular cartilage to sustained load and rapid impact force.[118] An hour of standing with a heavy load, as the knee was either maintained in full extension or alternately flexed and extended, created only a minimal increase in the joint's low coefficient of friction.[117,118] Immobilization of the joint in full extension was similarly innocuous. In contrast, the addition of brief impulsive loads at a rate of 60 impacts per minute for 1 hour generated early signs of articular cartilage degeneration.[116] Both the chemistry and physical quality of the cartilage were indicative of osteoarthritis. The significance of a forceful impact rather than a static load has been related to areas of stiffer subchondral bone.[116] The authors interpreted these findings as evidence of micro-fracture and repair of the cancellous lamellae. Subsequent studies have confirmed that repetitive, high intensity impacts rather than static loads are the primary destructive forces that cause articular cartilage degeneration.[81,82]

Degeneration begins with fraying of the superficial zone.[81,82] This is followed by the development of vertical clefts and fissures that release tissue fragments into the joint space, exposing an uneven bony surface. Healing is minimal because mature articular cartilage is avascular and has no neural feedback system. Limitations in repair highlight the need for early clinical measures to prevent the development of significant deformity. The aim is to avoid exaggerated impact at IC.

Swing is a period of minimally loaded motion. Knee flexion and extension are passive events when walking at the optimum velocity. This low-force motion is assumed to facilitate nourishment of the avascular articular cartilage by infusion. Salter et al demonstrated the ability to heal traumatized articular cartilage by the use of continuous passive motion.[121]

Varus angulation of the knee is a self-perpetuating deformity that follows the progressive loss of the knee's medial joint surface. The

difference in condylar size (medial greater than lateral) allows for unequal loading, but the medial condyle still is susceptible to overloading.[47,66] As the underlying bone deteriorates, the joint surfaces develop an increasing medial tilt.[125] Gait analysis of patients with a varus knee deformity demonstrates a relationship between knee osteoarthritis and a progressively increasing adductor moment of the tibia.[6,7,62,124]

The resulting malalignment not only adds to the severity of the deforming forces but also imposes greater demands on the supporting muscles.[116] This results in greater compressive joint forces, thereby adding to the demands on the articular cartilage.

Patient H: Osteoarthritis (Figure 15-14)

This individual sought medical care for bilateral knee pain. When standing, both knees were flexed and in varus. These are the most common deformities at the knee related to degenerative arthritis. Excess knee flexion during stance increases the quadriceps activity required for stability[25,100] and generates higher compressive forces on the joint. To lessen the quadriceps demand and associated joint force, sagittal plane vector analysis revealed that this patient used a forward trunk lean to position the body vector just anterior to the knee joint axis (Figure 15-14A). This posture was a costly compromise as excess flexion of the ankle and hip was required to maintain balance. Consequently, the extensor muscles (soleus, gastrocnemius, and gluteus maximus) had increased loads, which reduced the patient's walking endurance. The varus angulation was a self-perpetuating deformity that followed the progressive bone loss. The loss was greatest in the areas of maximum compression (ie, the medial condyles). Coronal plane vector analysis demonstrated an excessive medial torque (Figure 15-14B), which required bilateral knee osteotomies to correct the varus deformity and total knee joint replacements to attain smooth joint surfaces.

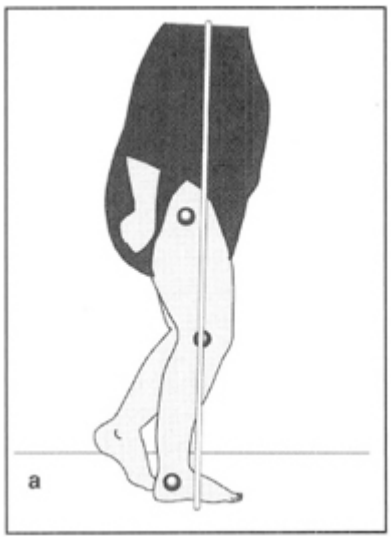

Figure 15-14. Patient H has a knee deformity arising from degenerative arthritis. (A) Forward trunk lean moves the vector anterior to the flexed knee, lessening the demand on the quadriceps.

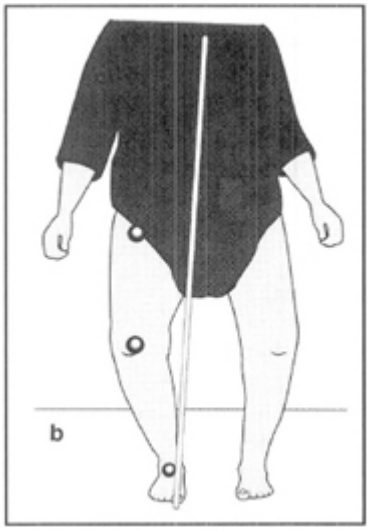

Figure 15-14. (B) Varus (excess adduction) with a marked medial vector.

RHEUMATOID ARTHRITIS

This systemic autoimmune disease attacks the synovial membranes that line the capsules of diarthrodial joints,[120] creating a vicious cycle of disability. The initial rheumatic response is thickening of the synovial membrane and excess synovial fluid. Subsequently,

swollen tissues encroach on joint spaces and are compressed with motion.[120] The result is pain. Patients protect themselves by decreasing their motion. Also, pain inhibits muscle activity.[30] The resulting muscle weakness denies the patient the usual protection from the traumas of walking.[35] Each step repeats the rheumatic cycle. The late effects are lax ligaments, increasing muscle weakness, bone destruction, and joint deformity.[27,129]

The foot is a primary source of disability in individuals with rheumatoid arthritis. In a study of 1000 individuals with rheumatoid arthritis, the foot caused walking disability in three-quarters of the cases and it had a 4-fold higher chance of being the only joint to impair gait than either the knee or hip.[51] The clinical picture in the foot includes hallux valgus and dorsal subluxation of the lesser MTP joints with clawing of the proximal interphalangeal articulations.[35] Also, the metatarsal fat pad moves away from its normal protective plantar location. The source of these additional deformities appears to be excessive action of the long toe extensors to assist forefoot protection by ankle DF.

Rheumatic involvement of the MTP joints is often a significant source of disability and pain. The metatarsal joints are small and tightly bound to each other by dense ligaments, so distension and malalignment are not early findings. The articular cartilage on the head of the metatarsal covers both the plantar and distal surfaces of each bone. This lengthy contour implies 2 joints: plantar and phalangeal. During gait, a compressive plantar force begins with the attainment of foot flat and then increases in intensity as the body weight vector advances across the forefoot.[23] During late terminal stance, the vector is concentrated over the MTP joints and the compressive force is significantly increased with heel rise. Pain and higher walking pressures under the MTP joints have been associated with greater forefoot pathology in individuals with rheumatoid arthritis.[139] Unfortunately, the substitutions used by the patient to minimize pain in this region often tend to obscure the problem. It is common for clinicians to dismiss the initial signs of disability as minor.

Figure 15-15. Rheumatoid arthritis. Rocker shoes allow progression across the forefoot and toe rockers without the need for toe extension. This can lessen forefoot pain and allow the body weight to advance across the foot during gait.

Early in the course of forefoot rheumatoid arthritis, the pain of joint compression can be eased by splinting the MTP joint with rocker shoes (Figure 15-15). This avoids the usual changes in joint volume that accompany MTP DF during terminal stance and pre-swing.[38] With MTP joint pain minimized, the body weight vector is able to advance over the rocker contour of the shoe sole. This preserves plantar flexor muscle strength by allowing the muscle group to be challenged with each step. If the disease continues to deteriorate, the patient can reduce forefoot pain only by limiting advancement of the vector beyond the ankle joint axis. This deviation can lead to further atrophy of the gastrocnemius and soleus muscles as they have been deprived of their normal functional challenge.

Patient I: Rheumatoid Arthritis (Figure 15-16)

An obscure but very significant factor that limits the walking ability of persons disabled by rheumatoid arthritis is calf muscle weakness. This patient also had a mild (10°) knee flexion contracture. Loading the limb with the knee flexed induced a challenging torque to a quadriceps with only borderline strength (grade 3+). Knee flexion placed the body weight vector posterior to the knee joint axis (Figure 15-16A). The patient was able to meet the resulting internal extensor demand through 2 mechanisms. As the flexion contracture was less than 15°, the quadriceps had a favorable moment arm that made this demand no greater than would be experienced with a 5° position.[100] Also, dynamic flexion at the knee was minimized by the short step as there was no heel rocker to accelerate knee flexion. Vertical leg alignment allowed the weak calf muscles to stabilize the tibia without the challenge of an internal plantar flexor moment.

Figure 15-16. Patient I has soleus weakness associated with rheumatoid arthritis (vertical line = body weight vector). (A) Loading response with the vector posterior to the flexed knee and at the axis of the ankle.

Figure 15-16. (B) Early mid stance showing increased knee flexion displaces the knee joint axis more anterior to the vector.

Figure 15-16. (C) Terminal stance with the base of the vector at the midfoot, anterior to the dorsiflexed ankle and slightly posterior to the knee.

Progression of the body over the supporting foot in mid stance increased both ankle DF and knee flexion (Figure 15-16B). The cause was inability of the weak plantar flexor muscle mass (grade 2) to restrain the tibia. Soleus and gastrocnemius activity had been inhibited by metatarsal joint arthritis that made the forefoot intolerant of full weight bearing. Persistent quadriceps action provided the necessary knee stability. In terminal stance, further advancement of the body vector to the midfoot reduced knee flexion with a minimal increase in ankle DF (Figure 15-16C). The newly acquired tibial stability reflected passive tension of the gastrocsoleus as the ankle reached the limit of its DF range. Relative extension of the knee occurred as the femur advanced more than the tibia. This moved the vector closer to the knee and reduced the demand on the

quadriceps. The weak gastrocsoleus musculature prevented the patient from having a forefoot rocker. Advancing the vector to the forefoot would have lengthened the DF lever arm beyond that which the weak calf muscles could control. Continued footflat contact also reduced the weight applied to the painful metatarsal heads. Pre-swing knee flexion was lacking because there was no toe rocker to advance the tibia ahead of the vector (Figure 15-16D). Instead, heel rise was delayed until most of the body weight had been transferred to the other limb. The sum of these factors was a very short step.

Initial swing knee flexion was a deliberate act that lifted the tibia more than the hip flexed (Figure 15-16E). The foot's clearance of the floor was assisted by ankle DF to neutral. Mid swing advancement of the limb also was aided by the early ankle DF while the hip flexors pulled the femur forward (Figure 15-16F).

Figure 15-16. (D) Pre-swing includes a limited heel rise with the ankle dorsiflexed and inadequate knee flexion. The low level of the vector indicates only partial weight bearing.

Figure 15-16. (E) Initial swing with limited knee flexion and excessive ankle DF.

Figure 15-16. (F) Mid swing with excessive ankle DF limiting the amount of hip and knee flexion needed for floor clearance.

SUMMARY

Loss of muscle strength is much greater in rheumatoid arthritis than degenerative arthritis because of the more intense inflammatory process. Inhibition of muscle action relieves pain by reducing the compressive force generated by the stabilizing muscles. The secondary effects of enforced inactivity are reduced motion, development of significant disuse muscle weakness, and postural deformities.

IMPAIRED CONTROL

The precision, coordination, speed, and versatility that characterize normal walking are the outcome of selective muscle control. The neurons responsible for initiating optimum muscle performance have their cell bodies in the motor cortex of the cerebrum. Their awareness of the motor needs is maintained by a continuum of information from sensors throughout the nervous system (ie, the periphery, cerebellum, basal ganglia, and other deep brain centers).[10] Signals for purposeful motion are generated in the

functionally designated cerebral cortical motor area and then transmitted by the long axons passing through the brain and down the spinal cord to the analogous level in the anterior horn.[5] The axons of the upper motor neurons transfer their command signals by direct synapse to the lower motor neurons responsible for activating the motor units of the muscles controlling the desired function. Hence, the chain of selective muscle activation involves only the upper and lower sets of motor neurons.[45] The long upper motor neurons are a virtual expressway of motor control signals to initiate prompt and precise muscle action. The anatomical name of this expressway is the lateral corticospinal tract (or pyramidal tract).[5,10,45] Experimental stimulation studies have demonstrated that there is almost a one-on-one relationship between the upper motor neuron cells in the cerebrum and the lower motor neurons cells of specific muscles in the spinal cord.

This primary source of selective motor control is supplemented by a second, more primitive motor (extra-pyramidal) system that has cell bodies in the deeper layers of the motor cortex as well as the mid brain, basal ganglia, and brainstem.[39] Their axons descend in tracts that are in, or adjacent to, the medial corticospinal pathways. These tracts lack the selective quality of the lateral corticospinal pathways. Their axons terminate on interneurons in the spinal cord or medial brainstem, which activate the muscles in synergistic patterns.[5] Postural control and multisegmental reflexes are their main function. The neurons involved with limb motion combine the hip, knee, and ankle muscles into flexor or extensor synergies. Cortical input provides voluntary activation of the synergies but cannot select a single joint.

Selective motor control can be impaired by any mechanism that causes injury to the motor areas in the brain or the cervical and thoracic segments of the spinal cord. Clinical examples are stroke (cerebral vascular accident [CVA]), traumatic brain injury, tetraplegia, paraplegia, multiple sclerosis, cerebral palsy, hydrocephalus, infections, and tumors.

Stroke

Hemiplegia (unilateral paralysis of an arm and leg) is the typical impairment that follows a CVA.[18] A stroke occurs when the blood flow within an artery that feeds an area of the brain is abruptly interrupted by a clot (thrombus) or rupture (hemorrhage). The most frequent cause of a stroke is a thrombotic blockade of the internal carotid or middle cerebral artery (76%) or other arterial branches nourishing the motor area of the cerebral hemisphere. Similar pathology in a vertebral artery causes brainstem strokes (15%). Hemorrhage is an infrequent cause of a stroke (9%).[18] As the brain has an extensive vascular system, the precise location and extent of a lesion is highly variable, consequently defining the immediate disability is difficult.

Twitchell provided the first comprehensive description of the course of a stroke in 1951.[138] He found that the immediate reaction to the CVA was an acute loss of function (neurological shock). By the second day, hyper-reflexia and other signs of motor arousal began to appear. Within 1 to 38 days, plantar flexor resistance increased and early spasticity developed in the adductors and extensors of the hip and knee while the plantar flexors displayed clonus. Hip flexion was the first willed movement in the lower limb, quickly followed by a complete flexor synergy involving the hip, knee, and ankle. An extensor synergy developed in a similar manner. As spasticity increased, the arm and lower limb assumed the “typical” hemiplegic resting posture. The sequence of increasing reflexes, spasticity, primitive synergies, and eventual voluntary motion might be completed in a few days or require several weeks. Twenty percent of the patients had a protracted course that stopped before full recovery was attained. The custom of continuing bed rest until the patient displayed useful function probably contributed to his or her slow progress.

Today, the dominant sign of impaired motor control is muscle weakness.[93] Advances in modern medicine have replaced prolonged bed rest with early mobilization. The patients are entering rehabilitation programs within 6 to 10 days of their acute stroke.[91]

While hyper-reflexia is still present,[39] spasticity is mild and contractures generally are minor. The rapid development of new diagnostic techniques to better define human brain function is exponentially expanding. Much of our detailed knowledge of the neurological control of muscles has been derived from experimental studies of animals. While informative, these data can be misleading because humans lack the primitive neural paths that allow many animals to walk independently immediately after birth.

Travis and Woolsey (in 1955) challenged the assumption that the control of human motion depends on the pyramidal tracts by bilaterally dividing the tracts in monkeys.[134,135] The result of their first surgery was marked loss of mobility similar to the rigidity that Twitchell reported after a stroke. However, closer examination identified contractures as the cause of reduced mobility rather than a change in neurological control. Repeating the surgical transection of the pyramidal tracts and adding joint ROM 10 times each day resulted in independent mobility. Now, the primary function lost by the monkeys was precise distal motor control such as that required to grasp the support bar with their toes or selectively poke just the index finger through a hole to reach the lock on the food door. Otherwise, the monkeys' primitive control centers provided all the basic functions.

Humans disabled by a severe stroke, however, may display primitive flexor and extensor patterns of limb motion when they begin to walk. The most recent research on human motor control, however, has demonstrated plasticity.[19,37,44,95,96] There is evidence that an impaired brain can lessen the impact of an acute injury by borrowing adjacent residual neurons or even forming new neurons to enlarge the neuronal area devoted to a particular function.[97] The wide range of initial disability following an acute stroke and the seeming inconsistency of recovery,[110] however, continue to challenge therapeutic planning.

Classification of Stroke Disability

The extensive arterial supply of the brain and the vulnerability of arteries to occlusion and injury are the source of the vast variety of strokes and levels of disability that follow. Mulroy's use of cluster analysis to classify the severity of functional impairment following an acute stroke has made a valuable contribution to gait analysis.[91] Stride characteristics and joint kinematics of persons recently disabled by a stroke (mean age 57.4 years; admitted for inpatient rehabilitation an average of 9.4 days following their first stroke) were used to classify the variable walking patterns. The computer selected 3 parameters (velocity, mid swing ankle DF, and mid stance knee flexion) to differentiate patient performance. Velocity assigned the patients to 3 groups: FAST (44% normal velocity), MODERATE (21% normal velocity), and SLOW walkers (10% normal velocity). Mid swing ankle DF separated the SLOW walkers who had moderate equinus (8° to 11° PF) from the FAST and MODERATE groups who accomplished DF (1° to 3°). Mid stance knee flexion divided the SLOW walkers into 2 groups: extended and flexed. The SLOW-EXTENDED group (11% normal velocity) substituted for their incapable quadriceps by using their stronger gluteus maximus to withdraw the femur and extend the knee. The result was slight knee hyperextension (6°). In contrast, the SLOW-FLEXED group (10% normal velocity) had sufficient quadriceps strength to support body weight with the knee in significant flexion (23°). While notably less than the SLOW-FLEXED group, knee flexion was also present in the FAST (7°) and MODERATE (14°) groups. It was diminished, however, due to the combined capacity of the gastrocnemius and soleus to restrain tibial advancement and the quadriceps to support the knee. The final 4 groups were FAST (44% normal velocity), MODERATE (21% normal velocity), SLOW-EXTENDED (11% normal velocity), and SLOW-FLEXED (10% normal velocity).

Exemplar Patient Examples From Cluster Analysis

To define the functional significance of Mulroy's classification criteria, one subject from each of the 4 classes was selected for a

more detailed analysis. With permission of the author,[91] data from the study (stride characteristics, kinematics, EMG, and manual muscle tests) were used to more fully interpret the significance of the classification. Joint motion was estimated from the gait video and the EMG was quantified and normalized.

Class I: FAST Walker (Patient J; Figure 15-17)

The subject representing the functional capability of this class was 58 years old and had a self-selected gait velocity 55% of normal. Both discriminating gait events were normal. In mid stance, the knee was fully extended (Figure 15-17A) and in mid swing, the ankle achieved neutral DF (Figure 15-17B). Yet, this subject's velocity was notably less than normal in part due to a lack of heel-off in terminal stance (Figure 15-17C). This inconsistency was explained by the additional available data and the functions observed in other phases of gait.

Manual muscle tests of the gluteus maximus and quadriceps (VI) were graded strong, while moderate was the grade for soleus. The examiner's use of the "strong, moderate, weak" grading system implied the tested muscles responded as components of mass extensor and flexor synergies. The quantified EMG showed a high intensity for both the VI and the gluteus maximus, while the soleus displayed less intensity. The anterior tibialis (as part of the flexor synergy), also, was graded strong. Quick stretch generated little spasticity. Only adductor longus persisted for more than one second.

The gait EMG displayed a grossly normal interference pattern but an extensor synergy was inferred by the simultaneous onset at IC of the gluteus maximus, VI, and soleus (Figure 15-17D). Precise phasing was blurred by scattered, brief bursts of signal and prolonged actions. Mean intensity of the muscles during walking was moderate for the gluteus maximus (22% MMT) and VI (18% MMT), while the weaker soleus registered a greater effort (39% MMT). EMG differences in the adjacent phases indicated more selectivity by the VI and soleus, which had reciprocal intervals of high and low intensity. During loading response, the VI was strong and the soleus EMG was sparse. The converse occurred in terminal stance, with the soleus strong and the VI low. Despite extra effort of the soleus, tibial

control was inadequate, leading to reductions in stride length (63% N) and cadence (87% N).

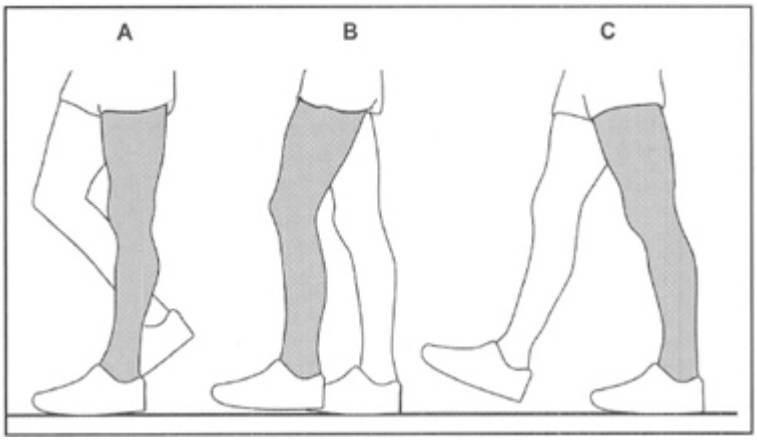

Figure 15-17. Patient J is representative of the FAST walker post-stroke. Discriminating events in both (A) mid stance and (B) mid swing were normal. (C) Lack of a heel rise in terminal stance identified plantar flexor weakness, which compromised stride length and velocity.

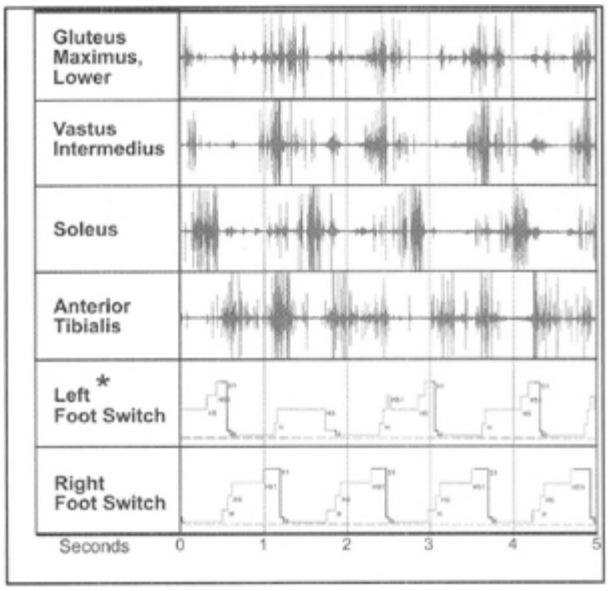

Figure 15-17. (D) Early stance soleus EMG activity low. Despite apparently good activity in the soleus during late stance, footswitch records indicated no heel off until the other foot contacted the ground, confirming plantar flexor weakness. The gluteus maximus and VI are slightly prolonged. (Note: * = reference limb.)

Even though the mid stance functions of knee and ankle were appropriate, the absence of a heel rise in terminal stance further confirmed plantar flexor weakness (see Figure 15-17C). The suggestion of knee hyperextension indicated that the stance limb lacked the strength to utilize the momentum generated by the forward swing of the sound limb. Hence, the weak plantar flexors (soleus graded moderate) were unable to lock the ankle so that body weight could roll forward onto the forefoot. This loss of progression deprived the subject of considerable step length.

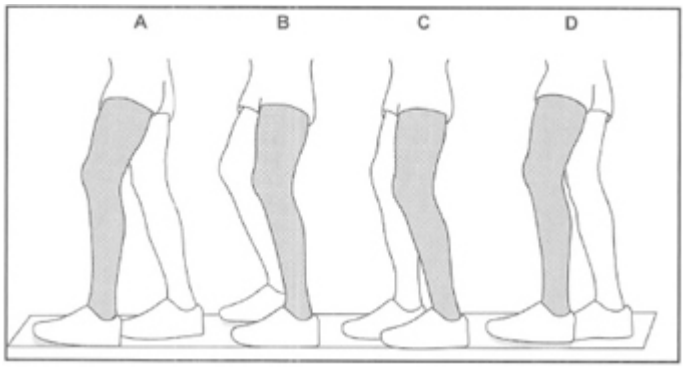

Figure 15-18. Patient K serves as the exemplar for the MODERATE walker post-stroke. (A) IC occurred with a low heel and flexed knee. (B) Mid stance DF, knee flexion and hip flexion excessive. (C) Pre-swing lacks forward progression over the toe rocker. Ankle PF, knee flexion, and heel-off are inadequate as the individual did not achieve a good trailing limb posture in terminal stance. (D) Mid swing ankle achieves neutral for foot clearance.

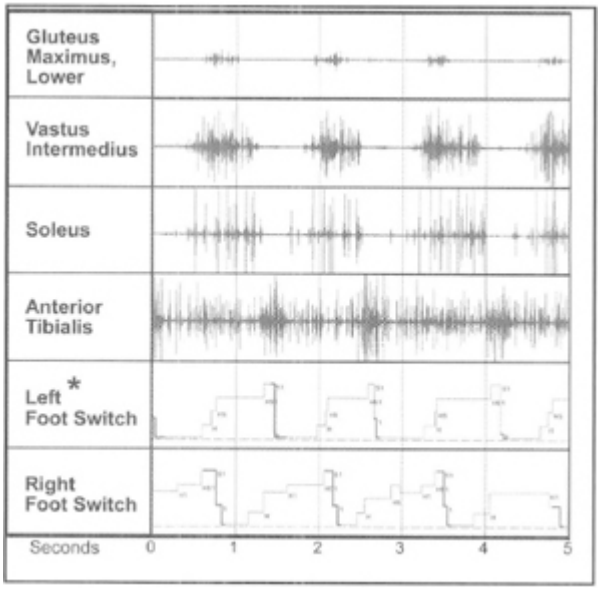

Figure 15-18. (E) EMG record reveals sparse soleus EMG activity indicative of inadequate strength to control tibial advancement. Prolonged VI activity supports a flexed knee during SLS. Continuous TA activity permits neutral ankle for mid swing foot clearance. In stance, the activity appears to be helping draw the posteriorly aligned HAT forward. (Note: * = reference limb.)

Class II: MODERATE Walker (Patient K; Figure 15-18)

The walking velocity of the 44-year-old representative subject was only 32% of normal. IC occurred with a low heel contact and excessive knee flexion (Figure 15-18A). The excess knee flexion continued through mid stance, indicating a performance error in one of the differentiating gait factors (Figure 15-18B). Step length was notably curtailed as evidenced by the close position of the 2 feet in pre-swing (Figure 15-18C). Pre-swing ankle DF was excessive, the heel failed to lift from the ground, and knee flexion was notably limited. By mid swing, the ankle achieved neutral DF (Figure 15-18D), indicating the patient's capacity to perform the second differentiating gait factor.

Manual muscle testing showed the greatest weakness was in the plantar flexors with a grade poor soleus. The gluteus maximus was

graded fair, the VI was graded fair plus, and the anterior tibialis was graded good. The spastic responses to quick stretch were brief with moderate EMG by the VI and gluteus maximus. A sustained but minimal pattern of spasticity was recorded by the soleus.

During walking, the EMG indicated that the onset of the soleus, gluteus maximus, and VI occurred slightly before heel contact (Figure 15-18E). Brief, low-intensity gluteus maximus activity (5% MMT) provided minimal stability for the hip during WA. The sparse, high-amplitude soleus activity (26% MMT) continued until pre-swing and provided insufficient force to control tibial advancement during stance. Moderate intensity activity of the VI (27% MMT) was prolonged throughout SLS to stabilize the flexed knee. The persistence of inadequate knee extension during SLS implied that the quadriceps were unable to overcome the excessive tibial tilt allowed by weakness of the soleus. Near continuous activity of the TA ensured adequate DF during swing and allowed a low heel IC. Continued TA activity during SLS was likely present to help advance the posteriorly located HAT as the quadriceps could not advance the femur over the unstable tibia. The result was a shortened stride length (38% N) while the cadence was nearly normal (83% N).

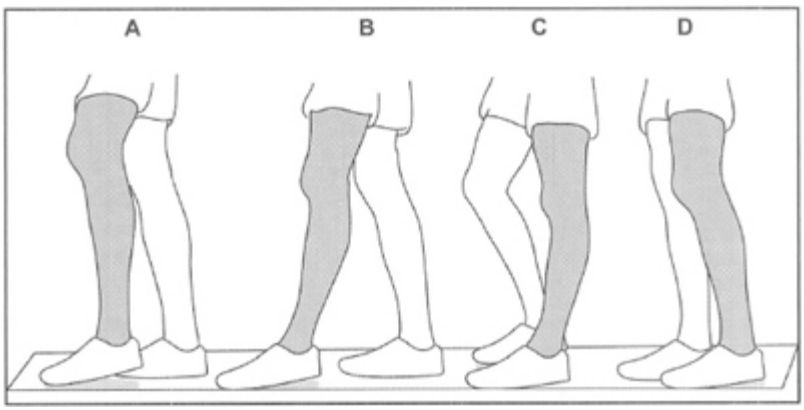

Figure 15-19. Patient L is representative of the SLOW-EXTENDED walker post-stroke. (A) Mid swing ankle PF was excessive due to weak pretibials. (B) PF persisted at IC. (C) Thigh retraction by the gluteus maximus in mid stance accentuated knee hyperextension and protected the weak quadriceps. Ankle PF persisted. (D) While deviations in previous phases resulted in a shortened step length, the pre-swing posture-illustrated patient had only minimal

contractures at the ankle. As weight transferred to the contralateral limb, the restraining force of the lower gluteus maximus ceased. The ankle dorsiflexed, revealing greater ROM than observed in previous phases. Knee flexion was initiated.

Class III: SLOW-EXTENDED Walker (Patient L; Figure 15-19)

The representative subject (age 45 years) had a walking velocity of only 17% of normal. Both discriminating events displayed notable errors. During mid swing, the ankle was excessively plantar flexed (Figure 15-19A). The equinus persisted through swing, resulting in a forefoot-first IC (Figure 15-19B). At mid stance, the knee was hyperextended, the ankle plantar flexed, and the foot flat (Figure 15-19C). During stance, the posterior tilt of the involved limb's tibia limited the forward swing of the contralateral limb, resulting in a very short step length (Figure 15-19D).

The EMG showed 2 patterns of muscle action. During the manual muscle test, the gluteus maximus (graded fair minus) had a normal dense interference pattern of moderately high action potentials (Figure 15-19E). In contrast, both the VI and soleus displayed a sparse signal pattern and were graded fair minus and poor minus, respectively. No EMG signal was recorded from the anterior tibialis, and the muscle was graded as 0/5 (absent). Quick stretch stimulated a short response (0.3 sec) in the gluteus maximus and VI. The soleus reacted with a long sequence of miniscule, isolated blips.

During walking, the onset of gluteus maximus activity was premature, occurring in mid swing. The signal pattern consisted of a long sequence of dense EMG, half with low intensity followed by an increase to moderately high intensity. A second shorter packet of signals followed in the last half of the prolonged, 2-second stance interval. Gluteus maximus activity contributed to thigh extension in SLS and compensated for the weakened vastii (see Figure 15-9C). Neither the VI nor soleus displayed any functional muscle activity (just occasional single spikes or brief bursts of EMG) (Figure 15-19F). Floor contact was a footflat pattern, which shifted between varus (H-5), valgus (H-1), and neutral (H-5-1).

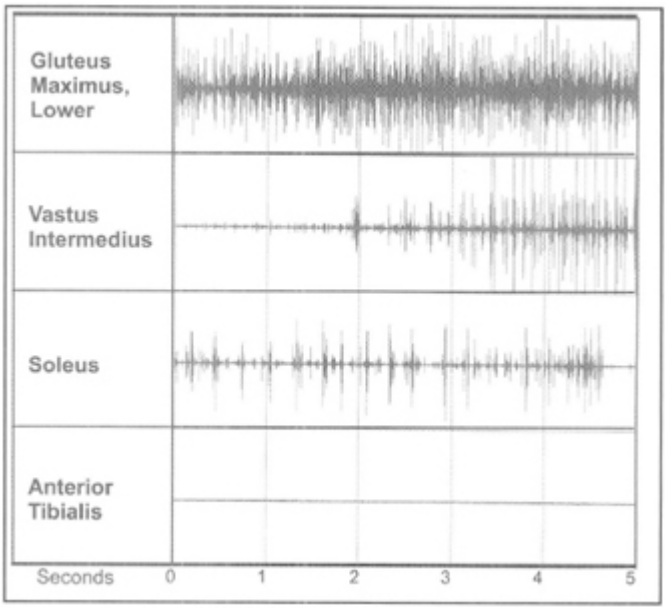

Figure 15-19. (E) EMG activity recorded during manual muscle tests revealed a normal dense interference pattern for the gluteus maximus. The VI and soleus displayed a sparse signal, while no significant EMG was documented for TA.

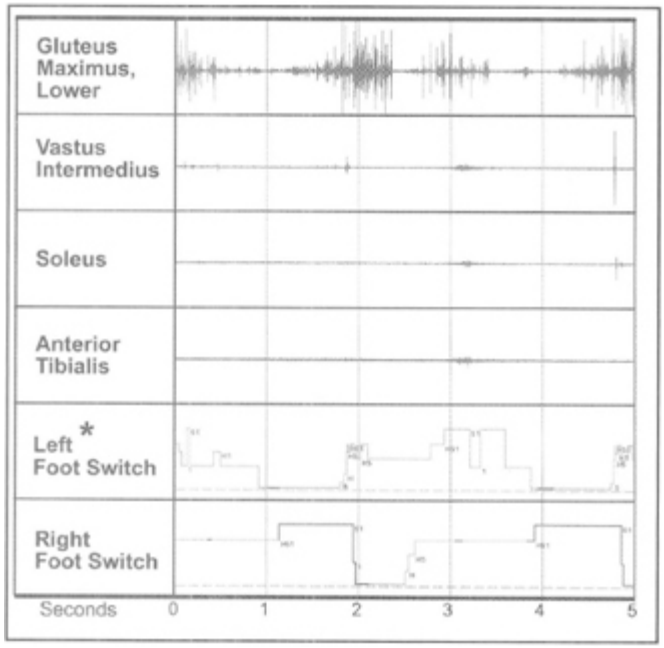

Figure 15-19. (F) During walking, only the gluteus maximus displayed any EMG activity. Moderately intense activity through the terminal stance restrained the femur and stabilized the weakened knee. Termination in pre-swing allowed the knee to flex. (Note: * = reference limb.)

Mid swing ankle DF (the second discriminating gait event) was incomplete, with the foot in equinus. The anterior tibialis displayed no EMG during either manual muscle test or walking.

Thus, the patient's hip muscles were the only ones with voluntary control. These limitations were reflected in his curtailed stride length (45% N) and slowed cadence (37% N).

Class IV: SLOW-FLEXED Walker (Patient M; Figure 15-20)

The representative subject, age 63, walked with velocity of just 9% normal. The flexed knee posture (10°) began at IC (Figure 15-20A) and rapidly increased during loading response as the tibia collapsed forward. By mid stance, the knee was flexed 20° (Figures 15-20B). The demand on the soleus was extreme, as the excessive ankle DF had advanced the body mass so far anterior that the patient's knee was in line with the great toe while the thigh remained nearly vertical. Pre-swing revealed a very shortened step (Figure 15-20C). The increase in flexion was a reaction to poor ankle control. In mid swing, the unloaded ankle plantar flexed 8° (Figure 15-20D). Thus, all gait phases indicated poor foot control. The result was a shortened stride length (27% N) and a slowed cadence (34% N). All muscles were graded weak by manual muscle testing. Only the soleus showed small signs of prolonged spasticity.

The EMG recordings of the manual muscle tests supported the poor muscle test responses. All 3 extensor muscles showed low-amplitude (3 to 8 digitized units [du]), fairly sparse signals. Walking stimulated a major increase in muscle and signal density, although qualities differed considerably (50 to 200 du). This improved muscle activation reflected the positive influence of an upright posture, which has been demonstrated both clinically and by experiment.[105] The difference in magnitude has been interpreted as the sign of a marked reduction in selective control. When the EMG representation of the manual muscle tests was raised to the customary normal minimum (50 mv), the normalized gait EMG presented a clinically logical pattern (Figure 15-20E). The 3 basic extensors became active at floor contact. High activation of soleus during WA indicated a need to

control forward fall of the tibia. Lower amplitude activity persisted through terminal stance. Prolonged VI activity through SLS was necessary to stabilize the flexed knee. Substantial assistance also was provided by the clinician. EMG activity in the weak gluteus maximus was virtually continuous and displayed an inconsistent sequence of high, moderately robust spikes interspersed with small potentials. With the clinician's assistance, the patient's hip extensor activity was sufficient to maintain the HAT over the supporting foot. Although anterior tibialis activity extended through swing, it failed to fully support the foot in mid swing (see Figure 15-20D). Basically, the patient's gait represented alternating primitive extensor and flexor synergies with inconsistent control.

The Hierarchy of Stroke Disability

Historically, the person disabled by stroke typically walked on his or her forefoot because the ankle was fixed in equinus (excess PF). The cause was attributed to extensor spasticity, a PF contracture, dorsiflexor weakness, or a combination. Now it is recognized that the equinus posture results from allowing the foot to dangle while sitting on a chair or lying in bed while recovering from a stroke. Today, the dominant gait impairment displayed by individuals recovering from a stroke is excessive ankle DF, which persists until the plantar flexor muscles regain adequate strength and control. Knee flexion in stance also is common.

Each individual selected to serve as an exemplar for the 4 classes of hemiplegic gait displayed characteristic patterns of walking that can be interpreted in terms of a basic hierarchy of impaired stance limb progression. None of the individuals had a heel rise during terminal stance nor did they display elastic recoil in pre-swing. All had a shortened stride length.

The demand for controlling limb progression begins with the onset of SLS. The body vector begins to move anterior to the ankle axis as the contralateral toe lifts from the ground. While the conspicuous deviation in mid stance is inadequate knee control, the basic impairment proved to be inadequate plantar flexor muscle activation. Both the manual muscle test and EMG confirmed the reduction in

soleus muscle activity. The minimum loss was an absent heel rise during terminal stance by the FAST walker. His EMG during walking displayed reduced soleus action, which correlated with lessened tibial progression and pre-swing push-off momentum for swing. The outcome was a shortened step and reduced velocity.

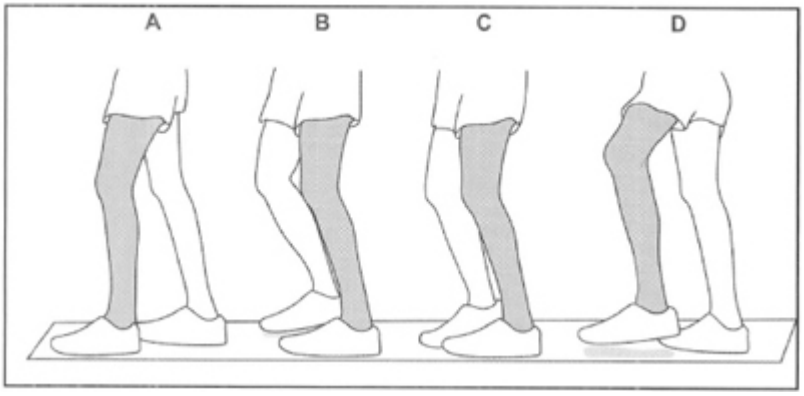

Figure 15-20. Patient M serves as the exemplar for the SLOW-FLEXED walker post-stroke. (A) Footflat IC with flexed knee. (B) Mid stance with extreme DF and flexed knee and hip. Clinician assistance required for stability. (C) Postures persist into pre-swing. (D) Mid swing ankle in slight PF.

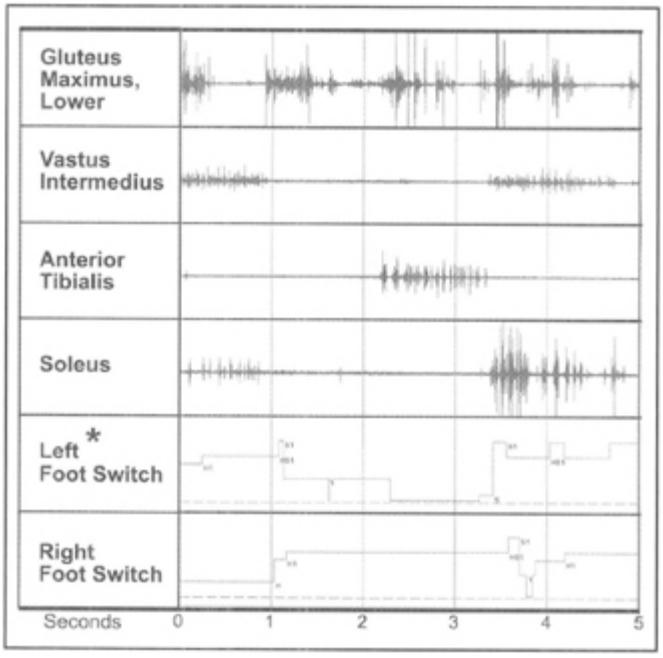

Figure 15-20. (E) EMG recording suggests an extensor synergy with the soleus, vastii, and gluteus maximus starting at the same time.

The maximum intensity of the soleus in loading response is consistent with an unstable tibia. Prolonged activity of the VI is required to stabilize the flexed knee. Near continuous inconsistent pattern of gluteus maximus indicative of poor trunk stability. Moderate level activity of weak anterior tibialis insufficient to support foot in mid swing. (Note: * = reference limb.)

The next level of stroke impairment (MODERATE) displayed insufficient quadriceps support of the knee. The quadriceps actually was challenged by the forward tilting of the tibia due to inadequate ankle control by the weak calf muscles. This prevented the quadriceps from pulling the femur ahead of the tibia. Thus, the knee remained in excess flexion in mid stance.

An alternate pattern of knee control was displayed by the subject from the SLOW-EXTENDED class. He was able to walk despite EMG evidence that neither the soleus nor quadriceps were active. EMG during both the manual muscle tests and walking showed good participation of the gluteus maximus. Having adequate hip extensor strength allowed the gluteus maximus to control the knee by retracting the femur. A modest ankle PF contracture provided the necessary ankle stability.

The fourth level of stroke disability was exhibited by the exemplar selected for SLOW-FLEXED. An even greater loss of quadriceps control at the knee as well as the hip extensors weakness necessitated additional support from the examiner to walk. Weak hip extension prevented the client from retracting the thigh to stabilize the weakened knee. Strength limitations in the muscles at all 3 joints left these patients with borderline walking ability.

Ankle DF in mid swing is a simple motion created by the anterior tibialis muscle. This muscle can be activated by selective control or as part of the primitive flexor synergy. Without the long toe extensors or peroneus tertius, however, the foot will go into varus. Twitchell found the flexion synergy to be the first voluntary function to return following the initial acute shock, yet inadequate ankle DF in swing is a frequent finding.[138] It was found in about half of the subjects in this study.

PATIENT N: DYNAMIC VARUS IN SWING (FIGURE 15-21)

The hemiplegic foot frequently displays varying degrees of varus during swing, stance, or throughout the GC.[109] Spasticity and the primitive locomotor patterns are 2 dominant sources of muscle dysfunction. The effect on the knee varies with the activity of the hamstrings and severity of the equinus.

The mode of impaired foot control displayed by this patient resulted from hemiplegia following a ruptured aneurysm. Swing phase varus was the dominant deviation. The apparent cause was strong TA muscle action as part of the flexor pattern, while the long toe extensors were far less active.

IC was made with the lateral side of the foot (H-5) while the knee was excessively flexed and there was normal hip flexion (Figure 15-21A). A prominent TA tendon was evident. The immediate concern was having a stable foot posture for WA.

Loading response involved a rapid drop of the foot into a safe plantigrade support pattern (H-5-1) with the tibia vertical (Figure 15-21B). This signified prompt TA relaxation as the extensor muscle synergy took over.

Persistence of the plantigrade foot alignment now depended on the intensity of the plantar flexor muscle action. Total foot contact in pre-swing implied adequate mediolateral balance (Figure 15-21C). It also signified insufficient ankle DF range to fully move the body weight over the metatarsal heads. The absence of a forefoot rocker during terminal stance and the diminished toe rocker in pre-swing denied the patient pre-swing knee flexion.

Initial swing initiation of limb flexion in preparation for floor clearance stimulated foot inversion (Figure 15-21D). Close inspection of the anterior ankle provided a visible muscle test. A prominent anterior tibialis tendon indicated strong muscle action while inconspicuous toe extensor tendons implied poor function of the lateral group. The latter also was confirmed by a lack of toe hyperextension at the MTP joints. Such findings implied the TA

muscle was the dominant ankle dorsiflexor. Limited knee flexion resulted in an early toe drag.

In mid swing, strong TA muscle action caused marked varus yet the foot appeared to be in slight equinus. There really were 2 patterns of foot DF (Figure 15-21E). The medial side of the foot (first 2 rays) was at neutral. Laterally, however, the foot was slightly plantar flexed. Hence, the TA had the strength to dorsiflex the ankle but its tendon alignment first inverted the foot. The limited ability of the strong TA action to raise the whole foot was most apparent from an anterior view of the foot (Figure 15-21F). Depression of the fifth ray was a potential source of foot drag. To avoid a drag, this patient, lacking free knee flexion, compensated by lifting the whole limb using contralateral vaulting (see Figure 15-21E).

Figure 15-21. Patient N has a hemiplegic varus foot. (A) IC with the heel and fifth metatarsal. There is excessive knee flexion. The TA tendon is prominent.

Figure 15-21. (B) Loading response with total foot contact (heel, 5th and 1st metatarsals). The tibia is vertical and the knee and hip are flexed.

Figure 15-21. (C) Pre-swing footflat contact (heel, 5th and 1st metatarsals). The ankle is mildly dorsiflexed and the knee slightly hyperextended.

Figure 15-21. (D) Initial swing foot inversion as flexor pattern is initiated.

Figure 15-21. (E) Mid swing with severe foot varus (inversion), prominent anterior tibialis tendon, and drop of the lateral side of the foot. Knee flexion is incomplete.

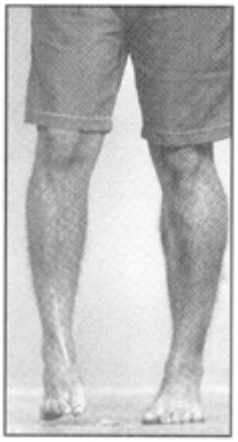

Figure 15-21. (F) Anterior view of the foot showing a drop of the lateral side. The TA is prominent but not the extensor digitorum longus and peroneus tertius. Toe clawing is by the short toe extensor tendons.

Figure 15-22. Patient O is a young child with a “typical” diplegic cerebral palsy and crouch gait. (A) Loading response with bilateral excessive ankle PF, excessive knee and hip flexion, and increased anterior pelvic tilt.

Figure 15-22. (B) Mid stance excessive knee flexion is less severe. Excessive ankle PF and anterior pelvic tilt persist.

Figure 15-22. (C) Pre-swing shows continuing severely excessive ankle PF. Knee flexion is inadequate.

Figure 15-22. (D) Mid swing PF is excessive. Increased hip and knee flexion assist with foot clearance. The ankle did not join the flexor pattern.

Spastic Cerebral Palsy

There are 2 characteristic gait patterns of children with spastic paralysis of diplegic, quadriplegic, or paraplegic origin: crouch and genu recurvatum.

Patient O: Crouch Gait (Figure 15-22)

This client displayed the typical pattern associated with a crouch gait, including bilateral excessive hip and knee flexion, excessive

ankle PF, and anterior pelvic tilt (Figure 15-22A). Even the vertical alignment of mid stance failed to correct the basic gait deviations, although their magnitude decreased (Figure 15-22B). During pre-swing, the subtalar joint everted as body weight progressed across the toe rocker (Figure 15-22C). During swing, hip and knee flexion were exaggerated to assist with clearance of the equinus foot (Figure 15-22D). This difficulty indicated ankle control was not part of the patient's flexor pattern.

Primitive patterning with excessive flexor muscle action at the hip and knee is the basic control deviation. Overactivity and contracture of the hamstrings is a particularly common finding.[132] The position of the ankle varies with the severity of calf muscle action. Individual patients, however, show considerable variation from the "typical" crouch gait model. Consequently, they have to be studied carefully and often the functional diagnosis depends on instrumented gait analysis.[32,33]

Figure 15-23. Patient P is a young boy with spastic diplegic cerebral palsy and spastic genu recurvatum. (A) IC by the forefoot with the ankle plantar flexed and knee flexed.

Figure 15-23. (B) The knee extends during loading response and ankle remains in PF.

Figure 15-23. (C) Mid stance shows knee hyperextension with excessive ankle PF and premature heel rise.

Patient P: Spastic Genu Recurvatum (Figure 15-23)

In contrast to the flexed knee posture of a crouch gait, spastic genu recurvatum is typified by movement of the knee into hyperextension during stance. The ankle is excessively plantar flexed and the hip may still have persistent flexion as the patient leans forward to balance over the plantar-flexed foot.

For this young boy with cerebral palsy, incomplete knee extension in terminal swing presented a poor limb posture for stance. IC occurred at the forefoot rather than the heel because there was inadequate knee extension and excessive ankle PF (Figure 15-23A). During loading response, the knee moved toward extension rather than flexion. This followed a drop of the foot into a delayed heel contact while the ankle remained plantar flexed (Figure 15-23B). In mid stance, the knee maintained slight hyperextension with a premature heel rise accompanied by excessive ankle PF (Figure 15-23C). These postures persisted in terminal stance as body weight advanced ahead of the foot (Figure 15-23D). There was a moderate trailing position at the thigh. The excessive ankle PF might have

been a voluntary substitution of vaulting to provide toe clearance by the other limb, which also had marked equinus and inadequate knee flexion. Pre-swing lacked knee flexion but the hyperextension was less as body weight transferred to the other foot (Figure 15-23E). Initial swing lacked knee flexion (Figure 15-23F). The toe drag appeared to increase the ankle PF and delay advancement of the thigh. Contralateral trunk lean was needed to free the foot due to the lack of initial swing knee flexion. In mid swing, hip flexion and knee flexion enabled the tibia to reach a vertical position. Vaulting on the contralateral side was required to clear the reference limb's excessively plantar-flexed foot (Figure 15-23G).

The electrogoniometer recording of the ankle showed continuous PF of approximately 20° (Figure 15-23H). There was a minimal (5°) decrease in the ankle angle at the moment of limb loading and a second, more prolonged decrease of similar magnitude at the end of stance.

The knee displayed 2 arcs of motion (Figure 15-23I). Neutral extension was present at IC, which promptly increased to hyperextension in loading response. This posture became maximal at the end of mid stance (20°). Then the knee rapidly returned to neutral by pre-swing. No additional flexion was gained in initial swing. Once the foot cleared the floor at the onset of mid swing, the knee flexed to a delayed peak posture of 30°. During terminal swing, the knee again extended to neutral.

Figure 15-23. (D) Terminal stance with a fully extended knee, excessive ankle PF, and excessive heel rise. The thigh has a trailing alignment. The other limb also has excessive ankle PF.

Figure 15-23. (E) Pre-swing lacks knee flexion. Heel rise persists, yet the ankle is only mildly plantar flexed. Weight appears to be primarily on the contralateral limb.

Figure 15-23. (F) Initial swing totally lacks knee flexion and thigh advancement. The ankle is excessively plantar flexed. Toe drag occurs despite pelvic hiking.

Figure 15-23. (G) Mid swing hip and knee flexion are equal and not excessive. A toe drag from the plantar flexed ankle is avoided by contralateral vaulting.

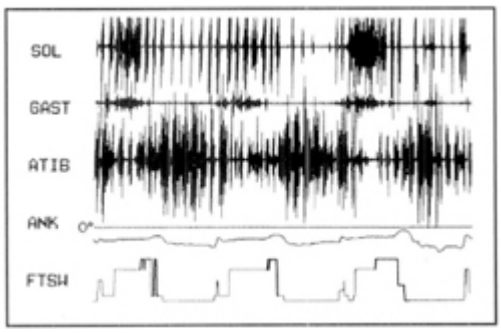

Figure 15-23. (H) Dynamic EMG and motion of the ankle. SOL = soleus; GAST = gastrocnemius; ATIB = anterior tibialis; ANK = ankle electrogoniometer; FTSW = footswitches for that limb.

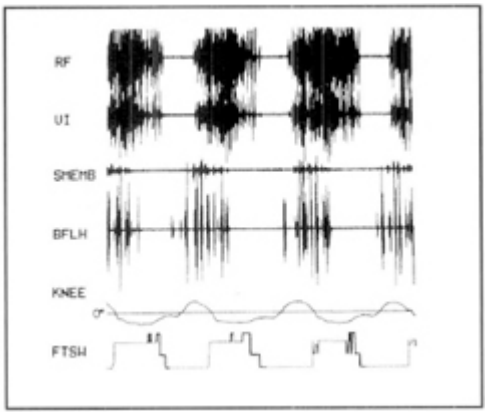

Figure 15-23. (I) Dynamic EMG and motion of the knee. RF = rectus femoris; VI = vastus intermedius; SMEMB = semimembranosus; BFLH = biceps femoris long head; KNEE = knee electrogonio meter; FTSW = footswitches for that limb.

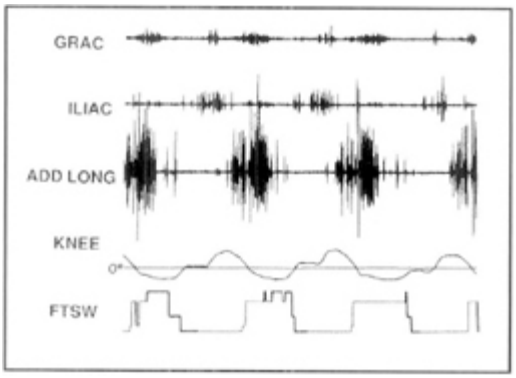

Figure 15-23. (J) Dynamic EMG and motion of the knee. GRAC = gracilis; IL = iliacus; ADD LONG = adductor longus; KNEE = knee electrogoniometer; FTSW = footswitches for that limb.

The EMG records demonstrated severe soleus muscle spasticity with clonic bursts persisting through swing as well as stance (see Figure 15-23H). This spastic action was superimposed on a primitive control pattern (dense, continuous EMG) with premature onset at the beginning of stance. The significantly diminished gastrocnemius EMG activity (primitive phasing but no clonus) implied limited protection of the knee from the hyperextension thrust. Continuous, high-intensity TA EMG throughout the GC implied strong DF action. The muscle's small size compared to that of the soleus (20%) made the TA an ineffective dorsiflexor in swing despite this being the period of major EMG.

EMG recordings of the knee muscles showed intense, premature, and prolonged action of the knee extensors (VI, RF) from the onset of terminal swing through terminal stance (see Figure 15-23I). While the VI beginning in terminal swing was normal, its intensity was excessive. Continuation beyond loading response was abnormal, and activity in the presence of a hyperextended posture was very inappropriate. In addition, the RF functioned totally out of phase and displayed excessive intensity. The intense, spastic action of the BFLH and the lesser action of the semimembranosus between mid swing and mid stance attempted to assist knee flexion in swing. At the same time, the hip extensor action of these hamstring muscles was antagonistic to thigh advancement, a critical element of swing phase knee flexion. In stance, contrary to expectations, the hamstrings offered no prolonged resistance to the knee hyperextension thrust imposed by the intense quadriceps action. Nor did the BFSH alter its action from swing to stance to provide protection.

Hip flexor control was provided by 3 muscles. It began in initial swing with the iliacus (Figure 15-23J). Low-level gracilis action was a useful synergist. Dynamic hip flexion was continued between terminal swing and mid stance by intense and excessive adductor longus action.

In conclusion, this boy's knee recurvatum resulted from excessive action of a highly spastic soleus and quadriceps. Hip flexor control would have been adequate if the limb had been unlocked. The adductor longus served as a useful swing phase hip flexor but likely

contributed an undesirable scissoring effect. The limitation of swing knee flexion related to 2 EMG findings. By their hip extensor effect, premature hamstring muscle action during initial swing inhibited hip flexor momentum. Also, the primary hip flexor action by iliacus, gracilis, and later the adductors longus provided an insufficient counter force. Intense RF action in mid swing inhibited knee flexion by its knee extensor effect and the less intense and smaller BFSH was an ineffective antagonist. The seemingly random mixture of appropriate, ill-timed, and out-of-phase muscle action illustrates the reasons why one cannot predict muscle activity in the spastic patient with impaired neural control.[31]

Figure 15-24. Patient Q presents with a stiff knee gait arising from spinal cord injury. (A) IC by the heel with normal limb alignment.

Figure 15-24. (B) Loading response lacks knee flexion. Otherwise limb posture appears normal.

Figure 15-24. (C) Terminal stance has continued heel contact. The ankle is dorsiflexed and the knee is extended. Step length is shortened by the body center remaining over the foot.

Figure 15-24. (D) Pre-swing knee flexion is lacking. The ankle is excessively dorsiflexed. There is good heel rise and a trailing thigh.

SPINAL CORD INJURY: STIFF KNEE GAIT

The unique characteristic of spinal cord injury is that there are 3 types of function. Control is normal above the level of the lesion. This offers the patient maximum substitution capability. A lower motor neuron lesion occurs at the level of the cord damage. Flaccid paralysis occurs within these segments. Distal to the site of injury, the impaired control is that of an upper motor neuron lesion. Now function represents an individual mixture of spasticity, impaired selective control, and primitive locomotor patterns. While spinal cord injuries tend to be bilateral, the functional involvement on the 2 sides can differ markedly both in severity and the neural tracts included.

Patient Q: Spinal Cord Injury (Figure 15-24)

This individual, who had experienced a spinal cord injury, had regained a community level of ambulation (velocity = 37 m/min, 42% normal). His gait, however, was significantly limited by inadequate knee flexion during both limb loading and swing.

IC was normal (Figure 15-24A). As the heel contacted the floor, the forefoot was well elevated by the neutrally aligned ankle, extended knee, and flexed hip. During loading response, the knee failed to flex (Figure 15-24B). This extended knee position continued through single stance. In terminal stance, the patient displayed prolonged heel contact (Figure 15-24C). Progression was gained by increasing ankle DF. Pre-swing knee flexion was severely limited, although unloading the limb allowed a late heel rise (Figure 15-24D). Initial swing knee flexion was very limited and there was poor advancement of the thigh (Figure 15-24E). Toe drag was avoided by a slight lateral trunk lean and ankle DF to neutral. In mid swing, floor clearance was gained by the well dorsiflexed ankle (Figure 15-24F). Hip flexion was notably greater than knee flexion. Hence, there was excessive knee extension. Terminal swing showed good limb control with selective ankle DF while the knee was extended (Figure 15-24G).

Motion analysis showed the primary functional deviations involved both the knee and ankle (Figure 15-24H). Loading response lacked the normal PF. Instead, beginning from a neutral position at IC, the ankle progressively dorsiflexed through the weight-bearing period until a peak angle of 15° was reached in late terminal stance. Slightly excessive DF (10°) was maintained through initial and mid swing. The ankle then dropped to neutral in terminal swing.

Figure 15-24. (E) Initial swing knee flexion is very limited and the ankle is dorsiflexed to neutral (excessive). Contralateral trunk lean assists foot clearance.

Figure 15-24. (F) Mid swing knee flexion is inadequate (less than hip flexion).

Figure 15-24. (G) Terminal swing limb posture appears normal. The foot is higher than normal because the other limb has not rolled body weight onto the forefoot rocker.

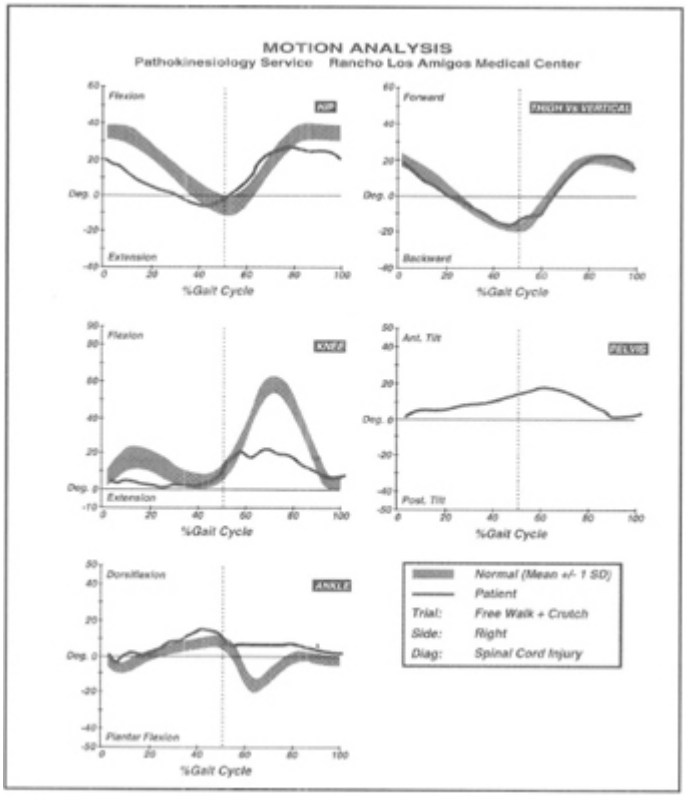

Figure 15-24. (H) Motion analysis. The vertical axis is in degrees of motion (flexion is positive). The horizontal axis is % GC. The vertical dotted line divides stance and swing (toe-off). The gray area indicates the one standard deviation band of normal function. The black line is the patient's data.

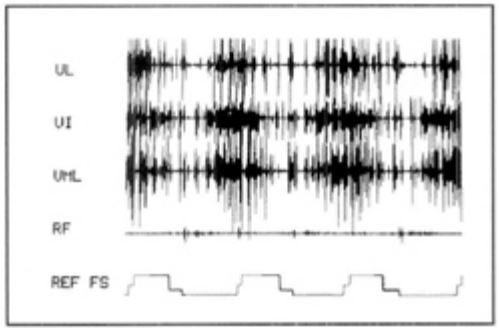

Figure 15-24. (I) Dynamic quadriceps EMG. VML = vastus medialis longus; VI = vastus intermedius; VL = vastus lateralis; RF = rectus femoris; FTSW = reference limb footswitches.

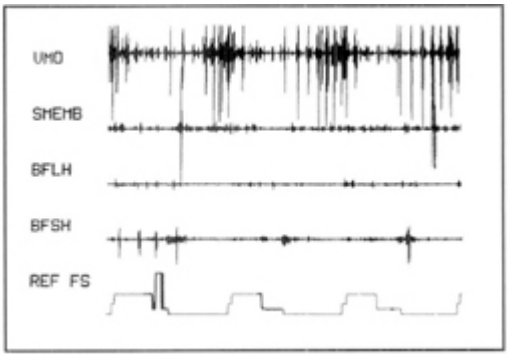

Figure 15-24. (J) Dynamic EMG. VMO = vastus medialis oblique; SMEMB = semimembranosus; BFLH = biceps femoris long head; BFSH = biceps femoris short head; FTSW = reference limb footswitches.

Knee motion was independent of the ankle. The extended position (5° flexion) at IC was maintained through the stance phases until pre-swing. Knee flexion began in this double support period, reaching 20° by the onset of initial swing. Further flexion during initial swing was minimal (5° additional motion). Beginning in mid swing, the knee extended to a terminal position of 5° flexion.

The thigh followed a normal pattern throughout the stride while there was a progressive anterior pelvic tilt in stance. Hip flexion at IC was limited (20°). From this point, the joint progressively extended, reaching slight hyperextension (5°) shortly before an early toe-off.

The footswitches showed a brief heel support interval (H), which quickly progressed to foot flat (H-5). Heel contact was maintained until the other foot was loaded. Toe-off was premature, occurring at 54% GC (versus the normal 62% GC). This is related to a short single stance time (27% versus 40% GC). The other limb had an extended SLS (49% GC). These differences reflected limited progression in stance and difficulty advancing the limb in swing.

Dynamic EMG of the muscles about the knee showed continuous activity of all the vastii. It was most marked in the vastus medialis longus (VML) and VI (Figure 15-24I). While the intensity was greater during stance, there also was highly significant extensor muscle activity in swing. Conversely, there was no RF action in either gait period. A brief burst of BFSH action occurred at the onset of IC, but the muscle was too small to counter the multiple vastii (Figure 15-24J). There also was no significant hamstring activity to decelerate

knee extension in terminal swing or to assist the heel rocker extensor thrust in loading response.

Thus, a fully extended limb, locked by continually active vastii, rolled forward across the ankle during stance. Excessive DF substituted for a forefoot rocker. This signified gastrocsoleus muscle weakness. An erect trunk posture was maintained by an anterior pelvic tilt compensating for restricted hip mobility (25° versus the normal 40° arc).

SUMMARY

Purposeful and efficient walking relies on an exquisite, seamless interchange of information (signals) between the sensory and motor systems. Disruption of neurologic pathways, by either disease or trauma, alters control. Intensive rehabilitation, aimed at not only minimizing impairments but also promoting neuroplastic changes of the nervous system, offers promise for improving walking in those with neurologic injury.

AMPUTATIONS

Limb loss from disease or trauma is part of mankind's history.[147] Development of prostheses to replace lower limb loss, however, progressed slowly. Amputations were infrequent except in wartime, and survival from any serious injury was poor. Thus, progression beyond a mere peg-leg extending from a socket or platform was slow.

During World War II, the high rate of casualties combined with an improved survival rate created a great need for prostheses to replace the soldiers' lost limbs. The available leather and wood devices, however, were lacking in comfort and effective function. Thus, in 1945, the National Academy of Science authorized a multicenter research and development program to define the scientific basis of prosthetic design and its application to care of individuals with an amputation.[147]

One such center was the Prosthetic Devices Research Project at the University of California, Berkley. This group of clinicians, engineers, and anatomists conducted the first comprehensive study of walking, prosthetic design, and residual limb care. The success of this collaborative effort introduced the field of modern prosthetic research and development.

The remainder of this chapter focuses on the impact of transtibial and transfemoral amputations on walking function. Patient-specific considerations as well as the benefits and limitations of prosthetic foot/ankle and knee designs will be highlighted.

Transtibial (Below Knee) Amputation

Preservation of the knee affords individuals with a transtibial amputation greater functional potential and efficiency than if the limb is severed at the transfemoral level.[145] When both lower limbs are affected by amputation, a not uncommon occurrence with diabetes or peripheral vascular disease, the functional importance of maintaining the knees becomes even more apparent.[142] An analysis of outcomes following intensive prosthetic rehabilitation delivered between 1970 and 1982 revealed that nearly 50% of individuals (10 of 21) with bilateral transtibial amputations were able to walk independently at a community or limited community level following rehabilitation. However, none of 17 individuals with bilateral transfemoral amputations were ambulatory in the community. Although one person with bilateral transfemoral amputations was able to walk for limited distance in the house, 8 people relied on a wheelchair for mobility and the remaining were bedridden. Recent advances in prosthetic componentry have substantially improved functional outcomes following bilateral transfemoral amputations.[86,103] However, the earlier findings highlight the critical role of preserving the knee.

Prosthetic prescription today is based on 3 levels of analysis. First is a clinical assessment of the individual's physical and life style needs. Second is the selection of the optimum prosthetic foot design for the individual's functional needs.[72] Third, is the determination of

the quality of the gait accomplished by the patient with the selected prosthesis and alignment.[151]

Design of the prosthesis also must meet the challenges presented by the 3 basic tasks of walking.[99] Specifically, the WA heel rocker is challenged by the need to absorb shock and allow forward progression without an intact ankle joint. SLS requires stability in the sagittal and frontal planes as body weight transfers over the ankle and forefoot rockers. SLA depends on elastic recoil propulsion from the toe rocker and uninhibited momentum.

Table 15-4

Lower Limb Prosthetic Design Classification

Transtibial	*Transfemoral*	
	Swing Control	Stance Control
Articulated Single axis Multi-axis	Friction	Friction
Passive SACH SAFE	Hydraulic	Polycentric
Dynamic response Dynamic keel Dynamic pylon	Pneumatic	Hydraulic
Computerized	Computerized	Computerized

In anticipation of these demands, the designers of prostheses continually seek new materials with more appropriate flexibility, elasticity, and strength. Surface contouring is supplemented with malleable metals and plastics that can store and release energy as body alignment alters their shape.[46,140] Today, there are many prostheses with similar configurations, yet each design has specific functional characteristics that could be an advantage or disadvantage for the individual who has experienced an amputation.[28] Clinicians responsible for prosthetic prescriptions must be aware of the difference among a multitude of prostheses on the market. When analyzing prosthetic gait, clinicians need to be able to

differentiate the impact of patient's weakness[114] and contractures[92] from the limitations imposed by prosthetic design choice.[99]

Transtibial Prosthetic Componentry

In 1945, the only available ankle joint was a single axis articulation between the shank and foot. The common disadvantages were frequent prosthetic breakdowns and excessive weight. By 1955, after extensive efforts failed to solve the problems, engineers pursued a new approach in prosthetic design.[49] The articulated joint was replaced with a unitized structure that integrated a solid ankle cushioned heel (SACH) foot into a single structure.[28,48] This structure became the basic model for future prosthetic designs seeking to reproduce normal limb function. The emergence of materials that could store and release energy stimulated new, more mobile unitized designs classified under the umbrella term as *"dynamic elastic response" feet*.[108] Each new product has offered some functional gains but also included new disadvantages. The underlying challenge has been the need to replace the selective actions of human muscles and tendons with contoured wood, flexible metals, plastics, and polymers. Most recently, microprocessors have been introduced as a source of selectivity for ankle control.[9]

When prosthetic development was a small program, clinicians could identify each design by name. The vast array of devices currently available has necessitated development of a preliminary classification system that will be used in this chapter (Table 15-4).[87] Current transtibial prosthetic designs fall into 4 classes: articulated, passive, dynamic response, and computerized.

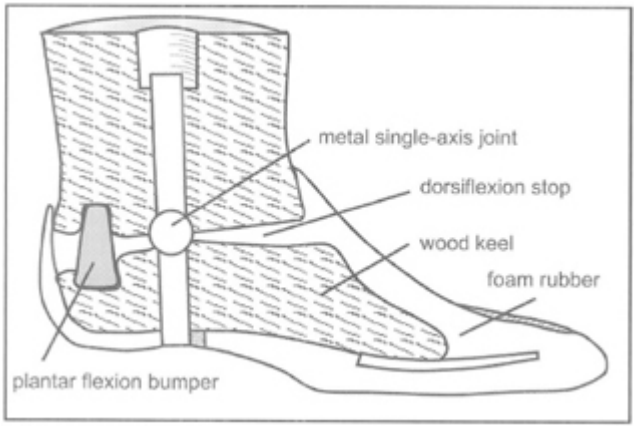

Figure 15-25. Articulated prosthetic foot.

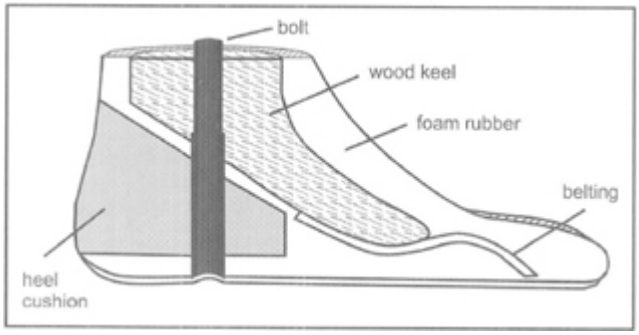

Figure 15-26. Passive prosthetic foot.

Articulated

Two segments, the foot and shank, are joined by a mobile joint (ie, the ankle). Motion at the joint is limited by bumpers (Figure 15-25). Two prosthetic designs are included.

1. Single axis: This ankle allows only DF and PF.
2. Multi-axis: This more recently developed design adds small arcs of medial-lateral and rotational motion to the DF and PF.

Although the greater ability to accommodate uneven terrain is a benefit to farmers and hikers, the weight and limited durability of the units are disadvantages compared to unitized foot designs.

Passive

The foot and ankle are integrated into a single structure (Figure 15-26). There are 2 models that differ in flexibility.

1. SACH Foot: This is virtually rigid.[49] A wooden keel replaces the ankle joint. Compression of the large cushioned heel reduces

the impact of initial floor contact. The combination of a curved keel, contouring of the plantar surface, and slight yielding of the dense plastic foam forefoot aids progression across the foot. This relatively rigid prosthesis has one notable disadvantage. Walking at a fast speed is difficult.

2. SAFE Foot: By 1978, the availability of more flexible materials allowed an effective challenge to the dictum that stability required rigidity. A keel composed of highly resilient rubber replaced the customary wooden model. The forefoot was made of more flexible foam segments that were interspersed between wooden blocks. This prosthesis permits better sagittal progression, some medial and lateral mobility, and improved stance stability.

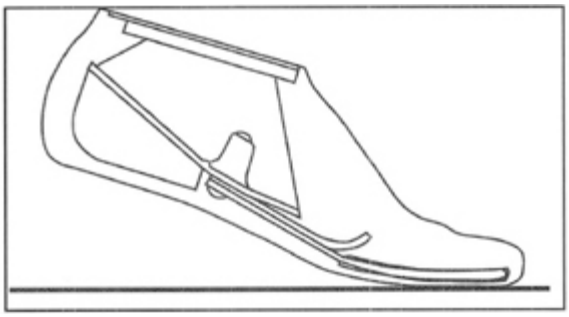

Figure 15-27. Dynamic response keel prosthetic foot.

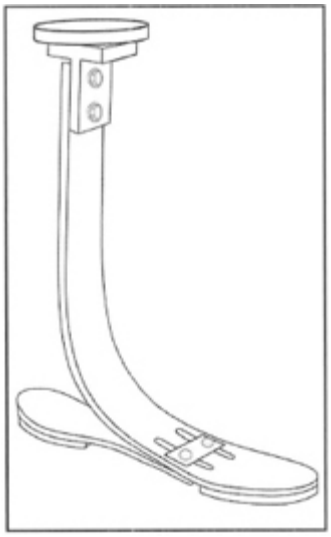

Figure 15-28. Dynamic response pylon prosthetic foot.

Dynamic Response

When materials with “elastic energy storage and release” qualities were first introduced, all the potentially energized prosthetic designs were put under the dynamic elastic response (DER) umbrella. The first designs involved just the foot. Flexibility was improved and stability was preserved. New designs, which extended integration of the dynamic materials from the socket to the base of the toes (pylon and keel), generated identifiable power.[46] It had been suggested that the term *dynamic response* be restricted to the better power-producing prostheses. This, however, introduced confusion from conflicting literature. It seems more appropriate to retain the 2 subdivisions under dynamic response:

1. Dynamic keel: The prosthetic foot is fitted with an energy-producing keel (Figure 15-27). A series of leaf springs replaced the rigid keel.[21,87] The initial goal was to energize the foot for running.[21] The added flexibility gives individuals with an amputation the freedom to walk more efficiently at faster speeds.[94] This design has good durability and a low cost. Subsequent investigators have advanced the flexible keel concept with a variety of foot designs.[59]
2. Dynamic pylon: This prosthesis was designed to generate “push-off” energy. A dynamic segment was created by replacing the rigid pylon and keel with a flexible element that extended from the socket to the base of the toes (Figure 15-28). Notable tension develops as the advancing body mass flexes the shaft and then is abruptly released by toe-off. The reaction generates a pre-swing burst of power exceeding 50% of normal.[46] During gait, lower sound limb loading forces were recorded when using a dynamic pylon foot design (Flex foot, Össur Americas, Aliso Viejo, CA) compared to 4 other nondynamic pylon feet (110% versus 128% to 135% body weight).[115] Terminal stance DF was greater using the Flex foot than the other designs (23° versus 12° to 20°).[102] This suggests that the Flex foot may more effectively control forward fall of the COG prior to IC. The importance of minimizing sound

limb loading forces is underscored by the high incidence of osteoarthritis and knee joint pain documented in the sound limb of long-time prosthetic users.[43]

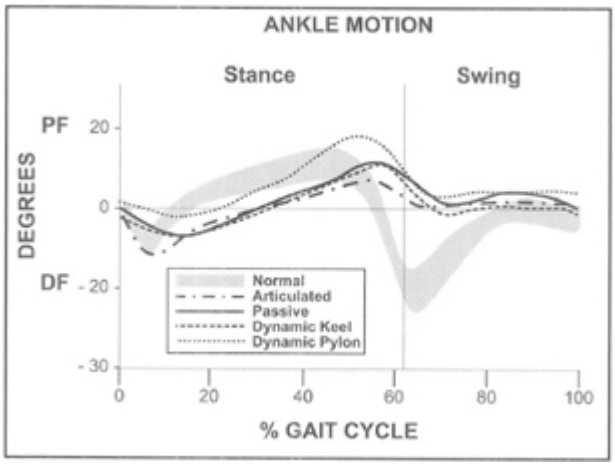

Figure 15-29. Comparison of transtibial prosthetic ankle motion while walking using the single-axis, dynamic response keel (Seattle Lightfoot) and dynamic response pylon (Flex foot) to that recorded in individuals without amputation. (Adapted from Perry J, Boyd LA, Rao SS, Mulroy SJ. Prosthetic weight acceptance mechanics in transtibial amputees wearing the Single Axis, Seattle Lite and Flex Foot. *IEEE Transactions on Rehabilitation Engineering*. 1997;5(4):283-289.)

Computerized

Recently, microprocessors have been introduced that provide direct control of the prosthetic ankle joint.[9] The prototype includes a carbon leaf spring dynamic response foot and ankle system. An electronic mechanism controls the magnitude and timing of the forces and power produced by the prosthetic ankle.

Comparison of Dominant Functional Characteristics of Prosthetic Foot Designs

The natural progression in the design of transtibial prostheses led to the 4 proposed generic categories. A previous study, which quantified the patterns of ankle motion performed during a GC, provided a measure of the way different prosthetic foot/ankle designs approximated normal ankle function.[102]

The level of prosthetic function was identified by the magnitude and duration of the ankle's motion pattern during 3 events: 1) the heel rocker, 2) SLS (ie, the ankle and forefoot rockers), and 3) the toe rocker. Specific effectiveness of each prosthetic class can be defined by the similarity of prosthetic motion to normal movement patterns.

Transfer of body weight to the leading limb is normally smoothed by a small arc of ankle PF (8°) during the first 7% of the GC (Figure 15-29).[102] The ankle then reverses movement direction and achieves a footflat pattern by 12% of the GC. The resulting impact force ranges between 50% and 125% of body weight.[127]

The articulated prosthesis' response to the impact of initial floor contact was 12° of gravity-induced PF (see Figure 15-29).[102] The foot drop most closely paralleled the normal rate but the slow response of its posterior rubber bumper prolonged the motion and allowed the heel lever to fade. WA PF in passive, dynamic keel, and dynamic pylon prostheses was less than half the normal arc, and the reversal of the ankle toward DF was slightly delayed. Consequently, DF progression by these 3 prosthetic designs was diminished during WA.

Table 15-5

Foot-Floor Contact Patterns During Weight Acceptance

	Foot Flat (% Gait Cycle)	*Contralateral Toe-off (% Gait Cycle)*
Single axis foot	17	17
Dynamic response keel (Seattle Lightfoot)	21	16
Dynamic response pylon (Flex foot)	19	16
Foot (no amputation)	12	12

Adapted with permission from Perry J, Boyd LA, Rao SS, Mulroy SJ. Prosthetic weight acceptance mechanics in transtibial amputees wearing the Single Axis, Seattle Lite and Flex Foot. *IEEE Transactions on Rehabilitation Engineering.* 1997;5(4):283-289.

The examination of variations in foot-floor contact patterns provides insights into the impact of each prosthetic design on WA stability (Table 15-5). The articulated prosthesis reached the stability of footflat contact slightly before contralateral toe-off. However, neither dynamic response prosthesis accomplished this level of security. Instead, the prosthetic limb still was balancing on the heel for a notable period even after the contralateral limb had lifted from the ground (see Table 15-5).

During SLS (see Figure 15-29), the dynamic response pylon yielded into approximately 15° DF in late stance, exceeding motion documented in those without amputation (~12° DF). Forefoot rocker DF was notably curtailed using the single axis and dynamic response keel feet (~ 5°). During the toe rocker, only the nonprosthetic condition demonstrated the normal rapid arc of PF (see Figure 15-29).

Flexibility of materials within the body of a unitized prosthetic foot determines the device's power generation capability. Elastic tension is absorbed as weight rolls across the forefoot during terminal stance. Then, as body weight abruptly transfers to the lead limb during pre-swing, stored energy is released. In a comparison of 3 representative prosthetic foot designs, ankle power generation during the pre-swing toe rocker was found to vary (Figure 15-30).[46] The passive prosthesis (SACH) generated a power that was only

10% normal. The power of the dynamic keel prosthesis (Seattle, Model & Instrument Works, Inc, Seattle, WA) was 25% normal, while the dynamic pylon (Flex foot) attained 61%. The clinical significance of ankle power in pre-swing has been the subject of considerable debate for many years. Recent studies, utilizing ultrasound technology, have clarified the role of the plantar flexors in releasing energy that helps advance the limb during this final period of stance.[16,42,60,64,80] While less than normal, the greater power-generating capability of the dynamic keel and pylon prostheses compared to the passive foot suggests potential advantages for replacing lost ankle function.[46]

Patient R: Transtibial (Below Knee) Amputation (Figure 15-31)

This experienced walker used a dynamic keel prosthetic foot (ie, the Quantum Foot, Hosmer Dorrance Corp, Campbell, CA). He retained normal hip and knee function following his amputation.

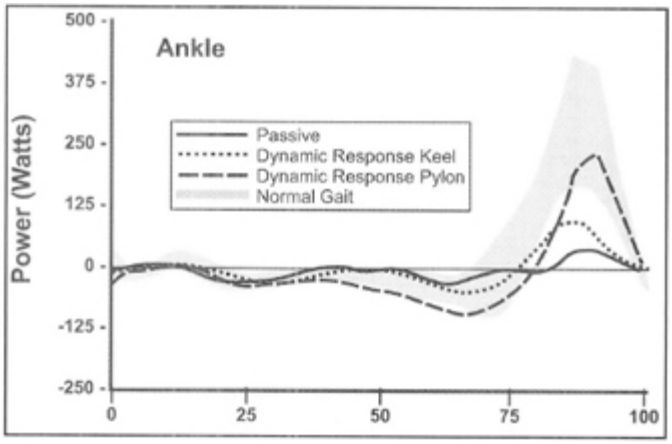

Figure 15-30. Comparison of prosthetic ankle power generation while walking using the passive (SACH), dynamic response keel (Seattle Lightfoot) and dynamic response pylon (Flex foot) to that recorded in individuals without amputation. (Adapted from Gitter A, Czerniecki JM, DeGroot DM. Biomechanical analysis of the influence of prosthetic feet on below-knee amputee walking. *Am J Phys Med*. 1991;70:142-148.)

Figure 15-31. Patient R had a transtibial amputation. (A) IC with normal limb alignment.

Figure 15-31. (B) Loading response with normal knee flexion and ankle PF.

Figure 15-31. (C) Mid stance with slight flexion at knee and hip and ankle DF.

Figure 15-31. (D) Terminal stance with a low heel rise and excessive ankle DF provides good progression.

IC was made by the heel with the limb normally postured (Figure 15-31A). Loading response displayed normal knee flexion and ankle PF. Hip flexion was maintained (Figure 15-31B). This loading pattern implied that the prosthetic foot's cushioned heel compressed to simulate PF and reduced loading impact. Halfway through mid stance, the ankle was in slight DF while both the knee and hip were minimally flexed (Figure 15-31C). The trunk was over the midfoot and the other foot was slightly trailing. Terminal stance had a low heel rise, excess ankle DF, full knee extension, and a trailing thigh position (Figure 15-31D). Hence, there was good prosthetic tibial control to provide an extended knee. At the same time, the prosthetic ankle yielded for optimum progression and step length. By the end of pre-swing, the knee had normal flexion although the thigh had not advanced enough to attain a fully vertical position (Figure 15-31E). This implied direct knee flexor action as a supplement to a limited prosthetic toe rocker. During initial swing, the knee achieved adequate flexion; however, the thigh failed to advance sufficiently (Figure 15-31F). The tibia reached vertical in mid swing; however, foot clearance was excessive due to greater-than-normal thigh flexion (Figure 15-31G).

Motion analysis showed a relatively normal pattern at each joint (Figure 15-31H). The ankle, beginning in neutral alignment at IC, plantar flexed approximately 10° in loading response. From this position, the change toward DF was initially abrupt but then slowed during mid stance followed by accelerated DF in terminal stance to 20°. Throughout swing, the ankle was at neutral (0°). The knee advanced from full extension (0°) at IC to 20° flexion by 18% GC.

This was followed by progressive extension, reaching 4° hyperextension at 40% GC. There was then a sharp reversal into flexion during the last portion of terminal stance, which continued through pre-swing and initial swing. The timing of peak knee flexion was normal (72% GC) but the magnitude was excessive (73°). Extension of the knee reached 2° hyperextension just before IC (95% GC). The thigh had a delayed onset of progressive extension during loading response but then achieved the normal 20° hyperextension expected in terminal stance. Subsequent thigh flexion was normal in early swing but excessive (30°) by mid swing.

Figure 15-31. (E) Pre-swing thigh advancement and ankle PF are less than normal, yet there is adequate knee flexion. This implies increased knee flexor muscle activity.

Figure 15-31. (F) Initial swing showing normal knee flexion but limited thigh advancement.

Figure 15-31. (G) Mid swing elevation of the foot is slightly excessive.

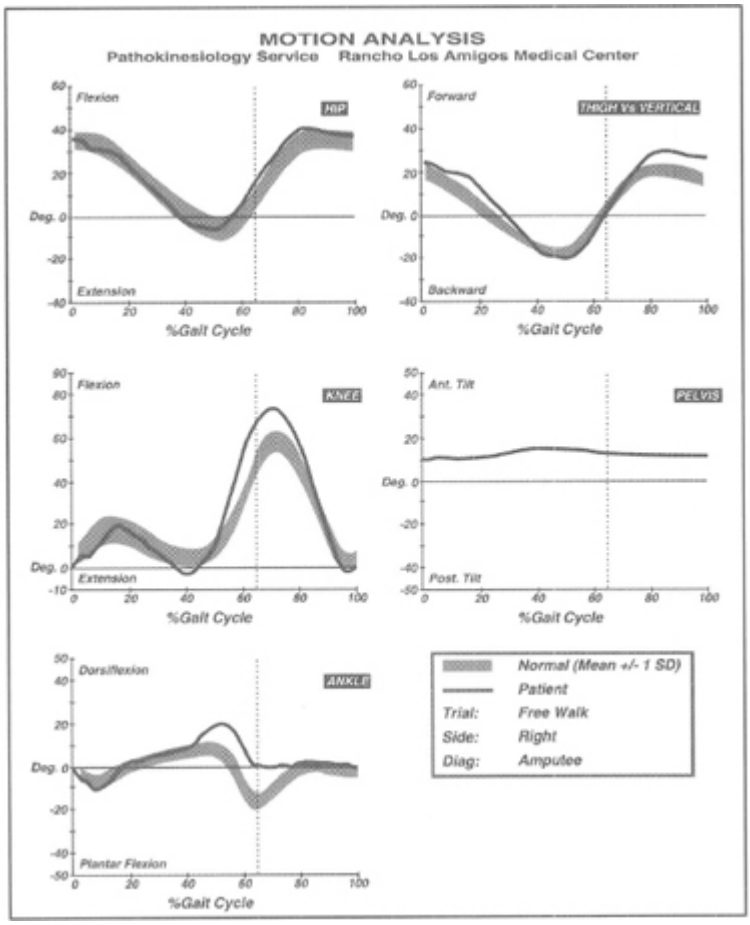

Figure 15-31. (H) Motion analysis. The vertical axis is degrees of motion (flexion is positive). The horizontal axis is percentage of the GC. The vertical dotted line divides stance and swing (toe-off). Gray areas indicate the one standard deviation band of normal motion. Black line is patient data.

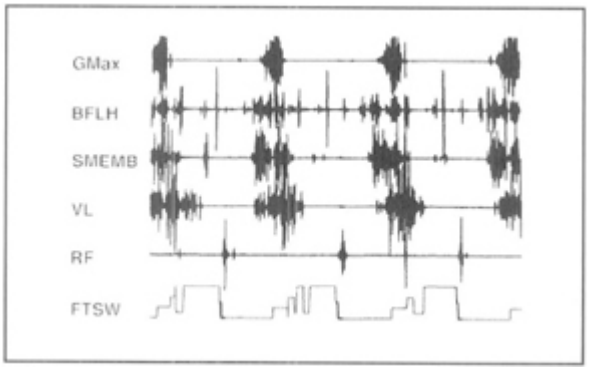

Figure 15-31. (I) Dynamic EMG. GMax = gluteus maximus; BFLH = biceps femoris long head; SMEMB = semimembranosus; VL = vastus lateralis (quadriceps); RF = rectus femoris; FTSW = footswitches.

EMG recordings of his key muscles showed increased intensity and some prolongation of action. At the hip, the gluteus maximus intensity was prematurely high as its activity accompanied a vigorous semimembranosus (Figure 15-31I). The BFLH had prolonged action. This muscle pattern implied a strong thigh retraction effort to stabilize the hip during loading response. Strong quadriceps action (VL) was appropriate for stabilizing knee flexion during early stance. The brief period of RF action as the foot lifted from the ground decelerated the overly rapid knee flexion that accompanied the accelerated ankle DF and heel off.

The patient's gait velocity was normal (93 m/min). His footswitch pattern showed rapid progression from the heel (H), across a brief footflat interval (H-5), onto sustained heel-off support (5-1). Thus, this physically fit man attained a gait that closely simulated normal function by modifying his muscle action to compensate for the small abnormalities in prosthetic function.

In summary, this patient displayed a strong quadriceps, being capable of accepting the added loading torque imposed by the limited mobility of the prosthetic foot. During the rest of stance, however, the quadriceps were protected by the tibial stability offered by the dynamic elasticity of the prosthetic "ankle."

Transfemoral (Above Knee) Amputation

An amputation at any level of the thigh removes the patient's knee joint, all direct control of knee extension (quadriceps), and a significant length of the femur. The gluteus maximus remains, but hip extension is weakened by loss of the distal insertion of the hamstrings and adductor magnus (even if myodesis or myoplasty is performed). Despite preservation of the gluteus medius and minimus, the hip abductor force is diminished by loss of the tensor fascia latae's distal insertion. Although the remaining proximal muscle fibers of the TFL may spontaneously reattach to bone, they will be weaker due to their reduced force-producing capacity in the slack reattached position.[50,143] Loss of the ankle and foot imposes the prosthetic restrictions already described under the transtibial amputation section.[86] Thus, the significant remaining muscles are the iliacus for flexion, the gluteus medius and minimus for abduction, and the gluteus maximus as the primary extensor.

A firm fit of the prosthetic socket encloses the residual thigh and enables forces generated by the hip muscles to control the prosthetic knee joint during swing and stance.[65] SLS ends with the limb in a trailing position. As few prosthetic feet have significant push-off power, flexion of the knee and advancement of the limb depends on flexibility of the prosthetic toe break and hip flexor muscle strength. During pre-swing, substantial unloading of the prosthetic limb needs to occur before the prosthetic knee is able to flex. Swing involves a voluntary "crack-the-whip" movement. The sequence begins with rapid, exaggerated hip flexion to lift the limb, flex the knee, and advance the foot. Prosthetic knee control (eg, a check strap) may be required to replicate the restraint previously provided by the RF muscle during pre-swing. Then, in the latter half of swing, a brief contraction of the gluteus maximus for fast hip extension (ie, a past retract) combined with inertia of the ankle-foot segment extends the knee. Floor contact by the heel anchors the foot, while continued action by the gluteus maximus ensures full knee extension during stance to avoid knee buckling.

Few individuals have sufficient strength, keenness of proprioception, and motor control acuity to avoid falls when they fail to reach the full knee extension required for stance stability. Consequently, a series of knee control devices have been designed. Friction disks were created to permit matching the rate of prosthetic knee flexion and extension to the sound limb and to the needs of a specific or varied cadence. Subsequently, polycentric, hydraulic, and pneumatic knee units were designed to offer control that was further refined.[13,87] Stance stability was more easily attained. The introduction of microprocessors to modulate hydraulic swing control represented another advance. The inclusion of microprocessors to control both stance and swing significantly improved walking stability.[14,69]

Transfemoral Prosthetic Knee Componentry

The significant amount of effort involved in artificial swing and the insecurity of SLS have led to the development of more than 100 types of prosthetic knee controls. Generic classification, however, has reduced the list for swing and stance to 4 categories: friction, hydraulic, pneumatic, and computerized (see Table 15-4).[87]

Swing Control

Mechanical designs to restrain the rate of swing initially used friction. Hydraulic, pneumatic, and computerized systems for modulating knee flexion evolved to address the more demanding needs of individuals capable of walking at a variety of cadences.

* Friction: Cadence control is the common purpose of friction. Constant friction is the simpler system.[87] The knee joint is fitted with a rubber bumper to dampen mobility so that the cadence of the prosthetic knee matches the rate of the sound knee. Constant friction units are considered appropriate for children and amputees with good muscle control who do not change their cadence. It is not indicated for a person with

weak hip flexors, poor balance, or expected to vary cadence frequently. Variable friction joints have a series of friction pads that adjust to changes in cadence. Friction knee joints are light weight and require little maintenance.

* Hydraulic: Knee control during swing by a hydraulic system is preferred over constant friction because a smoother gait is gained and multiple cadences are accommodated. Hydraulic pistons use oil,[87] which is virtually incompressible and provides a smoother action compared to friction and pneumatic mechanisms. Extremely cold temperatures may thicken the fluid in the hydraulic knee and alter function.
* Pneumatic: Air fills the pneumatic pistons. This makes them lighter than hydraulic pistons, but it also creates a little bounce. Pneumatic systems are less sensitive to extreme temperatures compared to hydraulic controls.
* Computerized: The last category uses a microprocessor system to control the knee during swing. In 1993, the unique approach of incorporating computer control for the prosthetic knee was introduced as the “Intelligent Prosthesis.”[11] Microprocessor control of the prosthetic shank during swing provided a more normal rate of terminal knee extension as walking speed varied. Individuals with a unilateral amputation experienced a 3% to 15% reduction in energy cost during walking.[20,29,131]

Stance Control

Maintaining stance stability, particularly on uneven surfaces, is a persistent challenge for those walking with a transfemoral prosthesis. The need to switch rapidly from a stable knee during stance to a mobile knee for swing presents design challenges. While early systems used friction and dynamic prosthetic alignment to promote stability, more recent designs rely on advanced sensors to automatically regulate stability and mobility.

* Friction: A constant friction knee has a single axis hinge that allows flexion and extension. The simplest design merely aligns the GRFV anterior to the prosthetic knee joint for

stance stability.[122] If the hip extensors are weak, then the knee center is moved back to allow some hyperextension. This adds stability but makes flexing the knee more difficult as the limb has to be completely unloaded before flexion can occur. The constant friction knee has been recommended for children and small women, but it has a tendency to buckle when the knee fails to fully extend for stance and on slopes and irregular terrain.[122] The addition of a “safety knee” helps address this limitation. It contains a spring-loaded braking mechanism that locks the knee when a portion of body weight is loaded onto the prosthetic limb. The knee can be locked in up to 15° to 20° of flexion, but the unit cannot support full body weight in extreme flexion. Unweighting the limb during pre-swing releases the lock. The safety system is adjustable but is reported to lack durability.[122]

* Polycentric: A second mechanical knee design for stance control has a 4-bar linkage joint.[87] This prosthesis was designed to reproduce the change in the instantaneous center of rotation pattern identified in the human knee. The four-bar linkage knee joint improves stability of a prosthetic joint and is appropriate for long or short residual limbs. Leverage can be modified to augment weak hip extensors.
* Hydraulic: These units rely on the flow of liquid within a system of cylinders and pistons to control motion of the knee. Silicone oil is frequently used as its viscous properties are less sensitive to temperature changes than water.[122] For active, vigorous persons, one popular hydraulic knee is the Mauch Swing-N-Stance (SNS) (Össur Americas), which provides control in both swing and stance. Despite the added stability features, patients report that falls while walking are still common. One contributory factor is that the Mauch SNS stability control mechanism reportedly disengages during WA.[14] This potentially denies the patient adequate protection from prosthetic knee collapse during this period in gait.[14]
* Computerized: The Endolite Intelligent Prosthesis (Endolite, Centerville, OH), introduced in 1993, provided only swing-

control.[11] The potential advantages of this prosthesis were limited by its lack of a stance control.[11] In 1999, the “C-Leg” was introduced by Otto Bock (Minneapolis, MN). A computer microprocessor now controlled the prosthetic knee during both stance and swing.[26,83,88] A knee angle sensor and force transducers within the prosthetic shank recorded data 50 times per second and performed real-time adjustments to the hydraulic knee unit.[26,83,88] Users of the C-Leg expressed a sense of being able to walk more rapidly and with less muscle effort.[148] Studies comparing function of this processor to nonmicroprocessor-controlled knee units demonstrated that wearing the C-Leg significantly improved the pattern of stair descent,[103] rate of hill descent,[68] self-selected walking speed,[98,103,123] balance,[69] and reduced risk of falls and stumbles.[11,14,68,98]

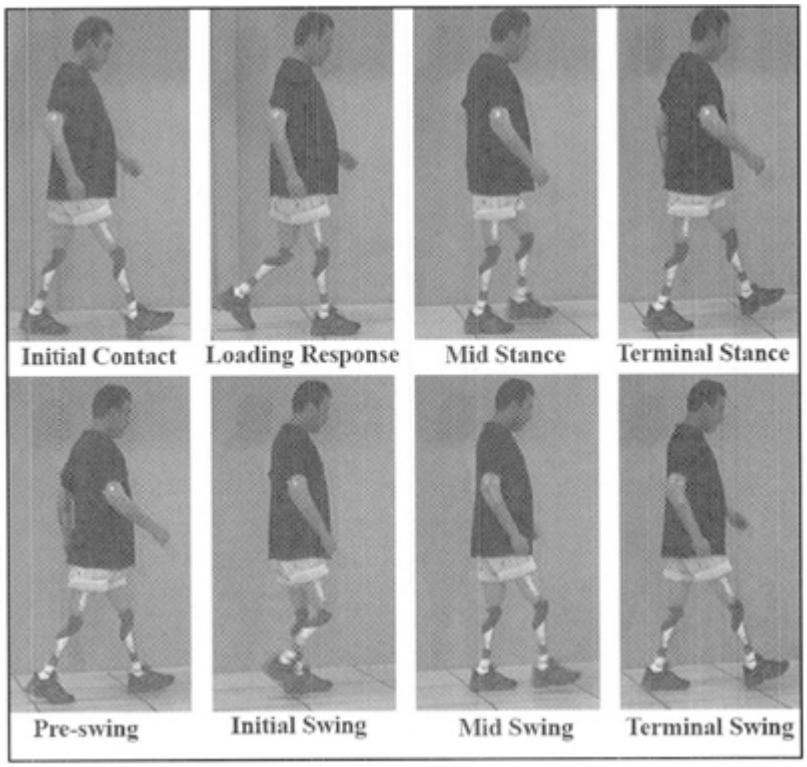

Figure 15-32. Patient S walking while using bilateral C-Leg prostheses and bilateral upper extremity prostheses. Note limited knee flexion during WA.

Patient S: Bilateral Knee Disarticulations and Transradial Amputations (Figures 15-32 to 15-34)

A single subject study of a young gentleman with bilateral knee disarticulations displayed a sharp difference in gait between mechanical hydraulic and microprocessor knee control.[103] Bilateral knee disarticulations and bilateral transradial amputations resulted from meningococcemia with purpura fulminans. While inadequate skin integrity delayed fitting the lower limb prostheses, once stabilized, he received nonarticulating prostheses (stubbies) with dynamic response keel feet, ischial-containing sockets, and supra-condylar suspension. Following 2 months of physical therapy, the individual was able to walk unaided on a level surface at 30 m/min (34% N) for 60 m, also uneven ground, ramps, and curb. One year later, full-length articulating prostheses with hydraulic knee joints (Mauch SNS), mobile ankles, and dynamic Seattle-Lite feet replaced the stubbies. After gait training, he walked independently 180 m on a level surface. Five years later, he received bilateral C-Leg 3C98 systems. Microprocessors controlled the hydraulic joints during both swing and stance. Inversion and eversion were provided by the Luxon Max feet (Otto Bock Health Care, Minneapolis, MN). He now had his preamputation standing height. Combined training sessions with the prosthetist and physical therapist helped the individual walk independently on level ground for more than 275 m (Figure 15-32) and ascend and descend curbs (Figure 15-33) and ramps (Figure 15-34) by 2 months. Stairs required 2 handrails.

After each prosthetic advance, the Pathokinesiology Laboratory documented his walking function. Marked changes in velocity were noted. Walking in the C-Leg was almost twice as fast as that of the Mauch SNS mechanical hydraulic system (71.5 versus 38 m/min) and provided a longer stride length. Walking distance also was farther with the C-Leg (1430 m compared to only 722 m in the Mauch SNS). While walking with the C-Leg, the pelvis was excessively anteriorly tilted (~15°). Motion at the hip and thigh demonstrated premature movement toward extension in WA, but

otherwise approximated normal. The knee remained in 5° to 10° flexion throughout stance and attained only 45° flexion during swing. The ankle lacked pre-swing PF. Moments and powers were significantly diminished. While the rate of oxygen cost was higher than normal for each type of prosthesis, the 20-minute oxygen cost was lowest with the C-Leg (120% N). The stubbies showed a moderate increase in energy cost (225% N) and allowed the patient to complete many of his daily activities, but he was short. The Mauch SNS system created the greatest demand for energy (304% N), which implies that greater muscle activation was required to control the prostheses.

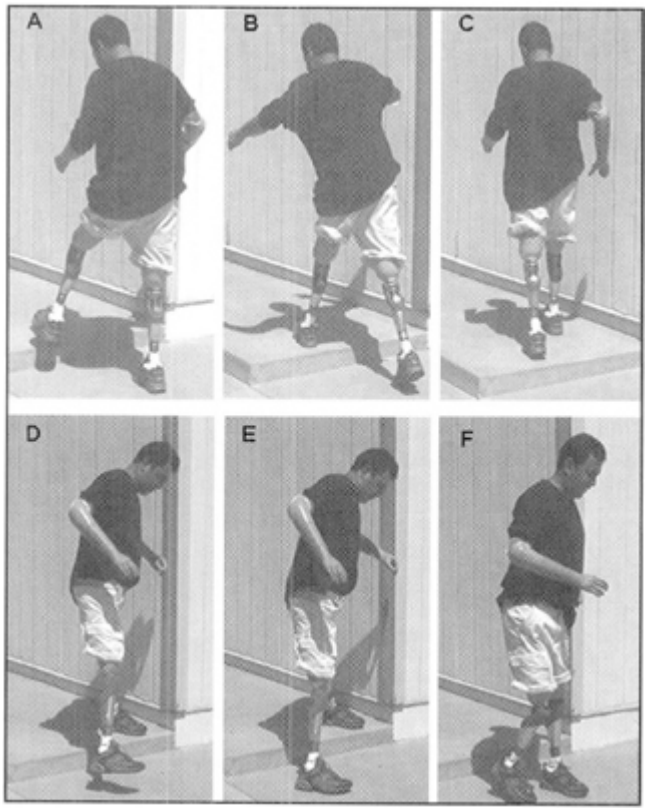

Figure 15-33. Patient S ascending (A-C) and descending curbs (D-F) while using bilateral C-Leg prostheses and upper extremity prostheses. Note the willingness to take a long step (A), and use of right trailing limb to assist with progression (B). Descending curbs was more challenging.

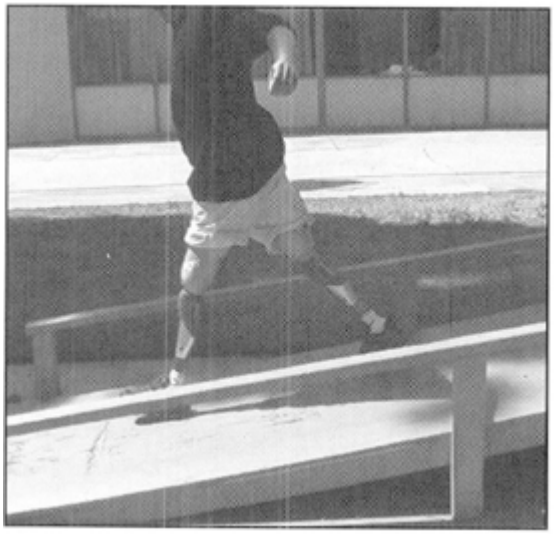

Figure 15-34. Patient S descending a ramp while using bilateral C-Leg prostheses and bilateral upper extremity prostheses. Note the confidence in taking a long step length and loading body weight onto a flexed knee during descent.

SUMMARY

Current prosthetic componentry has only partially restored walking ability. Loss of strength, sensation (plantar pressure and proprioception), joint mobility, and selective muscular control of prosthetic joints contributes to challenges with stability, progression, and efficiency.[34] Recent design advances, including microprocessor-controlled joints, offer promise for restoring greater function and independence.

CONCLUSION

Observational gait analysis combined with a thorough clinical evaluation provides a strong foundation for identifying gait deviations and underlying causes. When impairments are complex, instrumented gait analysis provides invaluable data to guide surgical and nonsurgical therapeutic interventions.

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Chapter 16

Pediatric Gait Analysis

Henry G. Chambers, MD

Locomotion is a complex task influenced by interactions between bony alignment, joint ROM, neuromuscular activity, and the laws of physics. In children, congenital deformities, developmental disabilities, acquired problems such as amputation or trauma, and degenerative changes can disrupt the balance between interacting factors, resulting in diminished gait efficiency. Performing a systematic evaluation of a child's walking pattern prior to implementing either physical therapy or orthopedic surgical procedures is critical for ensuring selection of optimal interventions to enhance walking ability. Observational gait analysis provides a tool for documenting key gait deviations and, when linked with a thorough clinical evaluation, can be used to determine underlying causes of walking challenges. When the factors contributing to gait abnormalities are not clear, performing an instrumented kinesiological analysis provides objective pre- and postoperative kinematic, kinetic, and EMG data to guide clinical decision making and subsequent outcomes assessment. These approaches enable treating professionals to understand the nature of the gait problem, gain insight into the etiology, and predict possible treatment options. It is certainly the only way that the technical outcome of a procedure that is designed to improve gait can be assessed objectively.

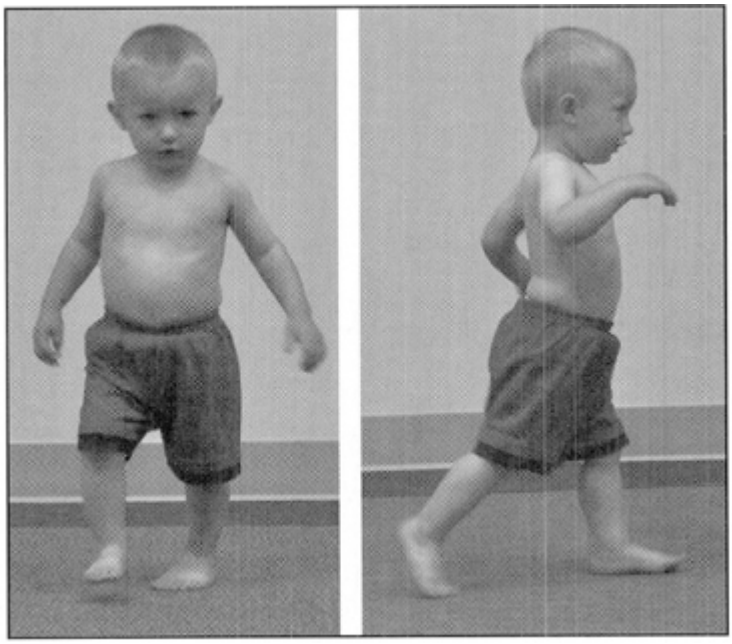


Figure 16-1. Fourteen-month-old toddler walking with arms in “high guard.” Note the increased ankle PF during lead limb loading response.

Gait analysis can range from simply observing a patient’s walking pattern to obtaining a fully computerized study incorporating three-dimensional motion analysis, EMG and energy measurements.[10,64,67,68,72,73] This chapter will demonstrate practical uses of such approaches.

THE DEVELOPMENT OF MATURE GAIT

Sutherland et al[62,65,66] conducted a cross sectional analysis of children from early walking until age 10. They found that when a child begins to walk, he or she uses a “high guard” position with the shoulders abducted and the elbows flexed (Figure 16-1). Reciprocal arm movement is lacking and the hip is usually externally rotated throughout the entire GC. The knees are in relative extension and the ankle demonstrates a toe strike at IC with increased PF during stance. There is usually a broad-based gait and significant

circumduction to clear the externally rotated and extended lower extremities.

As children grow older, their gait pattern begins to more closely approximate that of an adult.[62,65,66] By age 2, arm swing is reciprocal. A heel-first IC emerges, similar to the adult mode of foot strike. The presence of ankle DF during swing allows for limb clearance. Additionally, single limb stance time increases, reflecting greater stability of the stance limb.

By age 3 (Figure 16-2), most of the adult kinematic patterns are present; however, maturation continues until about age 7 when the adult gait pattern is present (Figure 16-3).[62,65,66] Five key parameters show a consistent pattern of change as gait matures:

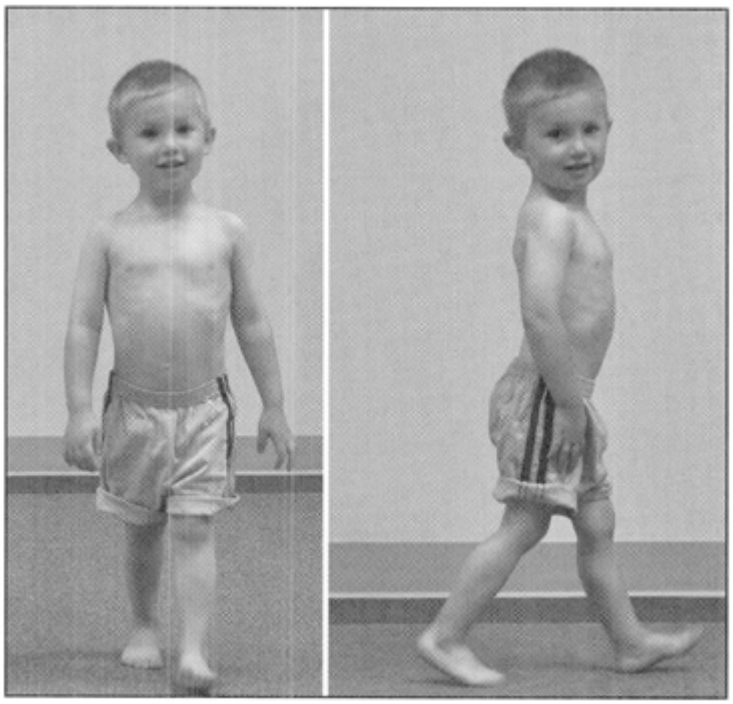


Figure 16-2. Three year old walking. Arms are lowered to side, base of support is narrowing.

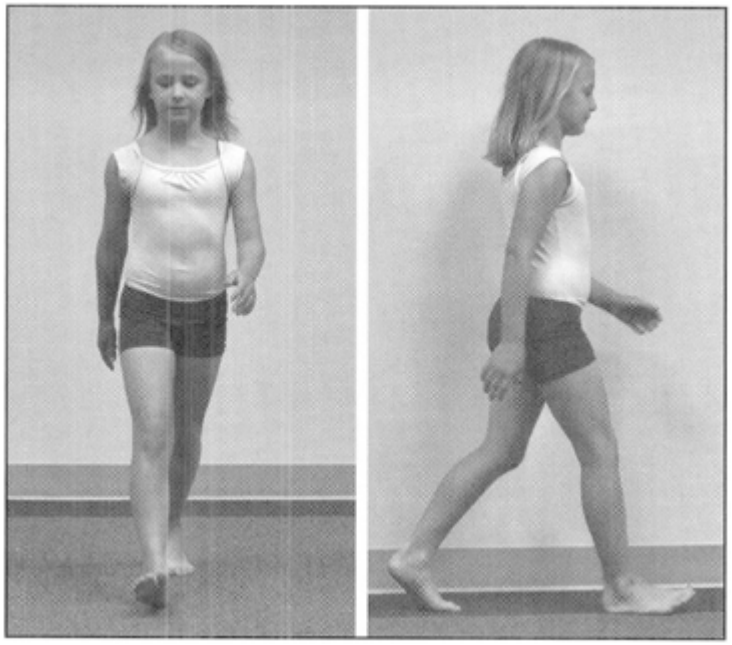


Figure 16-3. Seven year old with adult-like gait pattern.

1. Single limb stance duration increases with age and maturation
2. Walking velocity increases with age and limb length
3. Cadence decreases with age and limb length
4. Step length increases with age and limb length
5. The ratio of the inter-ankle distance to the pelvic width decreases with age and maturation

Todd et al[71] devised a graphical representation of normal gait based on speed, cadence, stride length, and body height derived from 2416 observations of 324 able bodied children. The resulting graphs allow clinicians to determine if changes in gait occur simply due to growth or if some intervention has led to the improved gait parameters (ie, speed and stride length; Figure 16-4).

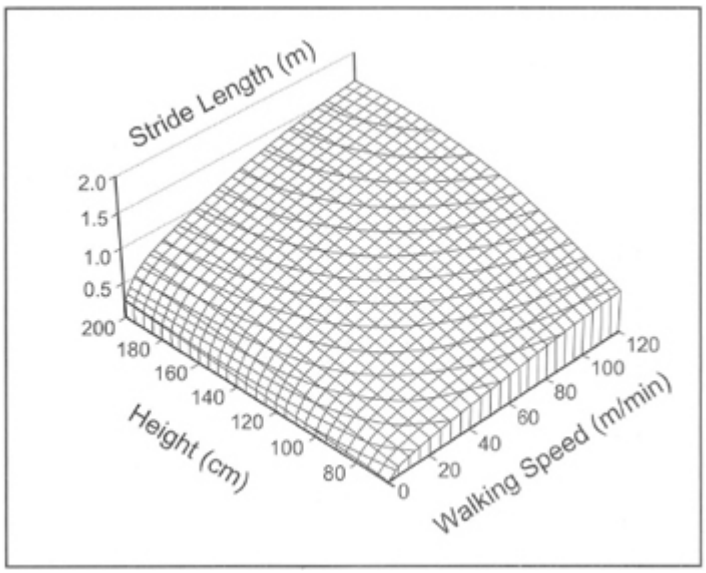


Figure 16-4. Graphical representation predicting stride length for girls as a function of body height and walking speed. (Reprinted with permission from Todd F, Lamoreux L, et al. Variations in the gait of normal children: a graph applicable to the documentation of abnormalities. *J Bone Joint Surg*. 1989;71(2):196-204.)

PRACTICAL USES OF GAIT LABS FOR CHILDREN

The most common use for clinical gait laboratories in the United States is the evaluation of children with developmental disabilities, particularly cerebral palsy and myelomeningocele. These children have very complex gait problems combined with an underlying neurological insult. It is often very difficult to completely evaluate these patients in a clinical setting and gait analysis has been very helpful in formulating treatment plans.[20] DeLuca et al[15] reviewed 91 patients who had recommendations for surgery from experienced physicians and then compared the recommendations based on gait analysis. They found that the addition of gait analysis data resulted in changes in surgical recommendations in 52% of the patients. These changes were associated with a reduction in surgery costs and also prevented potentially detrimental outcomes associated with performing an inappropriate surgery. A similar study involving 97

patients reported an even higher number of treatment plan alterations (89%) following review of data recorded in a gait laboratory.[31,32] Collectively, these studies point to the value of analyzing biomechanical gait data as a part of the surgical decision-making process.

The development of new surgical techniques[39] and orthoses has benefited from research performed in motion analysis laboratories. There is often a clinical question as to the need for an orthosis as well as the type of orthosis that should be provided. Several studies have evaluated the efficacy of different orthotic designs (eg, fixed versus articulating AFO, tone reducing devices, leaf spring AFO) in the management of children with varying medical conditions such as cerebral palsy and myelomeningocele.[7,11,45,54,70] The findings from these studies suggest that appropriate orthotic management can lead to improvements in walking patterns in select patients; however, gait and functional deficits often persist even with bracing.

CEREBRAL PALSY

Cerebral palsy is defined as a group of permanent disorders in the development of movement and posture causing activity limitations that are attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior; by epilepsy; and by secondary musculoskeletal problems.[55] This definition of cerebral palsy demonstrates the disorder's complexity as it affects not only the cortical brain, but all of the brain's functions, and in the case of gait, the effects on the musculoskeletal system.

Problems Encountered in Children With Cerebral Palsy

Children with cerebral palsy have many problems secondary to the brain insult. Challenges with motor control are common, including difficulty volitionally moving their limbs even if they do not have severe spasticity or weakness. For example, a child who has mild hemiplegia may continue to have a drop foot despite having a relatively strong TA. Lacking ability to volitionally activate the muscle during swing, the child's foot remains excessively plantar flexed and limb clearance is threatened.

Balance is a significant problem in most children with cerebral palsy. It is not exactly clear what the etiology of this abnormality is. However, it is probably secondary to poor connections between the cerebellum and most of the other higher centers of the brain, as well as connections to descending neurons to the spinal cord. Ataxia is manifest by a broad-based gait that often changes width across multiple steps.

Advancement in the management of spasticity in children with cerebral palsy has unmasked the presence of significant underlying muscle weakness. Previous clinical paradigms had suggested that a child with spasticity was actually strong as he or she was able to "use their spasticity" to stand and maybe even take a few steps. Research performed by Damiano and colleagues has demonstrated that many children with cerebral palsy have significant weakness and that physical therapy-strengthening regimens help improve gross motor function.[13,14,18]

Perhaps the most significant gait problems for children with cerebral palsy arise from movement disorders. Spasticity, defined as an overactive response of a muscle to rapid stretching, is often assessed using a reflex hammer to tap on (ie, stretch) the tendon. A positive response of the quadriceps would be extension of the knee following a patellar tendon tap. Walking also leads to a rapid stretch of a variety of tendons. For example, the quadriceps is rapidly elongated during both periods of double limb support (ie, loading response and pre-swing). The hamstrings are elongated during the latter half of swing as the knee extends in preparation for the next IC.

A simple way to conceptualize spasticity is to realize that almost all walking activity is reflex driven. The brain's major role is to exert a negative inhibitory (modulation) effect on this reflex arc. Following a

brain injury, inhibitory modulation is decreased and the excitatory reflex activity becomes the overriding mechanism at the muscle level.[26] There have evolved many treatments for spasticity, including selective dorsal rhizotomy,[3] botulinum toxin injections,[23] and oral medications.[49] These treatments have shown that decreasing spasticity combined with active physical therapy significantly improves the gait of children with severe spasticity.

Dystonia is a significant problem in children who are more severely involved.[40] This is thought to arise from the basal ganglia of the brain and is manifest with significant posturing and particularly problems along the long axis of rotation. Dystonia is often seen in children after they transfer from their wheelchairs or walkers to the bed. They may have severe posturing and extension of their upper and lower extremities (persistent neonatal reflexes). There are very few treatments for dystonia, although the intrathecal baclofen pump seems to help.[2,40] To date, however, the influence of intrathecal baclofen pumps on the ambulatory function of children has not been completely elucidated.[2]

Other movement problems include athetosis, which is a writhing-type movement, particularly of the fingers and the upper extremity as well as the trunk.[12,78] This is seen in children who have kernicterus or hyperbilirubinemia, which affects an area of the basal ganglia of the brain that contributes to these abnormal movements. This combined with ataxia can lead to an unstable gait with many extraneous movements.

Classification of Cerebral Palsy and Gait

The most common classification of cerebral palsy has been the classic “geographic” division of cerebral palsy into quadriplegia in which all limbs are involved; diplegia in which the lower extremities have more involvement than the upper extremities; hemiplegia in which one side of the body is affected more than the other, as well as other subdivisions such as triplegia, monoplegia, and double

hemiplegia (ie, bilateral involvement with upper extremities impacted more than lower extremities).[12] However, this classification system has many problems and the inter- and intra-rater reliability are poor. Hoffer et al, in a description of gait, divided patients into those who were community ambulators, household ambulators, and therapy ambulators.[30] This too had many problems with the reliability of placing patients in these groups.

The Gross Motor Functional Classification Scale (GMFCS), which has very good inter- and intra-rater reliability, provides a means of describing children's locomotor function ranging from running and walking independently to requiring assistance to move a wheelchair (Figure 16-5).[46] The GMFCS is becoming the standard description of the gait of children with cerebral palsy as it enables clinicians and researchers to have a common language. However, the GMFCS has limitations. For example, a child may walk well for a short period, but when he or she is on longer trips, he or she may use a wheelchair.

To address this limitation, Graham and colleagues[27] devised the Functional Mobility Scale (FMS) to further classify children who are ambulatory based on their walking ability at 5 meters, 50 meters, and 500 meters (Figure 16-6). When combined, the GMFCS and FMS provide a clear picture of the ambulatory capabilities of a child with cerebral palsy.

Another tool, the Functional Assessment Questionnaire, asks children and their caregivers a variety of questions aimed at determining the child's ability to walk short distances as well as manage common architectural barriers (eg, curbs, uneven terrain, stairs; Figure 16-7).[43] More advanced activities, such as running and climbing on uneven terrain, also are assessed.

In addition to function-based classification tools, approaches using quantified motion analysis to determine the normalcy of gait also have been implemented. Schwartz et al[58] devised a system of "normalcy" of gait using deviations from the normal walking patterns derived from three-dimensional gait analysis. This initial system has since been expanded and is frequently referred to as the Gillette Gait Index. It can be used in pre- and postintervention assessments to determine if the particular therapy, whether it be physical therapy,

spasticity management, or orthopedic surgery, has changed the gait of a child with cerebral palsy.[57,76]

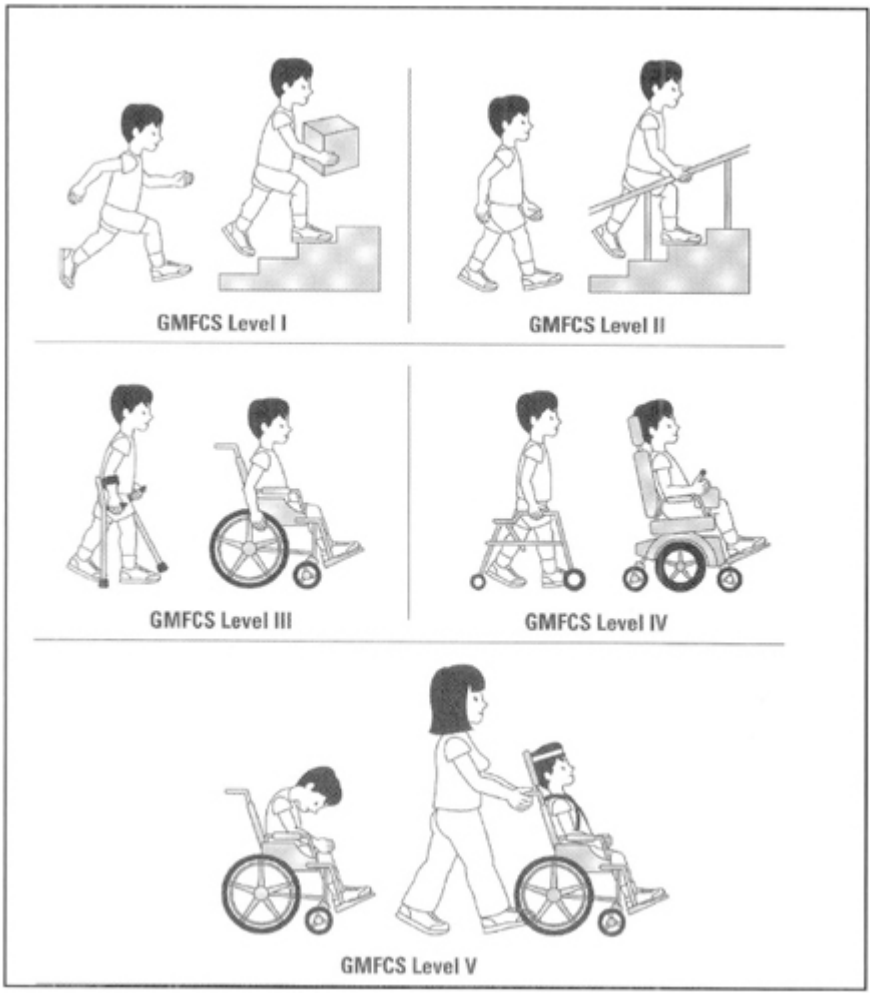


Figure 16-5. Gross Motor Function Classification System (GMFCS) for describing locomotor abilities of children with cerebral palsy. Level 1 represents the highest functional level (listed first), while level 5 represents lowest (listed last). (Courtesy of Dr. Kerr Graham.)

SPECIFIC GAIT PROBLEMS IN CEREBRAL PALSY

In children, both observational gait analysis and instrumented three-dimensional motion analysis are used to identify abnormal movement patterns during walking. To determine the underlying cause(s) of the deviations, a thorough clinical evaluation is required. Kinesiological EMG confirms the underlying muscular causes of deviations. Desloovere and colleagues[17] studied the relationship between clinical measures (eg, spasticity, ROM, selective joint movement) and quantified gait analysis findings obtained from 200 children with cerebral palsy. The authors reported that only 23% of correlation coefficients between the strength variables and quantified gait measures reached the fair (ie, r = 0.21 to 0.40) to moderate (ie, r = 0.41 to 0.60) level of association. Even lower percentages were reported for relationships between gait analysis data and measures of spasticity (19.4%), selectivity (17.7%), and ROM measurements (13%). The authors concluded that both clinical measures as well as instrumented gait analysis data are important when determining the underlying causes of deviations. Collectively, these tools permit informed decision making to guide determination of the most efficacious therapeutic intervention.[17]

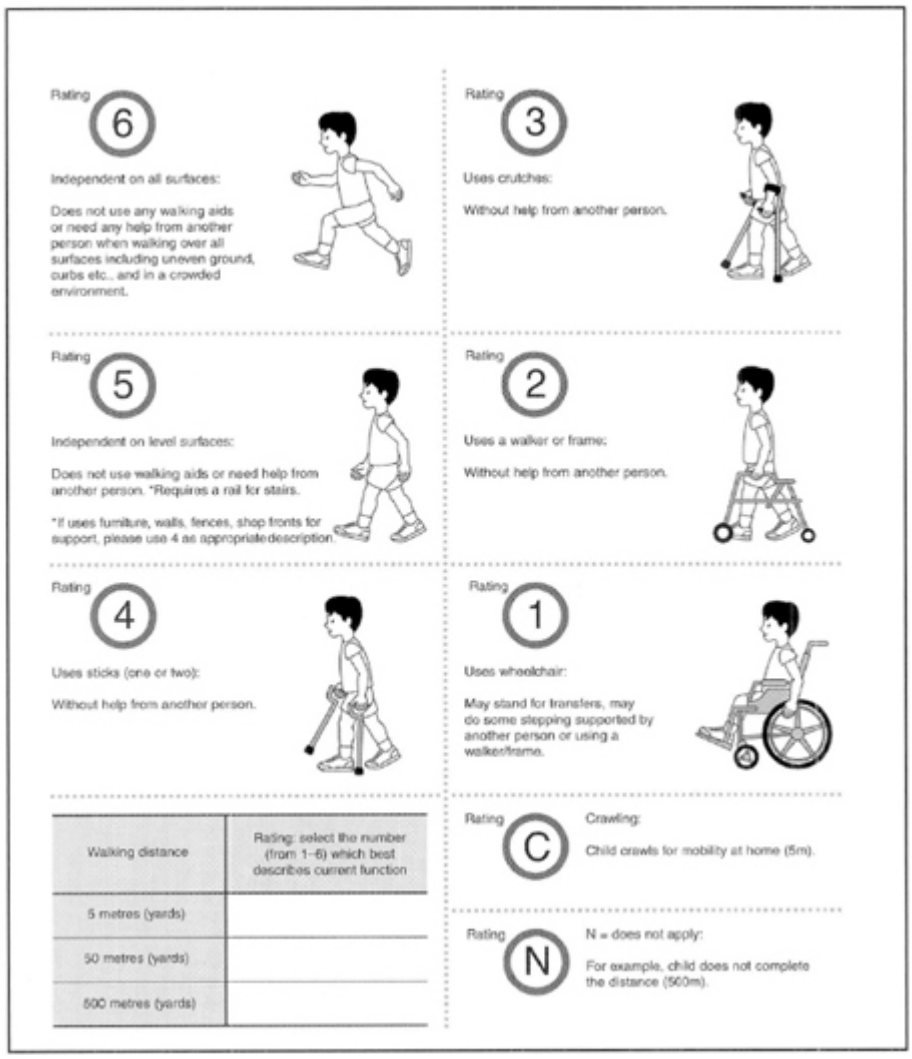
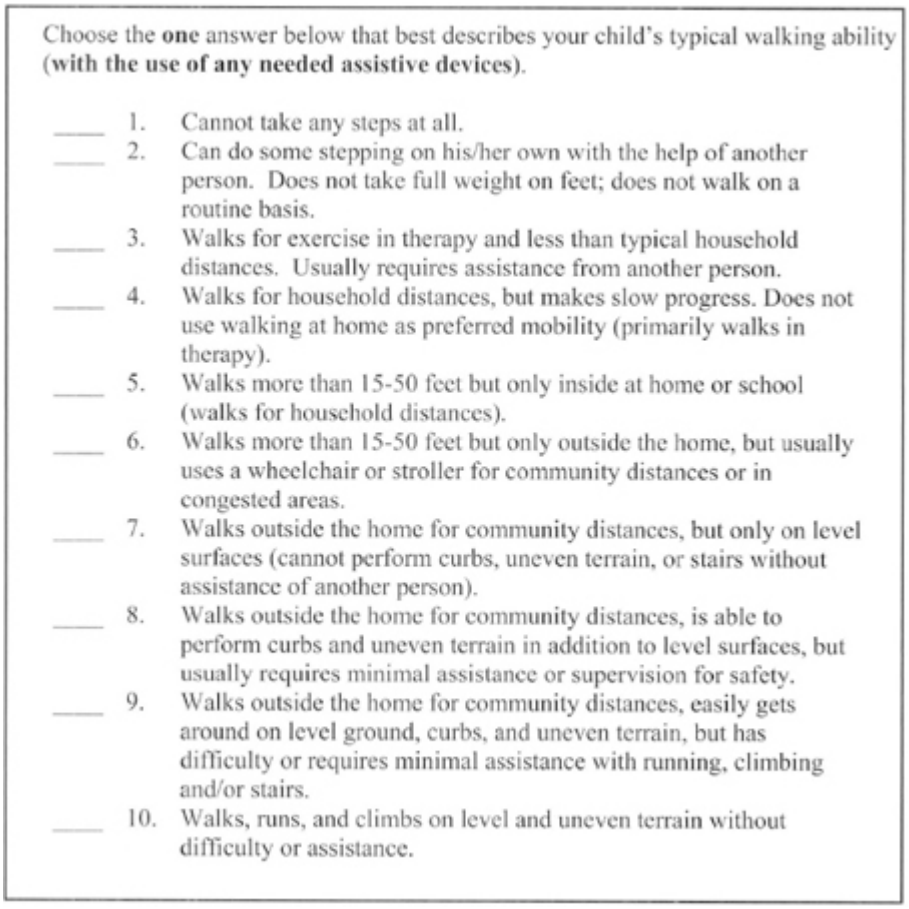


Figure 16-6. The Functional Mobility Scale allows classification of gait abilities across a range of walking distances. Grade 6 represents highest functional level and is listed first, while grade 1 represents the lowest functional level and is listed last. (Courtesy of Dr. Kerr Graham.)



Choose the **one** answer below that best describes your child's typical walking ability **(with the use of any needed assistive devices)**.

____ 1. Cannot take any steps at all.
____ 2. Can do some stepping on his/her own with the help of another person. Does not take full weight on feet; does not walk on a routine basis.
____ 3. Walks for exercise in therapy and less than typical household distances. Usually requires assistance from another person.
____ 4. Walks for household distances, but makes slow progress. Does not use walking at home as preferred mobility (primarily walks in therapy).
____ 5. Walks more than 15-50 feet but only inside at home or school (walks for household distances).
____ 6. Walks more than 15-50 feet but only outside the home, but usually uses a wheelchair or stroller for community distances or in congested areas.
____ 7. Walks outside the home for community distances, but only on level surfaces (cannot perform curbs, uneven terrain, or stairs without assistance of another person).
____ 8. Walks outside the home for community distances, is able to perform curbs and uneven terrain in addition to level surfaces, but usually requires minimal assistance or supervision for safety.
____ 9. Walks outside the home for community distances, easily gets around on level ground, curbs, and uneven terrain, but has difficulty or requires minimal assistance with running, climbing and/or stairs.
____ 10. Walks, runs, and climbs on level and uneven terrain without difficulty or assistance.

Figure 16-7. The Functional Assessment Questionnaire assesses locomotor function across a variety of tasks. Grade 10 represents highest ability (but is listed last), while grade 1 is lowest (but listed first). (Adapted from Novacheck T, Stout J, Tervo R. Reliability and validity of the Gillette Functional Assessment Questionnaire as an outcome measure in children with walking disabilities. *J Ped Orthop*. 2000;20(1):75-81.)

Gait abnormalities in children with cerebral palsy can affect movement at the hip, knee, and ankle.[8,9,15-17,36,50-53,75] Deviations often span multiple joints.

Hip

There are 3 significant hip deviations that commonly impact the child with cerebral palsy. These can occur alone or in concert.

Adduction

Muscle imbalance arising from spasticity of the hip adductors and flexors combined with weakness in the hip abductors and extensors can lead to hip subluxation or dislocation in both the minimally ambulatory and nonambulatory child. Increased hip adduction is usually secondary to spasticity or contracture of the hip adductors. Gait is disrupted when the involved limb crosses over the opposite leg (Figure 16-8).

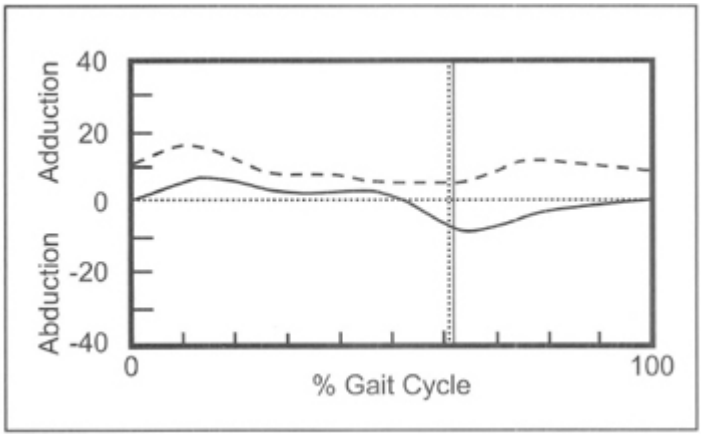


Figure 16-8. Increased hip adduction of the reference limb (dashed line) compared to normal (solid line) can disrupt stability during stance and limb advancement during swing.

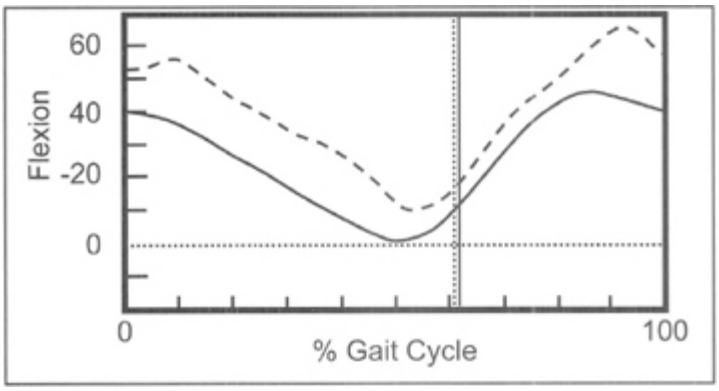


Figure 16-9. Excessive hip flexion of the reference limb (dashed line) compared to normal (solid line) can arise from overactivity or contracture of the hip flexors (eg, iliopsoas, sartorius). This child did not achieve a normal trailing limb posture in terminal stance. A 10° past retract during terminal swing better positioned the limb for IC.

Flexion

Increased hip flexion secondary to spasticity or contracture of the iliopsoas muscle or the other hip flexors such as the sartorius and even the RF muscle can lead to significant gait abnormalities. Excessive hip flexion (thigh relative to pelvis; Figure 16-9) can limit the child's ability to achieve a trailing limb during terminal stance, thus shortening step length. Additionally, increased anterior pelvic tilt can exaggerate lumbar lordosis. Stabilization of the flexed posture may require use of an assistive device or increased activity of the extensors.

Femoral Anteversion

One significant problem that is manifest at the hip of some children is a rotational abnormality secondary to increased femoral anteversion (Figure 16-10). The so-called "lever arm syndrome" is caused by persistent fetal femoral anteversion.[22,42] Most children are born with significant femoral anteversion because of positioning in utero. However, in a child who is unable to ambulate well, particularly those who might have a slight hip flexion contracture,[44] there is little chance for the physiological derotation of the femur.[5] This derotation is thought to occur as the femoral head interacts with the anterior ligament of Bigalow. Increased femoral anteversion is often combined with an increased tibial torsion and occasionally increased metatarsus varus in children with cerebral palsy. This can cause significant "intoeing." The increased rotation during walking is often misdiagnosed as tight adductor muscles as the hips appear to be adducting and crossing over the other limb. It also appears that the child has increased valgus at the knee. Over time, if the femoral anteversion is not corrected, the tibia assumes a posture of severe external rotation. The resulting severe malalignment includes excessive femoral anteversion and external tibial torsion, as well as valgus positioning of the foot and ankle (Figure 16-11). This can lead to patellar instability in children with cerebral palsy. The importance of lever-arm syndrome is that the muscles, which are already weak

and spastic, are operating in a plane that is not biomechanically normal and not conducive to normal muscle activity. Walking ability is further diminished.

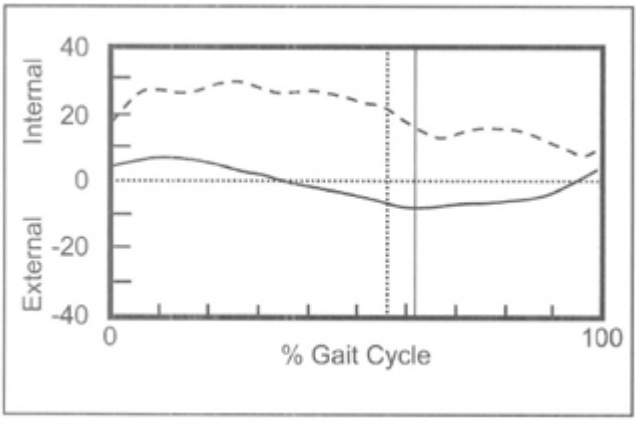


Figure 16-10. Increased femoral rotation of the involved limb (dashed line) throughout the GC can result from persistent fetal femoral anteversion leading to a “lever arm syndrome”.

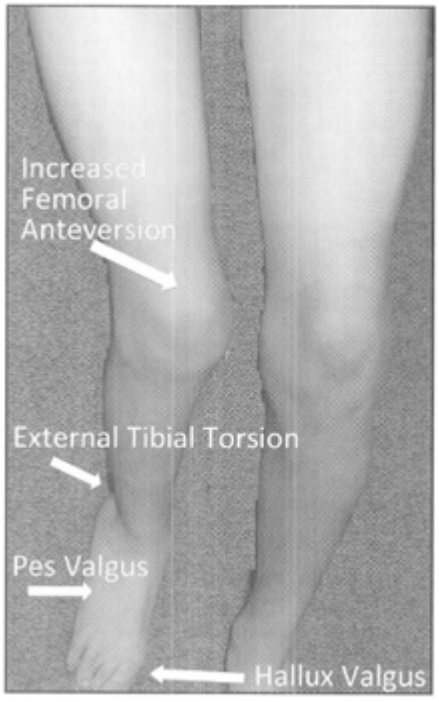


Figure 16-11. Lever arm syndrome. Persistent excess femoral anteversion can lead to increased external tibial torsion and a valgus foot and ankle.

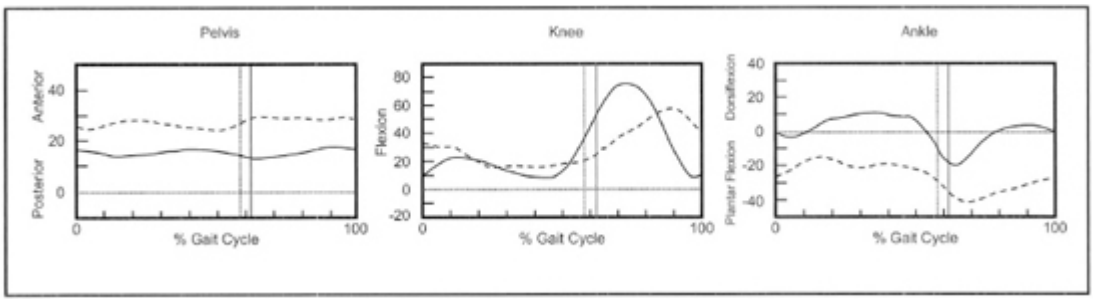


Figure 16-12. Jump gait movement deviations on the involved side (dashed line) compared to normal motion (solid line). The pelvis had an increased anterior tilt, which was a postural accommodation for increased hip flexion. Knee flexion and ankle PF also were increased.

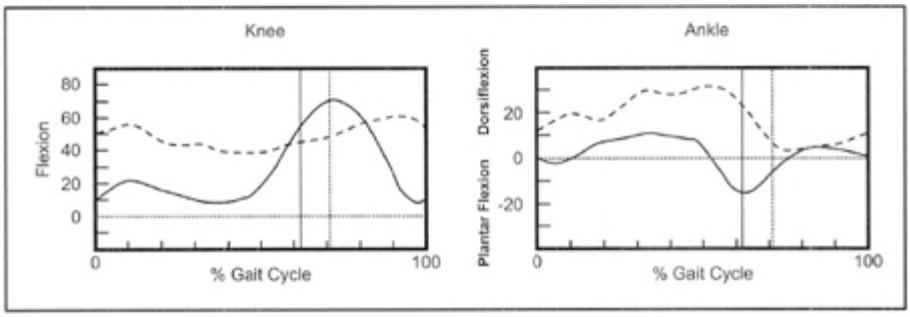


Figure 16-13. Crouch gait. The relatively immobile posture of the knee displayed excessive flexion during stance and inadequate flexion in swing. This is suggestive of a stiff legged gait pattern. The ankle also was in excessive PF throughout stance.

KNEE

Sutherland and Davids described 4 different types of problems that can affect the knee in the child with cerebral palsy.[63] It is artificial to isolate any one joint in cerebral palsy as the entire body is involved. However, breaking the problems down into separate components is often helpful in understanding the complex gait in a child with cerebral palsy.

Jump Gait

The jump gait is defined as having increased stance phase hip and knee flexion as well as equinus. This is the most common gait pattern in children with spastic diplegia. It is often accompanied by lever arm problems as well as a stiff knee gait (Figure 16-12).

Crouch Gait

The characteristic postures associated with a crouch gait include excessive stance phase hip and knee flexion, as well as increased ankle DF or calcaneus. This is often an iatrogenic problem caused by lengthening of the Achilles tendon, without addressing the contractures at the hip and knee. It can be part of the natural history of some children with cerebral palsy, particularly if they have pes valgus and are obese. These children can have a stiff knee gait component to their gait as well (Figure 16-13).

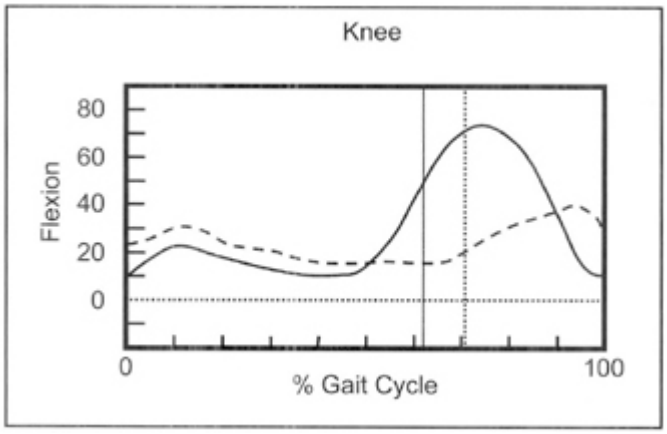


Figure 16-14. The excessive knee flexion in stance continued into swing as delayed and decreased knee flexion during swing.

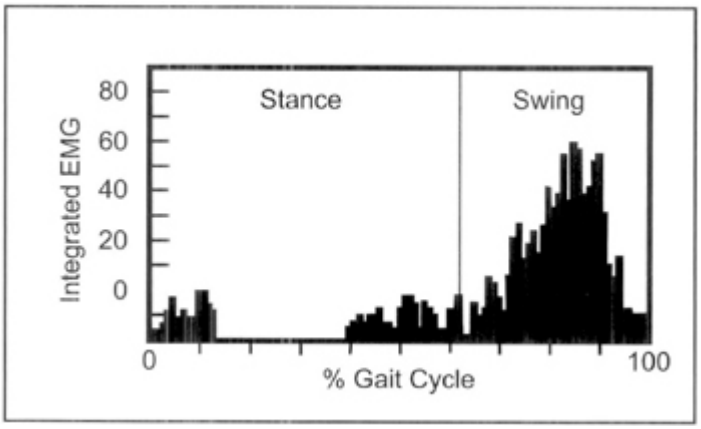


Figure 16-15. Fine wire EMG recorded from the RF in a person with stiff knee gait. Note the prolonged, high amplitude activity of the RF during swing.

Stiff Knee Gait

Stiff knee gait is defined as having decreased ROM of the knee throughout the entire GC. However, most problems arising from stiff knee gait occur in swing. During this period, there is diminished and delayed peak knee flexion, which prevents not only normal limb clearance but also the foot from being placed in the proper position prior to the next foot contact (Figure 16-14). The underlying cause can include excessive activity in any head of the quadriceps, which limits the ability to flex the knee (Figure 16-15).[33] Occasionally, overactivity of the hamstrings during pre-swing prevents forward advancement of the femur at the hip, thus depriving the limb of the normal 40° of passive knee flexion during this period.[33] Patients who have this gait deviation often use compensatory actions to enable limb clearance during swing. These include ipsilateral abduction, external rotation of the entire lower extremity, or contralateral vaulting in an attempt to clear the foot and place it in a better position for initial foot contact.

Recurvatum Knee

The least common of the 4 primary knee problems in children with cerebral palsy is genu recurvatum, also commonly referred to as

hyperextension (Figure 16-16). Plantar flexor spasticity[4] or contractures[8,48] can lead to knee recurvatum by preventing stance phase advancement of the tibia at the same rate as the femur and body above.[61] When the limb is in stance, the knee goes into recurvatum. Another less frequent cause of recurvatum occurs in children who have had surgical over-lengthening of the hamstring tendons at the knee.[60] In this case, the knee hyperextends during stance secondary to loss of the restraining static and dynamic forces that the hamstrings could provide. Sharps and colleagues[60] conducted a follow-up study of persons with cerebral palsy who received a proximal hamstring release and reported that only a small percentage (6.25%) of the 64 knees treated subsequently developed knee hyperextension. The hyperextension was characterized as mild (5° to 10°) and the average time until follow-up was 9 years, 5 months.

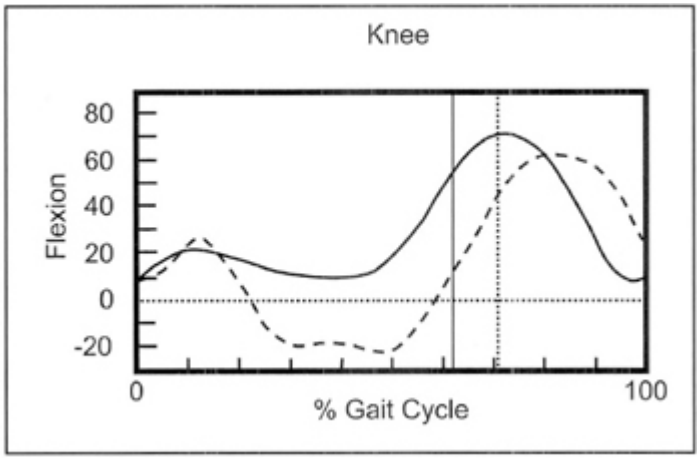


Figure 16-16. Despite initial knee flexion during loading response, recurvatum (hyperextension) dominated SLS (reference limb = dashed line; normative data = solid line).

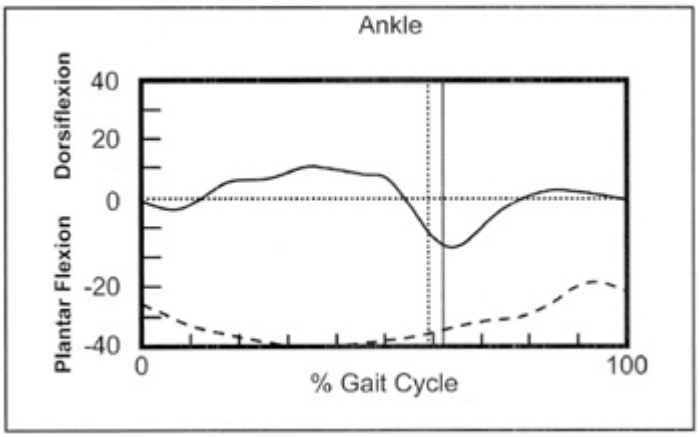


Figure 16-17. Increased ankle PF (equinus) during stance impairs forward progression, while excess PF during swing disrupts normal foot clearance (reference limb = dashed line; normative data = solid line).

FOOT AND ANKLE

There are 4 significant problems of the foot and ankle in children with cerebral palsy: equinus, calcaneus, varus, and valgus. These can occur singly or in combination.

Equinus

Increased ankle plantarflexion or equinus is secondary to either spasticity or contracture[48] of the gastrocnemius-soleus complex. This causes the ankle to plantar flex and the child to walk either on his or her forefoot or even just on his or her toes.[35,48,77] This is one of the most common gait abnormalities seen in children with cerebral palsy (Figure 16-17).[77]

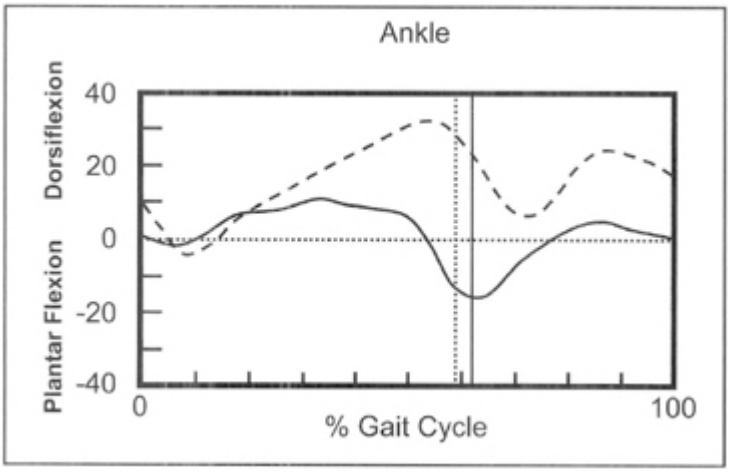


Figure 16-18. Progressive increase in DF through SLS accompanies the calcaneus foot (reference limb = dashed line; normative data = solid line).

Calcaneus

Calcaneus is a hyperdorsiflexion of the ankle, particularly in the stance phase of gait (Figure 16-18). This is frequently seen in concert with crouch gait and is usually secondary to surgery that is done to lengthen just the gastroc soleus tendon (Achilles tendon). This is often an iatrogenic problem arising from a lack of knowledge related to the concept of single event, multilevel surgery in which all of the abnormalities are addressed within the same surgery. For example, a child who has a jump gait with equinus should also have surgery to address the hamstring muscle contractures as well as the hip flexion contractures.

The incidence of developing a calcaneus ankle following isolated lengthening of only the calf muscles varies depending on gender and age at time of surgery. In a study of 134 children with cerebral palsy, Borton and colleagues[6] reported that girls had a greater chance of developing calcaneus post calf muscle lengthening than boys (49% versus 27%, $p = 0.002$) after adjusting for age. Additionally, those who underwent the surgery before the age of 8 were more likely to develop a calcaneus foot than children who had the surgery when older than 8 years (46% versus 17%; $p = 0.046$).

Varus

A varus deformity of the foot and ankle is usually seen in children who have spastic hemiplegia. There is overactivity of the posterior tibialis muscle or the anterior tibialis muscle and often it is present in both muscles.[37] Early swing phase inversion is usually produced by the TA, while stance phase inversion instability is commonly caused by the TP and the triceps surae muscles. It is very important in this particular deformity to have EMG to help determine which muscle(s) are contributing to the deformity and during what period of the GC.[37] For example, a split anterior tibialis tendon transfer may be suggested when these kinematics demonstrate inversion in swing and a relative lack of equinus, as well as when the EMG demonstrates anterior tibialis activity in stance phase and posterior tibialis silence in swing (Figure 16-19). However, a split posterior tibialis tendon transfer may be recommended when the kinematics demonstrate relatively constant foot inversion and equinus in stance and swing (Figure 16-20). The EMG criteria include prolonged activity of TP in stance and swing. Some patients even demonstrate a positive stretch response when the TP tendon is rapidly stretched.

Pressure mapping systems provide a helpful tool for determining the influence of the varus foot deformity on stance phase pressure patterns and center of pressure progression in children with cerebral palsy.[47] An increased pressure time integral impulse under the lateral border has been documented in the varus foot prior to surgical intervention.[47] Changes in pressure patterns following corrective surgery suggest that pressure mapping may serve as a useful tool for documenting outcomes.

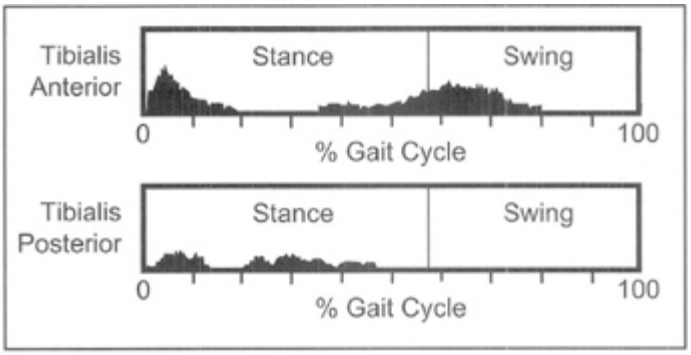


Figure 16-19. Fine wire EMG of TA and TP reveals premature TA activity during the latter half of stance in a client with varus ankle during gait. This patient would be a candidate for a split anterior tibialis transfer.

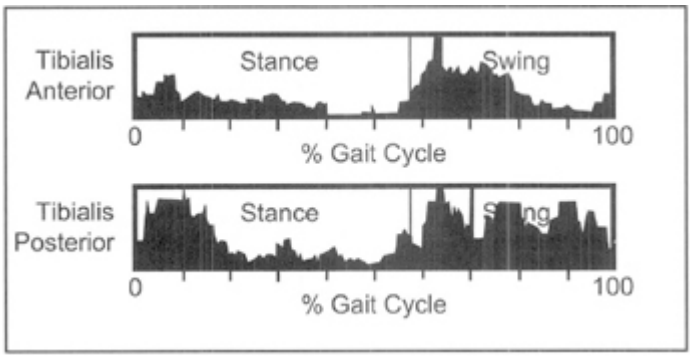


Figure 16-20. Fine wire EMG of TA and TP identifies continuous muscle activity in the TP in a client with a varus ankle. This client would be a candidate for a split posterior tibialis transfer.

Valgus

As opposed to equinovarus, which is the most common deformity in hemiplegia, the most common foot problem in diplegia is valgus. This is often associated with equinus, hind foot valgus, and then eventually mid-foot collapse. These children demonstrate stance phase instability, callosities on the medial aspect of the foot (particularly over the TN joint and hallux valgus) and shoe wear problems. As children get older, they eventually have pain when ambulating. As stated previously, this deformity is often seen in children who have increased femoral anteversion combined with increased external tibial torsion. The EMG may demonstrate increased peroneal activity, as well as gastroc soleus activity. Pressure mapping is again helpful in determining the amount of valgus that a child has and for documenting the amount of change after surgical intervention.[1,47]

CLINICAL EXAMPLE: INFLUENCE OF CEREBRAL PALSY ON GAIT

A complex interplay of weakness, tone, spasticity, and joint contractures can lead to significant gait challenges in children. The following case example highlights the role of quantified gait analysis in defining the presence of gait abnormalities, determining the underlying causes, and elucidating appropriate treatment intervention.

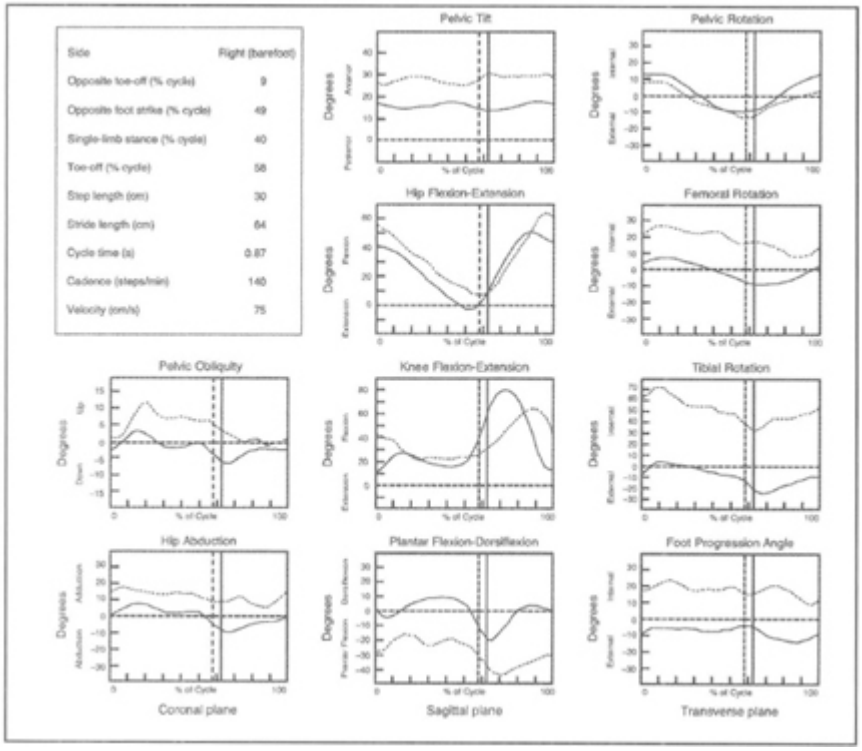


Figure 16-21. Preoperative time-distance parameters and kinematics for a 4-year-old boy with cerebral palsy (dashed lines) compared with an able-bodied 4-year-old child (solid lines). The vertical lines represent toe-off. Left column (coronal plane): Pelvic obliquity with stance side elevated and hip in excessive adduction. Center column (sagittal plane): Anterior pelvic tilt and hip flexion increased through stance, knee flexion greatest at IC, decreased 50% after loading response, during swing peak knee flexion was delayed and diminished. The ankle was in excessive PF throughout

the cycle. Right column (transverse plane): Hip, knee and foot are excessively internally rotated. (© 2002 American Academy of Orthopaedic Surgeons. Reprinted from the *Journal of the American Academy of Orthopaedic Surgeons*, Volume 10(3), pp. 222-231 with permission.)

Cerebral Palsy

A 4-year-and-5-month-old boy presented with bilateral toe walking and internal rotation of the limbs.[10] He wore bilateral AFO but fell up to 20 times per day. He was able to ride a tricycle, climb stairs, and had an endurance of about 0.5 mile. The experienced referring orthopedic surgeon thought that he should have bilateral heel cord lengthenings.

Physical examination demonstrated mild hip flexion contractures and increased femoral internal rotation (70°) bilaterally. He had a 150° popliteal angle and 15° ankle PF contractures. He had hyperreflexia and a positive Ely-Duncan test suggestive of RF spasticity.

Gait analysis, including three dimensional motion analysis and dynamic surface EMG, was performed to identify and quantify the gait deviations and to determine underlying causes. Sagittal plane kinematic data demonstrated increased anterior pelvic tilt, minimally increased hip flexion, diminished and delayed peak knee flexion in swing, and a marked increase in ankle PF throughout the GC (Figure 16-21).

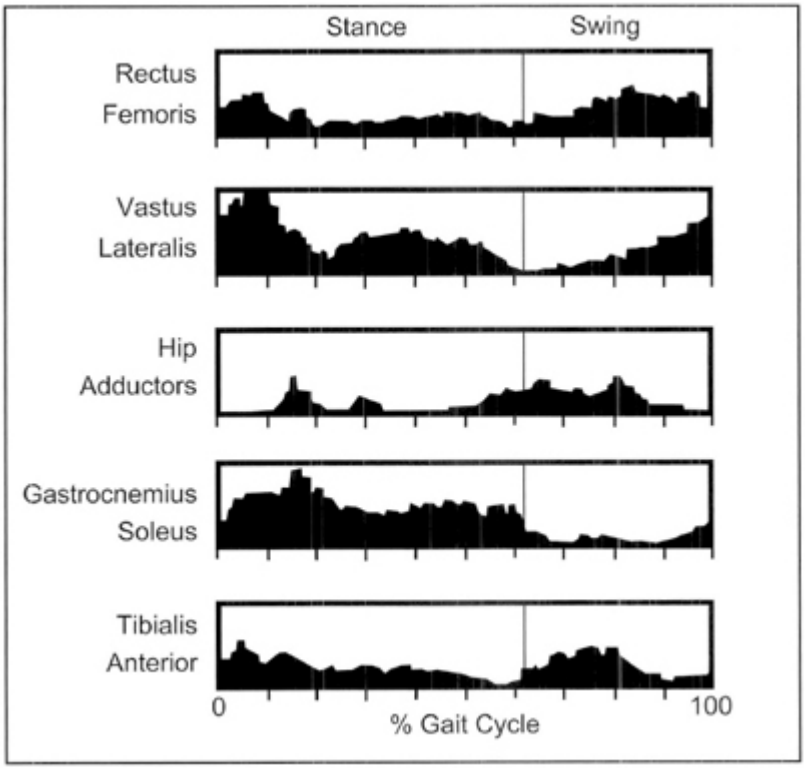


Figure 16-22. Preoperative electromyograms by surface electrodes. Rectus femoris and vastus lateralis are continuous throughout the cycle. The relative intensity of RF parallels that of VL. This is indicative of cross-talk. During initial swing, the bulge in RF EMG versus depression of VL indicates true RF activity. Gastrocnemius action begins prematurely in late swing. The low level EMG in early and mid swing are signs of cross-talk from pretibial muscles. (© 2002 American Academy of Orthopaedic Surgeons. Reprinted from the *Journal of the American Academy of Orthopaedic Surgeons*, Volume 10(3), pp. 222-231 with permission.)

Frontal (coronal) plane abnormalities included increased pelvic obliquity in stance phase and increased adduction throughout the cycle (see Figure 16-21). Transverse plane abnormalities included increased internal femoral rotation; internal tibial rotation, which followed the femoral rotation; and an internal foot progression angle (see Figure 16-21).

The EMG data showed full cycle activity of the RF but most importantly, increased activity in swing phase, full cycle activity of the vastus lateralis, minimal but out-of-phase activity of the adductors,

mostly stance phase activity of the gastrocsoleus, and full cycle activity of the TA (Figure 16-22).

Based on his physical examination, review of the videotape, and integration of the gait data, the following procedures were recommended: bilateral varus derotational osteotomies of the femurs, psoas lengthening at the pelvic brim, adductor longus recession, distal medial hamstring lengthening, rectus to semitendinosus transfer, and Strayer gastrocnemius recession. Although some of these procedures could have been predicted by a meticulous examination of the child, some such as the RF transfer required integration of kinematic and EMG data.

One year after surgery, the child was no longer falling and was playing soccer and learning to roller blade. The kinematic plots demonstrated that the parameters had all returned to near normal (Figure 16-23).[10]

MYELOMENINGOCELE

Myelomeningocele or spina bifida is a disability caused by the incomplete closure of the neural arches.[41,59] The extent of involvement can range from a small radiographic anomaly (spina bifida occulta) to a condition in which the entire spinal cord is uncovered (myeloschisis), which is fatal. The patients who survive will have a neurosurgical procedure to close their neural sacs and provide coverage of the spinal cord. All of these patients will have significant motor and sensory deficits. The extent of the neurological deficit is determined by the highest level of function that is present. Knowledge of the dermatomal and myotomal anatomy and functional assessment will enable the examiner to categorize the patients. It is customary to categorize the patients according to the last fully intact level of motor strength and there are many published articles that have used this approach. However, there is often asymmetry and there also are significant inter- and intra-rater reliability issues with this classification. A more practical classification has evolved over the past 20 years in which patients are classified as thoracic, high

lumbar, low lumbar, or sacral.[34] Use of this classification system can provide insights into the gait challenges faced by children with myelomeningocele.[56,73,74]

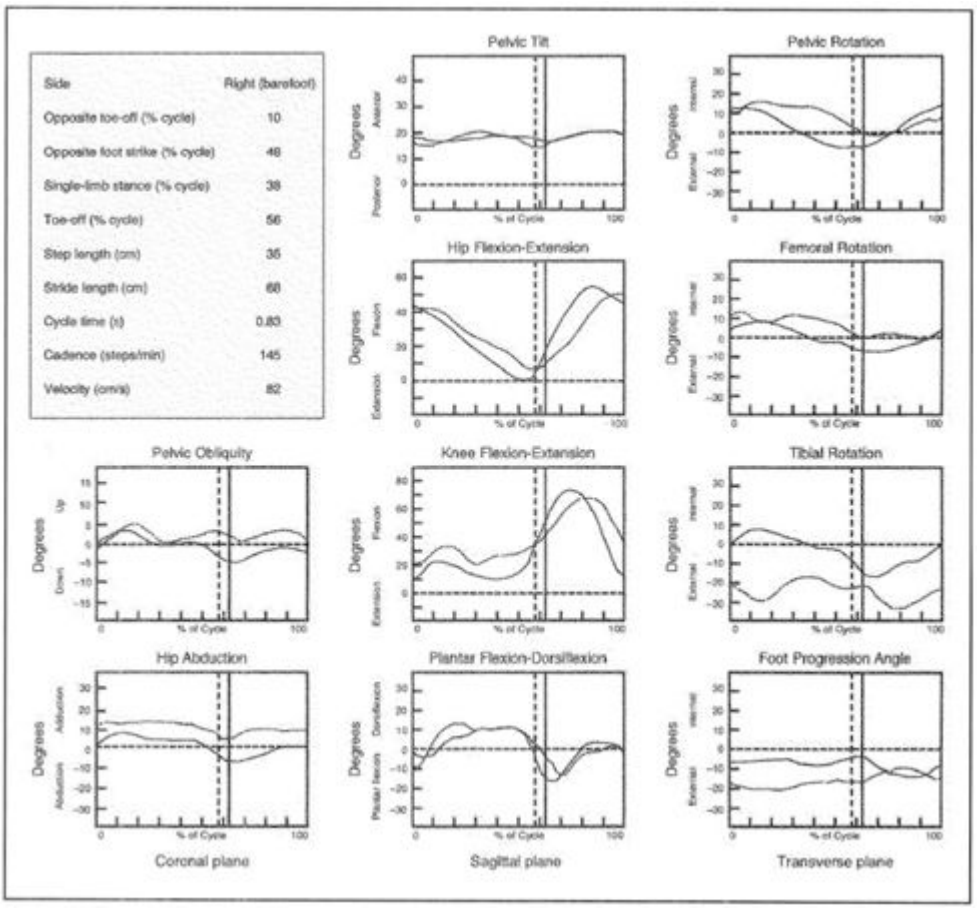


Figure 16-23. Postoperative time-distance parameters and kinematics for the same child, 6 years of age at testing. Pelvic alignment substantially improved (coronal and sagittal). Peak knee flexion during swing more closely approximates normal timing and amplitude. Following excess PF at IC, the ankle rapidly reverses to DF as body weight loads onto the limb. In the transverse plane, rotation at the hip, knee, and ankle is improved. (© 2002 American Academy of Orthopaedic Surgeons. Reprinted from the *Journal of the American Academy of Orthopaedic Surgeons*, Volume 10(3), pp. 222-231 with permission.)

The thoracic- and high lumbar-level patients lack gluteal, quadriceps, plantar flexor, and dorsiflexor muscle activity. Although

they may be able to walk as children using a reciprocating gait orthosis or parapodium, the majority stop walking by age 10 or 12 because the energy costs of continuing to walk are too great.[25,69,73]

Low lumbar-level patients demonstrate some quadriceps and medial hamstring strength but no functional gluteal, ankle plantar flexor, or ankle dorsiflexor strength.[28,29,73] The vast majority of these patients continue to walk throughout life, despite the presence of significant gait deviations and reduced walking efficiency.[21,24,38] Duffy et al[19] evaluated 28 children with low lumbar (L4 and L5) and sacral (S1) level myelomeningocele and identified that for each level, there was a pattern of gait deviations consistent with the expected muscle weakness (ie, hip abductor, calf, and pretibial). Common deviations included increased pelvic obliquity and rotation with hip abduction in stance and persistent knee flexion throughout the stance phase.

Gabrieli et al[21] used gait analysis to evaluate walking ability in 20 community-level ambulators with low lumbar myelomeningocele. All subjects had a unilateral hip dislocation or subluxation. Half of the group had a unilateral hip flexion or adduction contracture, while the other half had either full ROM or symmetrical contractures at the hip. Those with unilateral hip flexion contractures were more likely to have an asymmetrical gait pattern than those with no contractures or symmetrical hip contractures, suggesting that the unilateral contracture impacts gait symmetry more than the subluxation/dislocation. Hence, efforts aimed at maintaining full or symmetrical hip ROM may be warranted to maintain gait symmetry.

While patients with low lumbar myelomeningocele are able to ambulate, the walking pattern is slowed and less efficient.[38] A comparison of the energy cost of device-assisted gait (swing through or reciprocal pattern) to normal gait parameters revealed that velocity was slowed and energy cost increased for those with low lumbar myelomeningocele compared to normative values. Additionally, reciprocal gait resulted in slower and less cost-efficient locomotion than swing through gait, suggesting that efforts to teach use of a reciprocal gait pattern may ultimately lead to reduced walking ability.

Myelomeningocele often leads to a predictable pattern of muscle weakness and gait deviations. Slowed walking velocity and reduced gait efficiency are common consequences. While individuals with lower level involvement (ie, sacral and low lumbar) frequently continue ambulating throughout life, those impacted at higher levels (ie, high lumbar, thoracic) often walk when younger and lighter, but transition to a wheelchair when the interplay between body weight and muscle weakness prohibits meaningful gait.

CONCLUSION

Motion analysis is a diagnostic and outcome research tool that should be viewed similarly to modern radiologic imaging techniques of the musculoskeletal system. While many and possibly most pediatric gait problems can be evaluated with a systematic visual inspection and complete clinical evaluation, some problems require some or all of the components of the modern motion analysis laboratory. Pediatric gait, with its neurological control, muscular response, and bone and joint motion, is far too complex for even the best clinician to appreciate all of the intricacies with simple observation. Although an overall idea about the gait can be obtained in this manner, subtleties and fine nuances of gait cannot. Gait analysis provides an objective record of a child's gait before and after therapeutic intervention and should be considered a vital part of the clinician's decision making.

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Dedicated to the memory of David H. Sutherland, MD (1923-2006), who, along with Dr. Perry, was one of the pioneers of modern gait analysis. A former president of the American Academy for Cerebral Palsy and Developmental Medicine, Dr. Sutherland dedicated his career to the improvement of gait analysis technology and clinical application of its principles. Through his research, clinical care, and teaching, he will be remembered for improving the lives of people with cerebral palsy.

Section V

Advanced Locomotor Function

Chapter 17

Stair Negotiation

A staircase is a series of horizontal platforms (stairs) with a vertical offset between adjacent platforms to facilitate walking up or down a hill or other tilted surface. The magnitude of joint motion, the intensity of muscle action, and the forces generated during the ascent or descent of such offset platforms are determined by both the dimensions of the stair and the physical characteristic of the user. To clarify whether the structure or the person is being discussed, the word *stair* will relate to structure while subject performance on the stairs will be identified by the word *step*.

STAIR DIMENSIONS

The dimensions of a stair are its vertical height (riser) and the depth of the horizontal platform (tread). During each step, the stance limb supports and elevates the body while the swinging limb passes 3 structures: 1) the riser, 2) tread of the stair currently occupied by the stance limb, and 3) the riser of the next higher stair, where the swinging foot initiates floor contact and begins another complete step. In 1672, Francois Blondel published the first criteria for stair dimensions. His formula specified that the sum of 2 riser heights and one tread depth should equal 24 inches.[5,9] The dimensions in Blondel's formula have persisted as a guide for today's building standards. For example, in Downey, California, the construction code for a flight of stairs states the riser height is to be "no more than 7 inches" and tread depth "no less than 11 inches." When these dimensions are applied to Blondel's formula, the sum of twice the riser height (14 inches) and one tread depth (11 inches) totals 25

inches. Thus, the modern criteria exceed Blondel's formula by only 1 inch. In the studies referenced in this chapter, riser heights ranged from 5 to 9 inches, while tread depths varied from 8 to 16.5 inches. The wide variability in stair dimensions reflects, in part, the intent of some studies to determine the influence of step dimensions on motion, moment, and muscle demands (Table 17-1).[9,15,17]

Table 17-1

*Comparison of Stair Dimensions Used in Studies Evaluating Impact of Stair Inclination on Gait Characteristics**

Study	*Inclination (degrees)*	*Riser Height (cm)*	*Tread Depth (cm)*	*Blondel Formula (Tread + 2•Riser)*
Livingston et al[9]				
Steep	45	20.3	21.0	61.6
Optimal	34	20.3	30.5	71.1
Shallow	17	12.7	41.9	67.3
Riener et al[15]				
Steep	42	22.5	25.0	70.0
Optimal	30	17.0	29.0	63.0
Shallow	24	13.8	31.0	58.6
Stacoff et al[17]				
Steep	41	20.0	23.0	63.0
Optimal	30	17.1	29.0	63.2
Shallow	20	13.3	37.0	63.6
Building Code (Downey, CA)	33	17.8	27.9	63.5

*The stair construction formula developed by Francois Blondel specified that the mathematical total of the tread plus 2 times the riser height should be 24 inches (~ 61.0 cm).

Data from references:
Livingston LA, Stevenson JM, Olney SJ. Stairclimbing kinematics on stairs of differing dimensions. *Arch Phys Med Rehabil.* 1991;72(5):398-402.
Riener R, Rabuffetti M, Frigo C. Stair ascent and descent at different inclinations. *Gait Posture.* 2002;15:32-44.
Stacoff A, Diezi C, Luder G, Stüssi E, Kramers-de Quervain IA. Ground reaction forces on stairs: effects of stair inclination and age. *Gait Posture.* 2005;21:24-38.

A minimum tread depth of 11 inches, as is possible based on some building codes, would not be optimal. This depth barely exceeds the length of an average man barefoot (10.7 inches),[18] and thus, would not accommodate the shoe length needed to protect the toes. The obvious solution is to make deeper treads. The use of deeper treads is supported by the finding that increased depth allows

subjects to move faster.[9] The drawback of using deeper treads relates to the increased space requirements for a given set of stairs.

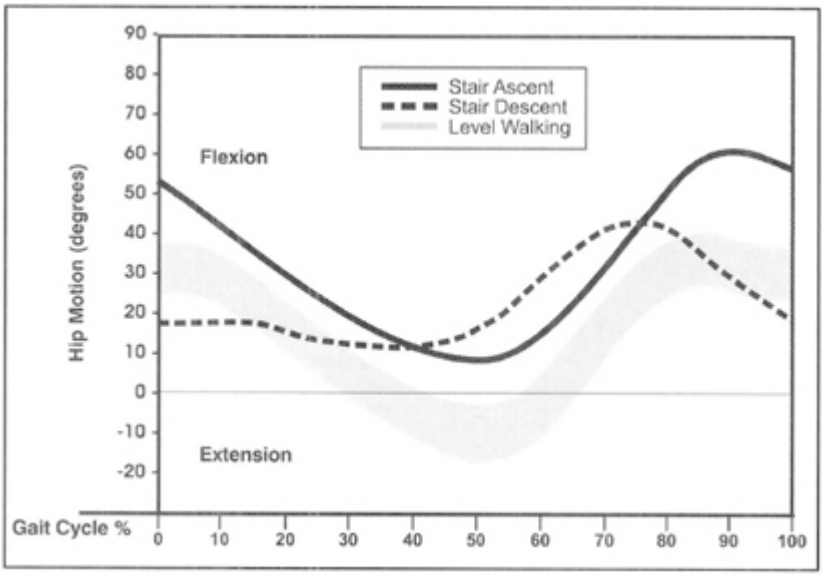

Figure 17-1. Comparison of hip joint motion during stair ascent, stair descent, and level walking.

Walking up a flight of stairs requires a significantly different pattern of joint and muscle action than is used to go down the same set of stairs. The functional requirements of stair ascent and descent will be defined by the limb performance on a “standard” (also called optimum) staircase with an average slope (~30°).

STAIR ASCENT

The transition from walking on level ground to ascending a flight of stairs increases the requirements for strength and joint mobility. The stance limb must lift the body mass in addition to preserving weight-bearing stability, while the swing limb quickly flexes to advance from the stair below to the stair above the stance limb.[15]

The study of stair gait generally begins with the subject standing on the floor (stair 0) facing the staircase. The initial step from the floor by each limb will not be the focus of this discussion, as the 2 stepping cycles are incomplete and can be misleading. The step

from the floor onto the first stair (0-1) is only an abbreviated swing. While the contralateral limb's movement from the floor to the second step (0-2) is longer, it is initiated from a more stable base.[1] The mechanics of a full step first occurs when the leading foot initiates contact on the third step. Now the limb is prepared to perform all of the normal "gait" phases. All subsequent ascending steps begin as each limb (in reciprocal fashion) contacts the next stair above the one currently occupied.

MOTION

IC on the higher stair tread is made with flexion at both the hip (50° to 60°) and knee (50° to 70°) (Figures 17-1 and 17-2, respectively).[12-15,19] Rapid extension of both joints immediately follows IC. The hip and knee continue to extend quickly throughout loading response and mid stance. By late terminal stance, a position of minimal flexion is reached at the hip (10° to 15°) and knee (5° to 15°) and is maintained until just prior to toe-off.[12-15]

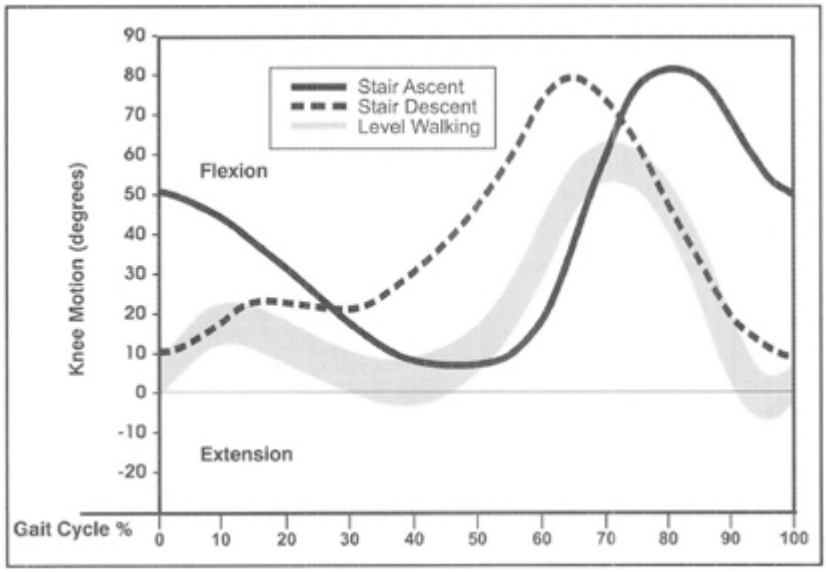

Figure 17-2. Comparison of knee joint motion during stair ascent, stair descent, and level walking.

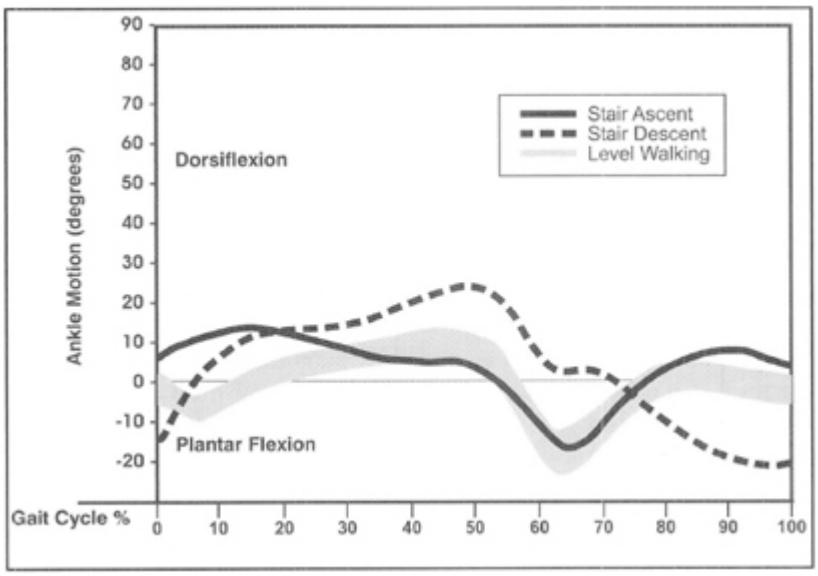

Figure 17-3. Comparison of ankle joint motion during stair ascent, stair descent, and level walking.

IC frequently occurs with the forefoot. Most frequently, the ankle is dorsiflexed (5° to 10°), although PF may be present if the knee and hip are in greater positions of flexion (Figure 17-3).[12-15] With the onset of WA, the ankle dorsiflexes an additional 5° as the body advances across the stair. The transition to terminal double limb support is accompanied by a rapid reversal into PF (10° to 20°) as weight is transferred to the lead limb.[12-15]

All 3 joints begin to flex with the onset of swing. To ensure the foot clears the stair's edge, mid swing hip and knee flexion reaches a peak at least 10° higher than that needed for stance. Then the hip and knee reduce their extra flexion in preparation for IC on the second higher stair. The ankle achieves 5° to 10° DF during the same period.[13-15]

Muscle Control

During stair ascent, the dominant muscle demand is to lift the body during each stance period. Among 7 EMG studies of muscle function during stair negotiation, 2 investigators used intramuscular wire electrodes to document the activity of the hip and knee muscles

during both gait and manual muscle tests.[10,13] This allowed the intensity of each muscle's effort to be quantified as a percent of maximum capability and the relative contribution by the individual muscles defined.

Many hip and knee muscles become active during the latter half of swing in preparation for stance phase demands for stability. Moderately strong hip extension at IC is provided by the lower gluteus maximus (30% maximum voluntary contraction [% MVC]) and adductor magnus (40% MVC).[10,13] Concurrently, the upper gluteus maximus (40% MVC) and gluteus medius (60% MVC) provide forceful lateral stabilization.[10] Activity at IC is notably less in the 2 major hamstrings, semimembranosus (0% to 15% MVC), and BFLH (0% to 10% MVC).[10,13] The quadriceps, represented by the vastus lateralis, is moderately active at IC (30% MMT) and then rapidly increases intensity to a peak of 60% MMT by mid loading response.[13] Surface EMG recordings of the vastus medialis[6] and vastus lateralis[11] demonstrate similar timing for peak activity of the quadriceps. Quadriceps activity subsides as the body mass advances over the increasingly extended knee in mid stance. In the latter half of stance, increasing activity of the BFLH (30% to 40% MMT) and semimembranosus (up to 50% MMT) augments control at both the hip and knee.[10,13] Prior to toe-off, the BFSH rapidly increases activation to a peak of 45% MMT to assist with the knee flexion required to clear the next step.[13] Low-level activity of the RF (<20% MMT) occurs just prior to toe-off in approximately half of subjects tested.[13] This action continues through mid swing. In terminal swing, the BFSH ceases activity nearly simultaneously with the onset of the vastus lateralis.[13]

All of the studies of ankle muscle control during stair ascent used surface EMG.[1,6,7,11] The muscles studied include the soleus, medial gastrocnemius, and TA. During loading response, activity of the soleus rapidly increases and crests just prior to contralateral toe-off. Following a period of reduced activity during mid stance, activation of both the soleus and medial gastrocnemius progressively rises and peaks at the initiation of pre-swing (50% GC).[6,11] As the limb prepares for swing, TA activity is initiated and increases steadily until

reaching maximum in mid swing.[6,11] TA activity gradually diminishes during the latter half of swing and ceases following the acceptance of body weight in loading response.[6,11]

FORCES

Ground Reaction Force

During stair ascent, the pattern of the instantaneous vertical force that is imposed on the ground has 2 peaks.[8,11,14,15,17] The first occurs at the beginning of SLS and has a magnitude of approximately 1.12 normalized body weight units (BW).[17] The second peak during pre-swing is slightly greater (1.23 BW).[17] These intensities closely approximate the first (1.19 BW) and second (1.17 BW) peaks characteristic of level walking.[17]

The AP GRF during stair ascent is similar in pattern but diminished in amplitude relative to that observed during level walking.[15] During the first half of stance, the resultant GRF is directed in a posterior direction,[15] helping to resist forward slippage of the foot on the stair.[2,3] In the latter half of stance, an anteriorly directed GRF resists posterior foot slippage.[2,3,15]

Moments

Stair ascent is dominated by internal extensor moments at the hip, knee, and ankle. The most commonly reported pattern at the hip is the presence of an internal extensor moment throughout stance.[1,4,14] Three research teams have noted transition to a brief, relatively low-amplitude flexor moment occurring in the latter third of stance.[11,12,15] A short internal flexor moment at the initiation of stance followed by an extensor moment has also been reported; however, this appears less common.[16] Variability in trunk position has been postulated to

contribute to the differences in hip moment patterns across studies.[14]

There is a brief, low-amplitude internal flexor moment at the knee immediately following IC.[4,11,12,14,15] This rapidly transitions to a higher amplitude extensor moment that predominates throughout the remainder of loading response and mid stance.[1,4,11,12,14,15] The peak amplitude of this knee extensor moment varies substantially across stair ascent studies, with values ranging from approximately 0.5 N•m/kg to 1.5 N•m/kg.[4,11,12,14,15] Trunk posture, which could notably influence the values, has not been systematically controlled.

The knee moment transitions from extensor to flexor during terminal stance. Pre-swing is consistently characterized by the presence of an internal knee flexor moment.[1,4,11,12,14,15]

At the ankle, the internal plantar flexor moment that occurs during stance has a double-peak profile.[1,11,12,14,15] The second peak occurs in terminal stance and is generally larger in amplitude (1.2 N•m/kg to 1.5 N•m/kg) compared to the initial crest during early stance (15% to 20% GC).[11,12,14,15]

Power

Throughout stance, a series of power bursts elevate and advance the body.[11,12,15] Positive bursts of power generation during loading response at the hip (1.01 W/kg at 7% GC) and mid stance at the knee (1.79 W/kg at 18% GC) assist with forward lift of the body.[12] In late stance, the ankle generates a notable burst of power (2.53 W/kg at 53% GC) that facilitates transfer of body weight to the lead limb.[12] Only power at the ankle is similar in magnitude to level walking.[12]

Functional Significance of Stair Ascent

The ease of walking up a flight of stairs depends on the individual's lower limb muscle strength and joint mobility.

Functionally, the stepping cycle during stair ascent can be divided into 3 periods: 1) WA, 2) SLS, and 3) SLA.

Weight Acceptance

During WA (Figure 17-4), the lead limb is obligated to accept body weight from the trailing limb and to initiate lift of the HAT over the new base of support. Stability must be maintained in concert with forward and upward progression.

Forefoot contact initiates the WA period. The hip and knee are flexed approximately 60°, while the ankle is dorsiflexed 5°. Vigorous concentric action of the single joint extensors of the hip (ie, lower gluteus maximus and adductor magnus) and knee (ie, vastus lateralis and vastus medialis) simultaneously extends the 2 joints.[10,13] By the middle of loading response, activation peaks in the lower gluteus maximus (30% MVC) and quadriceps (60% MMT)[10,13] in order to provide sufficient force to extend the joints under the increasing load of body weight.[15] Power generation at the hip and knee rapidly rises during this period.[15] Less intense action of the hamstrings (semimembranosus and BFLH) supplements hip extension and stabilizes the knee by opposing anterior displacement (drawer) of the tibia by the quadriceps.[10,13] In the frontal plane, the hip abductors (gluteus medius and upper gluteus maximus) augment stability and weight transfer over the new base of support.[7,10,11] At the ankle, WA is characterized by progressive DF from the initial position of 5° DF to a final maximum of 12° at the end of loading response. Tibial progression is restrained by rapidly increasing soleus activity, which reaches a maximum just prior to contralateral toe-off.[11]

Figure 17-4. Stair ascent muscle demands during WA. Gluteus maximus, adductor magnus, and gluteus medius forcefully stabilize the hip, while the quadriceps peak in activity at the knee. Low-level activation of hamstrings augments hip extension and resists anterior draw of the tibia. At the ankle, the soleus controls forward progression as body weight is loaded onto the limb.

Figure 17-5. Stair ascent muscle demands during SLS. Demand on the single joint hip and knee extensors progressively decreases as the joints assume a more extended position during SLS. Increasing activity of the semimembranosus and BFLH controls both hip and knee. Plantar flexors (soleus and medial gastrocnemius) increase activation.

Single Limb Support

Stable advancement of body weight from behind to in front of the stance limb is essential for progression during the SLS period of stair ascent (Figure 17-5). Additionally, continued lift of body weight must occur.

Contralateral toe-off initiates SLS and shortly after, the vertical GRF peaks (1.12 BW).[17] With each lower limb joint still flexed, extensor moments dominate the first half of SLS. Concentric activity of the single joint hip and knee extensors continues to extend the joints and lift the HAT. Bursts in power generation at the hip and knee peak at the onset of SLS and reflect the rapid extension of these joints.[15] As body weight progresses upward over the increasingly extended hip and knee, the need for dynamic muscle stabilization is replaced with passive stability arising in part from the alignment of the HAT relative to these joints. Internal extensor moments are replaced by internal flexor moments.[15] The hip and knee reach peak extension (ie, ~10° flexion at each joint) by the end of terminal stance[13,15] and the single joint hip extensors (ie, gluteus maximus, adductor magnus),[10,13] hip abductors (ie, gluteus medius and tensor fascia latae),[10] and single joint knee extensors (ie, vastus lateralis, vastus medialis)[6,7,13] are mostly quiescent. Now, increasing activity of the semimembranosus and BFLH provides the primary control at the hip and knee.[10,13] The ankle also contributes to elevating the body during SLS. Ankle DF progressively decreases during this period from a peak of 12° at the initiation of mid stance to

nearly neutral by the end of terminal stance.[13,15] The rapid rise in the plantar flexor moment during SLS[15] reflects the growing need for soleus and medial gastrocnemius activity to extend the ankle.[6,11]

Swing Limb Advancement

Contralateral foot contact on the stair above the reference limb signals the onset of SLA. Pre-swing prepares the limb for swing. It is a period of rapid PF as the ankle moves from a beginning posture of approximately 5° DF to a final position of 20° PF.[13,15] A peak in the plantar flexor moment[15] is accompanied by maximal activation of the medial gastrocnemius and soleus.[6,11] Concentric activity of the plantar flexors accompanies the notable burst of ankle power recorded during this period (2.2 to 2.5 W/kg).[12,15] Increasing PF in the trailing limb helps lift and shift body weight toward the upper lead limb. The hip and knee initially maintain their positions of minimal flexion (10°) achieved at the end of SLS, and then at the end of pre-swing begin to minimally flex. A rapid rise in BFSH activation during pre-swing assists knee flexion.[13]

When body weight is transferred to the contralateral limb, the reference limb is freed for swing. All 3 joints rapidly flex to ensure clearance of the foot over the risers (Figure 17-6).[13,15] By mid swing, peak flexion is achieved at the hip (~ 65° to 70° flexion) and knee (~80° to 95° flexion), and by terminal swing at the ankle (~ 10° DF).[13,15] During initial swing, the knee experiences a brief power burst as concentric activity of the BFSH peaks to flex the joint.[13] A burst of power at the hip during early mid swing accompanies the rapid increase in hip flexion. While a small percentage of subjects demonstrate a secondary peak in activity of the RF during this period,[6,11] the role of other hip flexors (eg, iliacus and psoas) has not been examined. TA activity also peaks during mid swing and assists with ankle DF.[6,11]

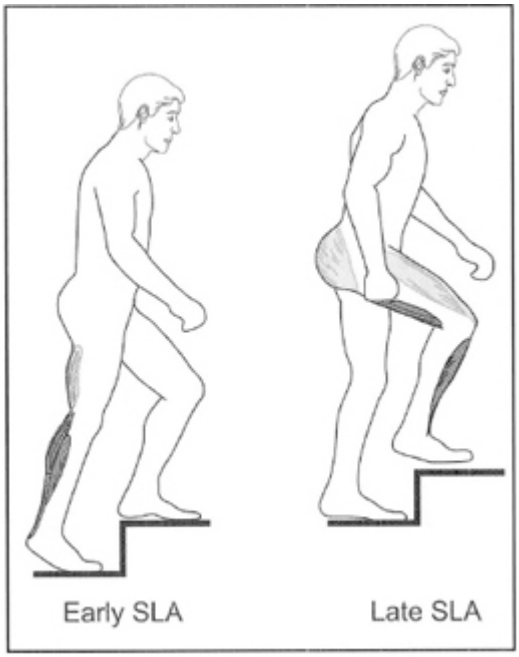

Figure 17-6. Stair ascent muscle demands during SLA. During early SLA, increasing PF activity in the trailing lim helps transfer body weight to the lead limb, while increasing BFSH activity facilitates knee flexion. As the foot lifts from the stair, BFSH and TA activation increases rapidly to ensure adequate flexion of the knee and ankle to clear the risers. Activation of the vastii and single joint hip extensor in late SLA prepares the limb for the demands of WA.

During the latter half of mid swing, the knee begins to extend as the limb reaches forward toward its final position of contact with the next step. Low-level activity of the vastus lateralis during terminal swing ensures adequate knee extension prior to IC and also prepares for the increased stability demands of WA.[11,13] At the hip, a subtle past retract (5° to 10°) during terminal swing positions the limb over the tread of the targeted step.[13,15] Activity of the hamstrings (ie, BFLH, semimembranosus, and semitendinosus) during this period restrains the forward motion of both the hip and knee.[11,13]

In summary, stair ascent is characterized by a series of joint motions and muscle actions that function to advance body weight forward and upward. Stability is provided primarily by the extensor muscles during stance. During swing, the flexor muscles initially help with rapid flexion of the joints to enable foot clearance and

advancement. Extension of the hip and knee at the end of swing prepares the limb for contact on the next higher riser.

STAIR DESCENT

During descent, the dominant functional task is to control the rate at which body weight drops. The greatest demand is placed on the knee. The ankle serves as the fulcrum for modification of limb alignment on the stair. The hip assists with SLA.

MOTION

Descent of the body to the next lower stair is accomplished primarily by increasing knee flexion (see Figure 17-2) and to a lesser extent dorsiflexing the ankle (see Figure 17-3).[13] Motion at the hip helps with lowering the body during the final portion of stance as well as advancing the limb during swing (see Figure 17-1).[13]

The downward step begins with the limb relatively extended at each joint. At the instant the forefoot initiates floor contact, the ankle is plantar flexed approximately 20°, the knee flexed about 10 to 15°, and the hip flexed approximately 20°.[1,13-15] During loading response, the ankle abruptly reverses to 10° DF and then slowly advances to 20° DF by the end of terminal stance.[13,14] Knee flexion increases throughout stance. The rate is gradual during loading response, with existing knee flexion augmented only 10° by the end of the phase. Knee flexion is more rapid during terminal stance and pre-swing, advancing the joint to between 80° and 90°.[13-15] Despite appreciable flexion of the knee and ankle during stance, the hip remains stable in approximately 15° to 20° of flexion throughout most of SLS. Then during pre-swing, hip flexion increases 15° as body weight transitions to the opposite limb.[13-15]

When the foot leaves the ground for swing, the ankle progressively plantar flexes, reaching a peak of approximately 20° by the end of terminal swing.[13-15] At the knee, further flexion abruptly

terminates during initial swing and is replaced by rapid extension through the remainder of swing.[13-15] Similarly at the hip, the flexion wave crests during initial swing (peak of 40°) and then the joint gradually extends to reach a position of 15° to 20° flexion prior to the next IC.[13-15]

Muscle Control

During the stance period of stair descent, coordinated muscle activity controls the body's lowering and advancement. In swing, joint motions are synchronized to ensure foot clearance and subsequent placement of the foot within the constraints of the next lower step.

Co-contraction of antagonists at the ankle provides stability during stance. Medial gastrocnemius followed by TA are activated during the latter half of swing in preparation for their stabilizing role during WA.[6,7,11] Shortly after IC, medial gastrocnemius activity peaks then rapidly declines and ceases during early SLS.[6,11] Now, TA in combination with rising soleus action becomes the dominant force assisting with balance control as body weight transitions over the limb.[6,11] During terminal stance, TA and soleus activity decline notably to near quiescence. As the foot prepares to lift from the floor during pre-swing, TA activity abruptly rises again. Initial swing is the period of greatest TA action.[6,11]

At the knee, the WA demands for stability and shock absorption are met by a substantial increase in muscle activity. RF (25% MMT),[6,11,13] vastus lateralis (25% MMT),[11,13] and vastus medialis[6,11,13] each experience a peak in activity near the time when the contralateral foot lifts from the ground[6,11] and then continue at a moderate level throughout SLS (10% to 20% MMT).[13] During the first third of stance, many individuals co-contract their hamstrings (BFLH, semimembranosus, and semitendinosus), but the demand on the hamstrings rapidly declines by mid stance.[11,13] Soon after the onset of pre-swing, BFSH activity is initiated, sharply rises (peak of 55% MMT during pre-swing), and then declines to a level of approximately 20% MMT, which is maintained through the end of mid

swing.[13] Starting in initial swing and continuing into the next GC, hamstring activity (BFLH, semimembranosus, and semitendinosus) assists with limb clearance and positioning the leg on the next lower step.[11,13]

The single joint extensor of the hip (lower fibers of the gluteus maximus) and the abductors (gluteus medius, upper gluteus maximus, and tensor fasciae latae) are primarily active at the transition from swing to stance.[10,13] Maximum activation in each muscle occurs just prior to or just after IC.

FORCES

Ground Reaction Force

The profile of the vertical GRF during stair descent consists of primarily one peak.[8,11,14,15,17] This crest (1.56 BW) occurs during loading response and notably exceeds the peaks occurring during the same period with stair ascent (1.12 BW) and level walking (1.19 BW).[17] By mid stance, the vertical GRF drops to a level less than body weight and stays below this threshold throughout the remainder of stance.[11,14,15,17]

The AP GRF during stair descent demonstrates a similar pattern of reversals to that recorded during level walking but is reduced in amplitude.[15] The resultant GRF is directed in a posterior direction during WA,[15] helping to resist forward slippage of the foot on the stair.[2,3] During terminal stance and pre-swing, the anteriorly directed GRF resists posterior foot slippage.[2,3,15]

Moments

Stair descent moments have a much different pattern than those calculated during stair ascent. The hip registers no extensor moments during stance but has 2 small flexor peaks. One occurs during loading response (0.5 N•m/kg) and the other at the transition

between terminal stance and pre-swing (0.6 N•m/kg).[11,15] Early swing is characterized by an even lower amplitude flexor moment,[11,15] while late swing may include a brief low-amplitude extensor moment.[11] During stance, the ankle and knee are dominated by extensor moments with 2 peaks. At the ankle, the first peak at the end of loading response (~1.2 N•m/kg) is of greater amplitude than the one occurring in terminal stance (0.8 N•m/kg).[11,15] The opposite pattern occurs at the knee, with the first peak extensor moment that happens at the beginning of mid stance (~1.1 N•m/kg) being of smaller magnitude than the one in terminal stance (~1.4 N•m/kg).[11,15]

Power

The predominance of power absorption occurring at the knee and ankle during the stance period of stair descent differs markedly from the power generation pattern observed during stair ascent.[11,15] Peak power absorption at the knee (-4.0 W/kg) occurs at the onset of pre-swing.[15] The ankle's time of maximum power absorption is loading response (-2.5 W/kg at approximately 10% GC).[15] In addition, a brief, low-amplitude burst of power generation occurs at the ankle during pre-swing (approximately 1.3 W/kg at 55% GC). At the hip, the first half of stance is characterized by low-level power absorption (peak, -0.3 W/kg at ~ 18% GC). However, the hip generates power (peak, 0.4 W/kg at ~45% GC) during the remainder of stance and initial swing.[15]

Figure 17-7. Stair descent muscle demands during WA. Medial gastrocnemius and anterior tibialis co-contraction stabilizes the plantar flexed ankle joint. Moderate knee extensor activity (vastii and RF) and co-contraction of the hamstrings stabilize the knee and assist with shock absorption. The hip is stabilized by the gluteus maximus, gluteus medius, and TFL.

FUNCTIONAL SIGNIFICANCE OF STAIR DESCENT

Going down a staircase presents a greater need for mobility than strength as muscle force is primarily needed to control the rate of body lowering. The successive “drop” of body weight onto each lower stair results in a high vertical GRF that must be dissipated to ensure adequate shock absorption. Additionally, restraint of forward progression is required to ensure that each foot lands within the constraints of the next lower stair.

Weight Acceptance

Contact of the forefoot with the next lower step initiates rapid loading of the reference limb (Figure 17-7). Reflective of the high rate of loading, the vertical GRF peaks earlier and exceeds values registered during the same period for both level walking as well as stair ascent.[15,17]

Shock is absorbed and the body is lowered in a controlled manner, in part, by rapid ankle DF, which moves through a 30° arc during loading response (from 20° PF to 10° DF).[13,15] The plantar flexor moment and activity of the medial gastrocnemius peak to ensure the ankle remains stable.[6,11,15] The combination of rapid motion of the ankle and a peak plantar flexor moment result in maximum power being absorbed at the ankle during this period (-2.5 W/kg at 10% GC).[15]

The knee also controls lowering of the HAT. A 10° increase in knee flexion[13,15] during this period assists with shock absorption. Growing activity of the knee extensors (RF and vastus lateralis, ~25% MMT)[6,11,13] prevents the knee from collapsing. This period is the first and the smaller of 2 stance-phase episodes of power absorption at the knee (-1.0 W/kg).[15]

Figure 17-8. Stair descent muscle demands during SLS. TA and soleus co-contraction during the first half of SLS provide a stable base for progression of body weight. Moderate vastii and RF activity stabilizes the knee.

In contrast to the notable arcs of motion occurring at the ankle and knee, the hip's initial position (~ 20° flexion) does not change appreciably during WA.[13,15] Peak activity of the gluteus maximus, gluteus medius, and tensor fasciae latae near the time of IC ensure that the hip provides a stable base for the HAT in both the sagittal and frontal planes.[10,13] Hamstring activity (BFLH, semimembranosus, and semitendinosus) augments stabilization of the hip.[11,13]

Single Limb Support

From the instant the contralateral limb lifts from the stair above until it contacts the stair below, the stance limb must assume primary responsibility for controlling the fall of body weight (Figure 17-8). At the ankle, DF continues to advance and lower the HAT, but at a more restrained rate (only a 10° increase during all of SLS).[13,15] The decrease in the plantar flexor moment during this period reflects reduced soleus activity and simultaneous co-activation of the TA during early SLS.[6,11]

Following an initial plateau in knee movement during mid stance, the joint commences a period of flexion that lowers the HAT and contralateral limb toward the next stair during the remainder of SLS.[13,15] The rate of flexion is restrained by the vastii and RF,[6,7,11,13] which contribute to peaks in the knee extensor moment at both the beginning of SLS (~1.1 N•m/kg) and again at the end (~1.4 N•m/kg).[11,15] Power absorption at the knee reaches a maximum at the transition from terminal stance to pre-swing (-4.0 W/kg).[15]

Similarly, at the hip there is an initial plateau in movement during the first half of SLS as the contralateral limb starts to swing forward from the stair behind. Then hip flexion increases steadily to help lower the HAT and opposite limb toward the stair below. The flexor

moment peaks at the end of SLS (0.6 N•m/kg),[11,15] reflecting in part the increase in RF activity recorded during the same period.[6] Activity of other hip flexors (eg, iliacus and psoas) has not been documented during stair descent, hence their contribution to the flexor moment is not yet known. Generation of power at the hip is greatest at the end of SLS.[15]

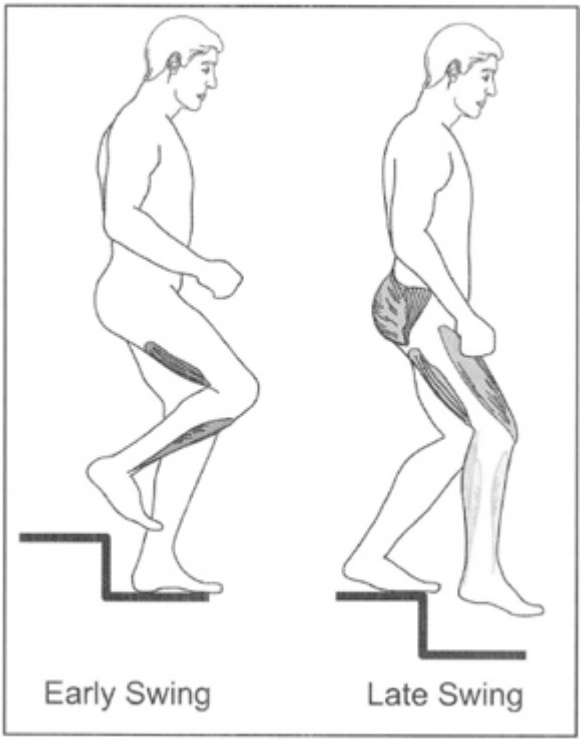

Figure 17-9. Stair descent muscle demands during SLA. Vigorous TA and BFSH activity during early swing (pre and initial) ensures foot clearance during swing. Activation of the hamstrings (semimembranosus, BFLH) in terminal swing helps position the limb within the confines of the next lower step. Rising activation of the medial gastrocnemius, TA, vastii, RF, gluteus maximus, gluteus medius, and tensor fasciae latae prepares the limb for the demands of WA.

Swing Limb Advancement

Joint motions during swing are synchronized to ensure foot clearance and subsequent placement of the foot within the confines

of the next lower step (Figure 17-9). Transfer of body weight to the opposite lower leg frees the trailing limb ankle to rapidly plantar flex. As plantar flexor activity during this period is minimal,[6,11,15] elastic recoil arising from residual tension in the plantar flexors is likely a key factor leading to the brisk 15° of PF (from 20° DF to 5° DF) and low amplitude burst of power generation (approximately 1.3 W/kg at 55% GC) that occurs during pre-swing.[15] Subsequently, as the foot prepares to lift from the step, TA activity rapidly rises to help control the posture of the ankle. A brief plateau of the ankle at neutral (0° PF) during initial swing ensures that the foot clears the stair, then the ankle resumes progressive PF throughout the remainder of swing.[6,11] By the end of terminal swing, the ankle is plantar flexed 20° with the forefoot positioned over the lower step.[13-15] Co-contraction of the plantar flexors and dorsiflexors prepares the ankle for its stabilizing role during WA.[6,7,11]

Transfer of body weight to the opposite lower limb is accompanied by the reference knee rapidly flexing during pre-swing.[13-15] To ensure that balance and stability are preserved, vastus lateralis activity controls the rate of knee flexion during early pre-swing.[13] Peak power absorption at the knee occurs during this period (-4.0 W/kg).[15] At approximately the same time as the vastus lateralis ceases activity, BFSH activity is initiated and sharply rises to a peak of 55% MMT by the end of pre-swing.[13] The BFSH provides a dynamic force to ensure adequate knee flexion (a peak of 80° to 90°) is achieved during initial swing for foot clearance. The knee then progressively extends throughout the remainder of swing.[15] Hamstring activity (BFLH, semimembranosus, and semitendinosus), initiated in initial swing and continuing until the following period of stance, controls the rate of knee extension and helps position the leg on the next lower step.[11,13]

Similar to the knee, the hip also goes through a period of increasing flexion as body weight is lowered during pre-swing onto the contralateral limb.[13-15] By initial swing, the hip achieves a peak position of 40° flexion and then gradually returns to 15° to 20° flexion prior to the next IC.[13-15] Swing phase hamstring activity (BFLH,

semimembranosus, and semitendinosus) likely restrains advancement of the thigh and helps position the limb on the next stair.[11,13] During terminal swing, activation of the single joint extensor of the hip (lower fibers of the gluteus maximus) and the abductors (gluteus medius, upper gluteus maximus, and tensor fasciae latae) also prepares the limb for the stability demands associated with body weight rapidly dropping onto the limb following IC.[10,13]

In summary, stair descent requires controlled lowering of body weight. During WA, shock is absorbed and body weight is lowered as the ankle dorsiflexes and the knee flexes. The hip remains stable. In SLS, the significant arc of knee flexion and to a lesser extent hip flexion and ankle DF control the descent of the HAT. During SLA, the knee and hip initially further flex as weight is lowered onto the opposite limb and the foot lifts from the stair. Once in swing, the hip, knee, and ankle nearly simultaneously move toward extension as the limb prepares to contact the targeted stair.

INFLUENCE OF ENVIRONMENTAL AND HUMAN FACTORS ON STAIR DEMANDS

Stairs are a common obstacle in both home and community environments. The biomechanical requirements associated with successful stair negotiation are influenced not only by environmental factors (eg, stair height and depth) but also human anthropometrics (eg, height and age).

Environmental Factors

Some cities have building codes for staircases with a greater or lesser slope than the criteria for the “standard” staircase.[9] Three investigators cited in this chapter used adjustable stair cases that facilitated evaluation of stair negotiation across 3 levels of steepness

approximating 20°, 30°, and 40° angles of inclination (see Table 17-1).[9,15,17] The steeper flight of stairs had corresponding greater riser heights and shorter tread depths, while the shallow staircases has the reverse dimensions of a low riser and deeper treads. The magnitude of the differences, however, still remained compatible with Blondel's formula (Tread + 2•Riser ≈ 60 cm).[5,9] Several gait parameters are influenced by staircase inclination.

Both the ROM and the maximum flexion angles of lower limb joints increase as the staircase becomes steeper.[15] Hip flexion (range and angle) increased 12.4% during ascent and 15.7% during descent, yet the difference in stair slope between the most shallow and the steepest staircase was 75%.[15] During stair ascent, maximum knee flexion increased 12.1% and descent knee flexion increased 14.3% on the steepest staircase.[15] Peak ankle PF increased 25.0% during ascent and 17.3% while descending the steeper staircase. These findings suggest that the 3 major joints of the lower extremity collectively share in accommodating to the demands presented by staircases of differing inclines.

Only the vertical component of the GRF was significantly impacted by the staircase slope during stair descent.[15] During early stance, the peak vertical GRF was 14.8% greater when negotiating the steep versus shallow staircase. The path of the COP remained in the metatarsal area during both stair ascent and descent and did not vary significantly in length (~10 cm) across stair inclinations.[15]

Sagittal plane joint moments increased significantly when the staircase became steeper except at the hip during descent.[15] During ascent, the maximum hip extensor moment was 37.9% greater on the steepest compared to shallowest staircase. In contrast, during descent, the peak hip flexor moment lessened 27.1% with greater angles of inclination. Subtler differences were also documented at the knee, with steeper staircases resulting in higher extensor moments during both ascent (10.6% in early stance) and descent (18.4% in late stance). The steepest staircase also resulted in the greatest early stance ankle plantar flexor moments for both ascent (12.8%) and descent (18.7%).

Joint powers were more sensitive to changes in staircase inclination than either motion or moments.[15] Power generation was greatest at the hip (51.7%, early stance), knee (25.1%, early stance), and ankle (45.4%, late stance) while ascending the steepest compared to shallowest staircase. During descent, steeper stairs were associated with more power generation at the hip (24.3%, late stance) yet it was power absorption that increased at both the knee (26.3%, late stance) and ankle (67.3%, early stance).

Staircase inclination also dictates a subject's stride characteristics as the structural boundaries of a stair limit the length of the individual's step and thus the stride length when negotiating stairs.[9,15] The more vertical the slope, the shorter the stride length (steep, 0.58 m; standard, 0.73 m; shallow, 0.88 m).[9] Step rate was fastest on stairs with a steep slope and slowest on stairs with a shallow slope.[9] During descent, the percentage of time spent in stance decreases as the angle of inclination increases.[9,15] During ascent, the slope does not notably impact the stance duration.[9,15]

Human Factors

An individual's height influences joint motions and stride characteristics as evidenced in a study among 3 groups of 5 women classified as short, medium, or tall.[9] During stair ascent, shorter subjects used greater maximum knee flexion (92° to 105°) than taller participants (83° to 96°). Differences were not statistically significant during descent. Subject height influenced step rate during stair descent even though limb length was not strictly proportional to body height. The shorter women (1.56 m) had a faster cadence (126 to 140 steps/minute) than the taller (1.72 m) women (107 to 115 steps/minute).

Stair negotiation imposed a greater muscular demand on older versus younger adults.[8] Healthy community dwelling older (mean age, 72 years) and younger (mean age, 26 years) women negotiated a 9-step staircase (rise = 16 cm; depth = 23 cm; no rail) at a predetermined speed of 35 steps/minute while surface EMG was

recorded. During both stair ascent and descent, the older women's muscle activation (expressed as a percentage of maximum voluntary contraction) was approximately two-fold higher than the younger females in the vastus lateralis, vastus medialis, RF, biceps femoris, semitendinosus, and TA. During stair descent, muscle activity also was greater in the gastrocnemius and soleus in the older versus younger women.

CONCLUSION

Essential to both stair negotiation and overground walking is the requisite accomplishment of stability, forward progression, shock absorption, and limb clearance during each GC. Differing between the tasks, however, are the patterns and magnitudes of joint motions and muscle activation necessary to produce synchronized and uninterrupted movements. Compared to walking on a level surface, the increased arcs of motion required at each joint during stair ascent and descent may lead to challenges for clients lacking adequate range. The only exception was at the hip during stair descent, which presented a lower demand for joint flexion. Muscle demands in the extensors were generally higher during stair ascent than descent, which may present difficulties for persons with lower extremity weakness.

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Chapter 18

Running

Marilyn M. Pink, PhD, PT

Now that you understand walking inside and out, does that necessarily mean you understand running? Hmmm… not exactly. While running is a natural extension of walking, the magnitude of difference mandates that running be studied separately. Therefore, the purpose of this chapter is to first describe the terminology and timing of the running phases, then to describe the ROM and muscle activation patterns in the lower extremity while running, followed by a description of the pressures borne by the foot. These discussions will focus on the "training pace" of recreational runners (approximately a 6.5-minute mile) in order to offer the widest applicability to the clinician. Once the mechanics of the training pace are discussed, then the relative differences to faster and slower paces will be introduced.

TERMINOLOGY AND TIMING OF RUNNING

The running cycle has 4 functional periods: stance, early float, mid swing, and late float. The 2 "float" periods during running are the events that differentiate it from walking. During float, neither foot is

on the ground. The float period follows each stance period because toe off by the trailing limb takes place before the leading foot contacts the ground. The stance interval of running is a period of SLS, while mid swing is the airborne period that occurs during the same time the other limb is in stance. During running, there are no periods when both limbs are simultaneously in contact with the ground (ie, double limb support).

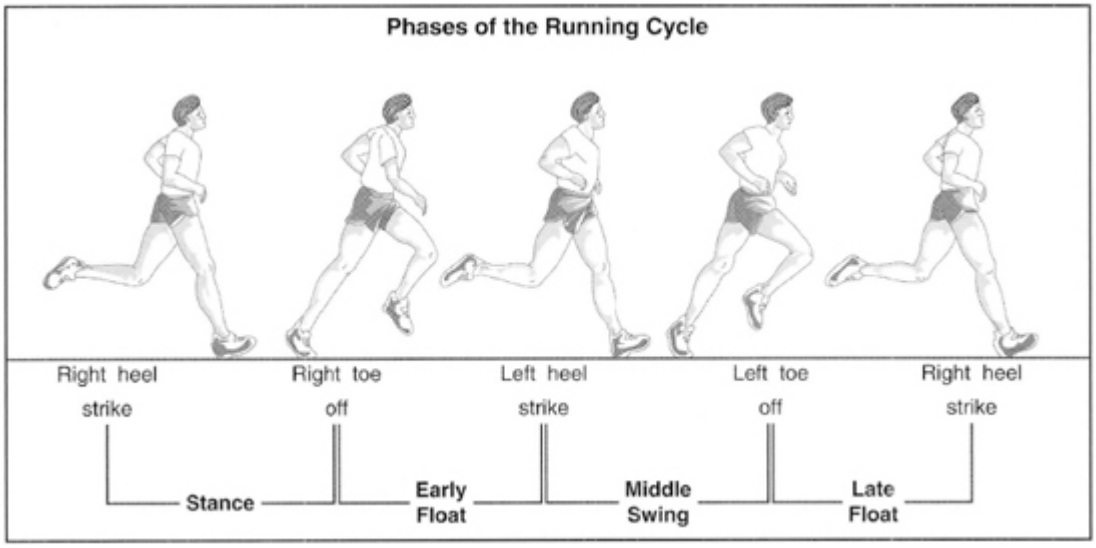

Figure 18-1. Phases of the running cycle. (Reprinted with permission from Reber L, Perry J, Pink M. Muscular control of the ankle in running. *American Journal of Sports Medicine*. 1993;21(6):805-810.)

Reflective of the shortened stance periods (and lengthened swing periods), the ratio of stance to swing time in running is 35:65 in contrast to the typical ratio of 60:40 in walking. Approximately 35% of the time is spent in single leg stance, while swing (airborne) includes the 3 intervals of early float (neither foot on ground, 15% cycle), middle swing (opposite limb in single stance, 35% cycle), and late float (neither foot on ground, 15% cycle). The exact percent of time in each period varies depending upon the pace of running, and thus the values are offered as an approximation. The timing differences between walking and running necessarily affect both ROM and muscle demand.

A typical sequence of periods in one running cycle is presented for the right limb (Figure 18-1).

* Stance: 35% GC
  Single limb stance
  Right heel strike to right toe off
* Early float: 15% GC
  Right toe off to left heel strike
* Middle swing; 35% GC
  Contralateral limb is in single leg stance
  Left heel strike to left toe off
* Late float: 15% GC
  Left toe off to right heel strike

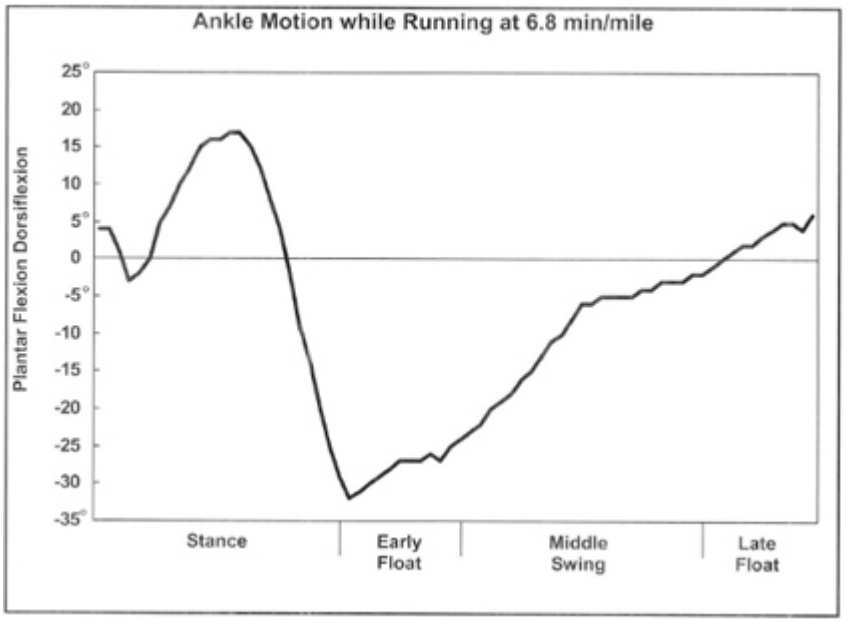

Figure 18-2. (A) Ankle motion while running at 6.8 min/mile. (Reprinted with permission from Pink M, Perry J, Houglum P, Devine D. Lower extremity range of motion in the recreational sport. *American Journal of Sports Medicine*. 1994;22(4):541-549.)

STANCE

Stability and shock absorption during body WA are the 2 key functions of the lower extremity in stance during running. Between 75% to 90% of runners make IC with the heel in stance.[5,8,28] The others either make contact with the foot flat or the forefoot. The events during running parallel the basic phases of walking, but the rapid rate at which body weight is dropped onto the heel compresses the stance period into 3 phases. Loading response includes the same events as walking but they differ in magnitude. Mid stance is a brief transition interval. Terminal stance is the final weight-bearing period, as there is no double limb support (ie, pre-swing).

Motion

The impact of IC on the heel at the onset of stance has a direct influence only on the motion at the ankle (Figure 18-2A). The knee and hip merely respond to the limb's total load (Figures 18-2B and 18-2C, respectively). During the first 15% of stance, the ankle responds to the heel rocker with a quick move through an 8° arc into PF from its initial dorsiflexed position. Then, following forefoot contact, the ankle abruptly reverses into DF. The knee, from an initial 15° flexed position, increases its flexion.[20] The hip stays in flexion, with minor variations in the degree (15° to 25°).[20]

Peak ankle DF (20°) and flexion of the knee (40°) and hip (25°) are reached by the middle of stance. At this point, the COG is vertically at its lowest position and the body weight is directly over the foot. Following a plateau, which varies in magnitude and duration, the 3 joints almost simultaneously rapidly lose flexion. The ankle moves into 30° PF. Flexion of the knee decreases to 15° and the hip achieves 10° of hyperextension.

Muscle Control

As the foot accepts body weight, the posterior lower leg muscles function in concert[21] (Figure 18-3). The muscle activity in the gastrocnemius and soleus reaches amplitudes similar to that in

walking as they control the forward fall of the tibia. The timing of these muscles is such that the soleus reaches peak action slightly before the gastrocnemius. The slightly prolonged activity of the gastrocnemius reflects its muscular origin above the knee and its function as a knee flexor to prevent knee hyperextension during the latter half of stance. Knee flexor force is augmented by activity of the BFSH.

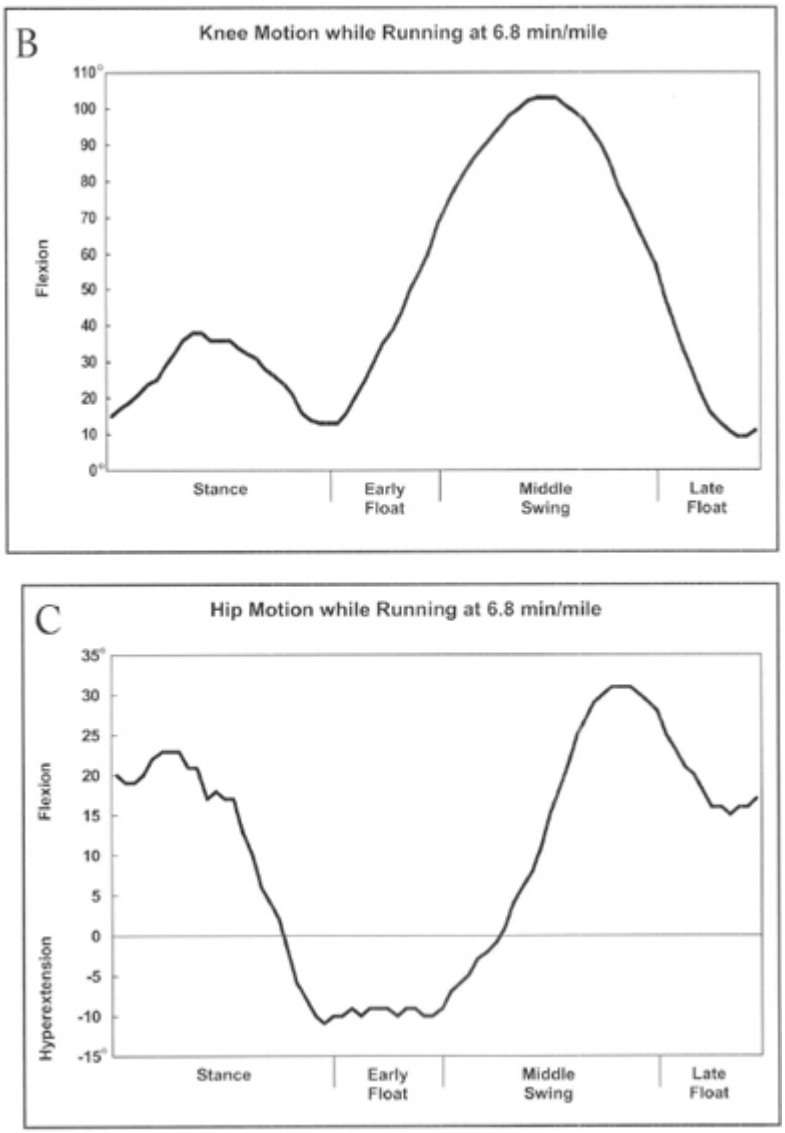

Figure 18-2. (B) Knee motion while running at 6.8 min/mile. (C) Hip motion while running at 6.8 min/mile. (Reprinted with permission from Pink M, Perry J, Houglum P, Devine D. Lower extremity range

of motion in the recreational sport. *American Journal of Sports Medicine*. 1994;22(4):541-549.)

While this is happening, activity of the TP and peroneus brevis exceeds the ambulatory amplitudes by 150% to 400%.[18,21] The TP peaks first, followed by the peroneus brevis. The TP controls the normal pronation forces during early stance by stabilizing ST motion.

As the COG continues to progress anteriorly, it is still medial to the ST axis. Thus, the laterally placed peroneus brevis increases its activity to control ST motion. While the primary function of the TP and peroneus brevis is control of the ST joint, the compressive forces generated by the abrupt turn of their tendons around the malleoli may assist the soleus and gastrocnemius in controlling forward fall of the tibia.

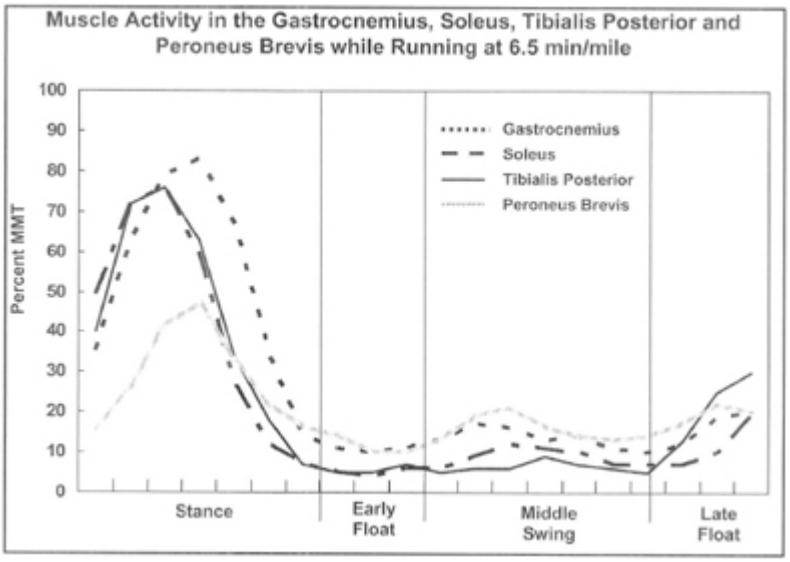

Figure 18-3. Muscle activity in the gastrocnemius, soleus, TP, and peroneus brevis while running at 6.5 min/mile. (Reprinted with permission from Reber L, Perry J, Pink M. Muscular control of the ankle in running. *American Journal of Sports Medicine*. 1993;21(6):805-810.)

In summary, as the body falls forward and downward, the posterior lower leg muscles control the forward advancement of the tibia. Additionally, the TP affords support to the longitudinal arch of the foot

and the peroneus brevis supports the lateral aspect of the ankle, all in an attempt to control ST motion.

It must be noted that all of the posterior calf muscles reach their peak EMG by the middle of stance and then activity rapidly diminishes. This EMG simply reinforces the fact that muscle contractions do not create the force of “push-off” at the end of stance. Recent ultrasound studies, however, have identified that the tendons and aponeuroses of muscles (ie, the series elastic elements or SEE) create a recoil burst of energy and augment push-off.[6] The recoil energy is generated during the first half of stance when the gastrocnemius and soleus exert a strong eccentric contraction to stabilize the ankle against the moment created by forward fall of the body’s COM. As the muscles vigorously stabilize the ankle by isometric contraction, the tendon and aponeurosis are stretched. In the latter half of stance, rapid upward and forward movement of the contralateral swinging limb reduces the load on the stance limb calf. The stored recoil energy is then sufficient to rapidly plantar flex the foot. Hence, recoil tension in the aponeurosis and tendon provides a non-contractile force (no EMG) for push-off at the end of stance.[6]

During stance in running, the TA demonstrates less intensity than it does in walking; however, it is constantly active[21] (Figure 18-4). Two factors lessen the need for a more vigorous anterior tibialis. The ankle is already dorsiflexed. Also, body weight moves rapidly across the foot, curtailing the effect of the heel rocker.

The patterns of muscle activation of the vastii during running are quite similar to that of walking[13,18] (Figure 18-5). These muscles peak in loading response. At this point, there is an extensor moment and the quadriceps helps control the amount of knee flexion with an eccentric contraction as well as absorb the shock at impact. The peak amplitude of the vastii greatly exceeds that of walking by about three-fold. While the knee flexes 18° during WA in walking, the knee flexes 38° in running. Among the heads of the vastii, the medialis and lateralis demonstrate more activity than the intermedius. The oblique line of pull from the medial and lateral vastii appears to be more important in stabilizing the patella in the trochlear groove than the straight line of pull from the intermedius muscle.

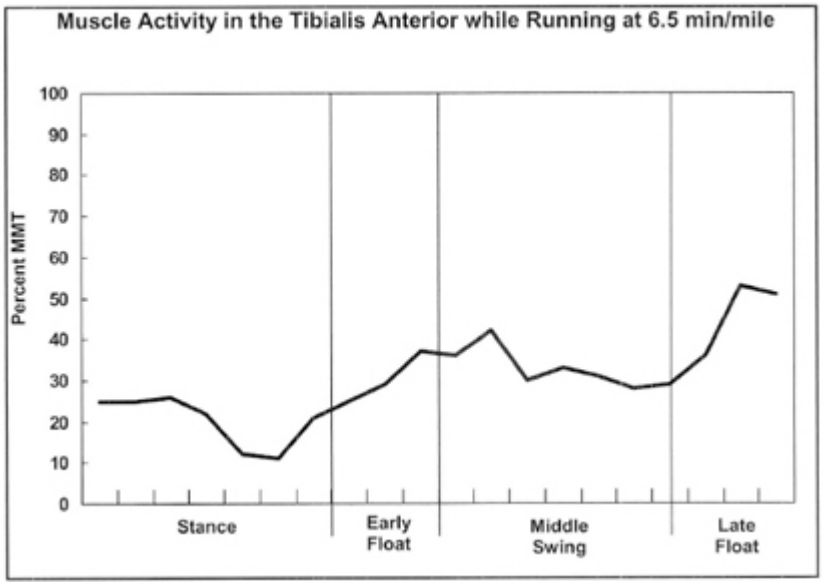

Figure 18-4. Muscle activity in the tibialis anterior while running at 6.5 min/mile. (Reprinted with permission from Reber L, Perry J, Pink M. Muscular control of the ankle in running. *American Journal of Sports Medicine*. 1993;21(6):805-810.)

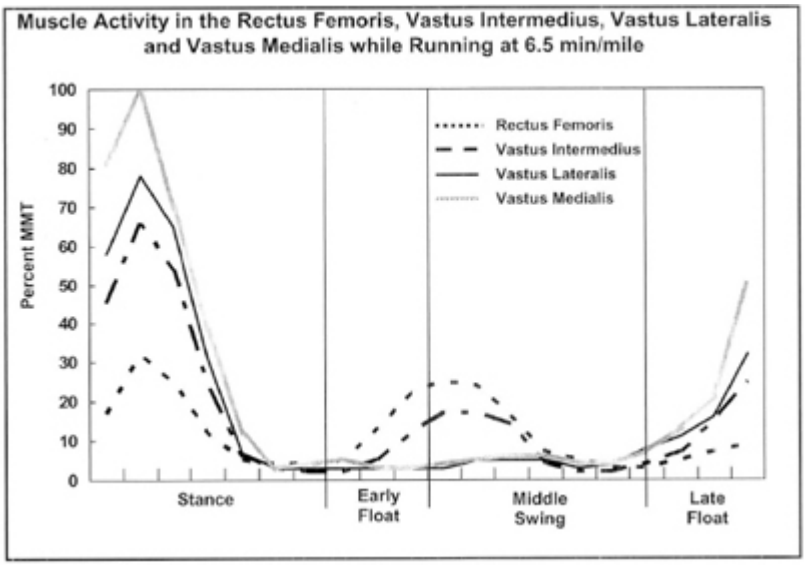

Figure 18-5. Muscle activity of the RF, VI, vastus lateralis, and vastus medialis while running at 6.5 min/mile. (Reprinted with permission from Montgomery WH, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. *American Journal of Sports Medicine.* 1994;22(2):272-278.)

Even the RF, which is silent during WA in gait, demonstrates a peak in running of approximately 30% MMT[12] (see Figure 18-5). This may reflect the increased total force in running as compared to walking (2 to 3.6 times the force in walking).[22,28] Without this contraction of the quadriceps, the knee would undoubtedly buckle as it accepts body weight.

After WA, the quadriceps muscle activity begins to diminish.[13] The activity lessens before the knee reaches the greatest degree of flexion.[8] This is because the advancing body vector serves as a knee extensor, thus less action is required of the quadriceps.

All of the hip extensors are active as the foot accepts body weight. The lower gluteus maximus peaks at IC (41% MMT; Figure 18-6) as does the adductor magnus (58% MMT) (see Figure 18-6). The large size and the dual innervation of the adductor magnus allows it to function as both a hip extensor as well as an adductor for hip/pelvic stability. The BFLH is active in IC (35% MMT) (see Figure 18-6) and continues its activity into the middle of stance (52% MMT) as it, along with the short head, controls the rate of knee extension (Figure 18-7). The semimembranosus demonstrates less activity than the BFLH during WA (25% to 30% MMT) (see Figure 18-6), and then continues to decrease.[13] At this point, the contralateral swinging limb is advancing, and thus passively rotating the unloaded pelvis forward. Therefore, the need for the stance leg semimembranosus activity to assist with rotation is reduced. Also, the swing motion increases the adducting strain on the knee. Hence, increased lateral stability is provided by the biceps femoris.

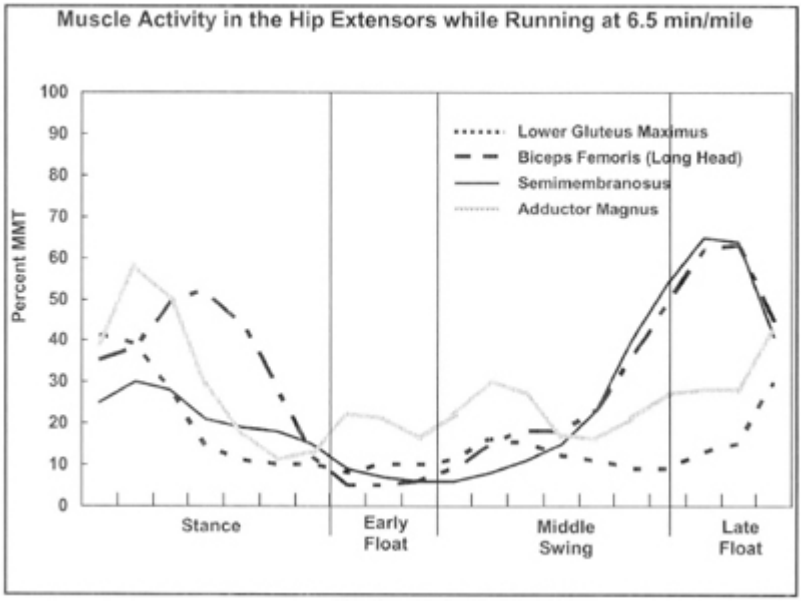

Figure 18-6. Muscle activity of the hip extensors while running at 6.5 min/mile. (Reprinted with permission from Montgomery WH, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. *American Journal of Sports Medicine.* 1994;22(2):272-278.)

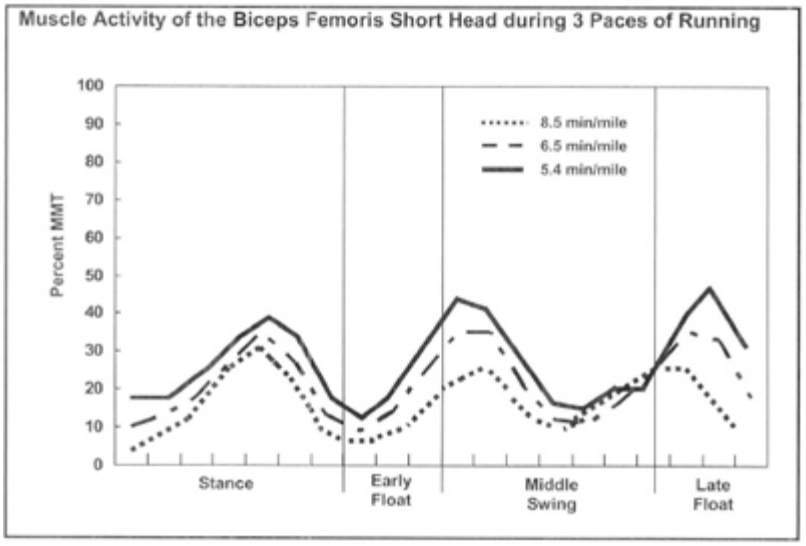

Figure 18-7. Muscle activity of the BFSH during 3 paces of running at 8.5 min/mile (jog pace); 6.5 min/mile (train pace); 5.4 min/mile (race pace). (Reprinted with permission from Montgomery WH, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. *American Journal of Sports Medicine.* 1994;22(2):272-278.)

The tensor fascia lata also increases its function relative to walking by 30% to 100% during WA (Figure 18-8).[13] The tensor fascia lata along with the adductor magnus provides medial/lateral stability of the hip and pelvis. Neither muscle becomes inactive during stance since that stability is a requisite in weight bearing.

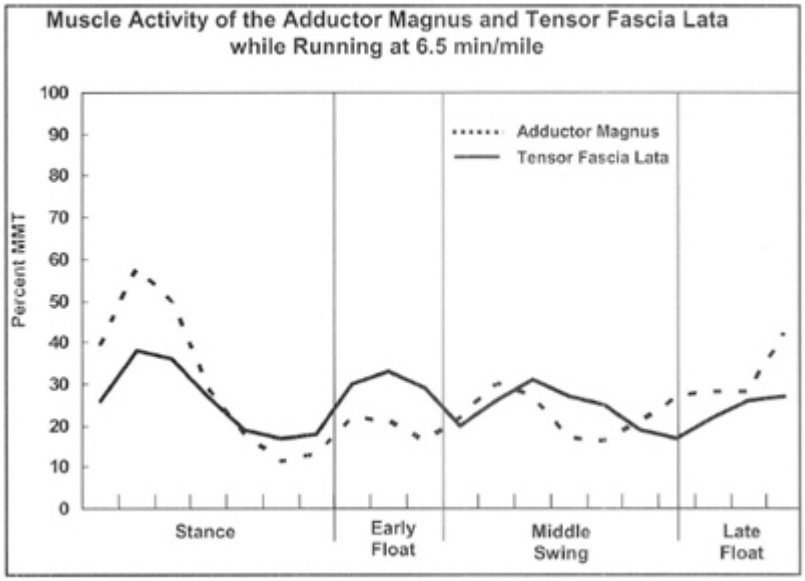

Figure 18-8. Muscle activity of the adductor magnus and tensor fascia lata when running at 6.5 min/mile. (Reprinted with permission from Montgomery WH, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. *American Journal of Sports Medicine.* 1994;22(2):272-278.)

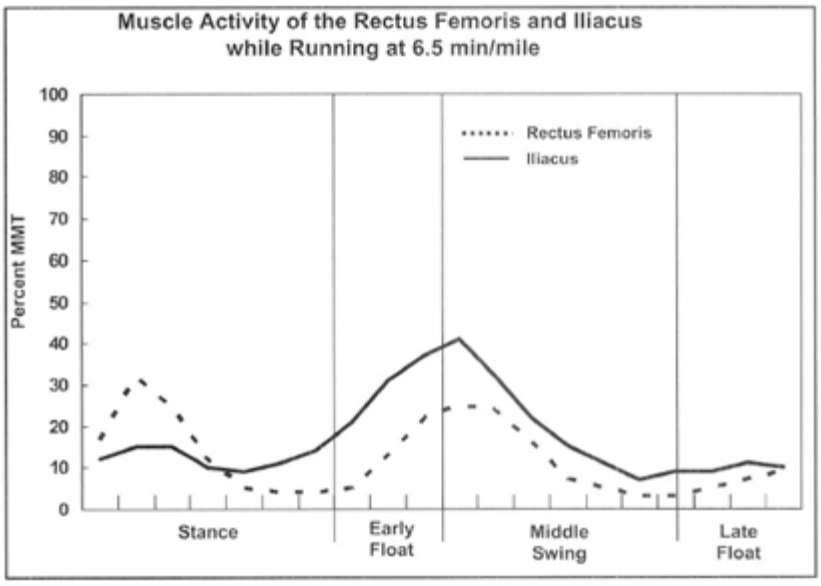

Figure 18-9. Muscle activity of the RF and iliacus when running at 6.5 min/mile. (Reprinted with permission from Montgomery WH, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. *American Journal of Sports Medicine.* 1994;22(2):272-278.)

The iliacus demonstrates relatively little activity in stance[13] (Figure 18-9). It begins to increase its action at the end of stance as it prepares to flex the hip during the swing phases.

PRESSURE

As previously mentioned, the majority of runners make ground contact with the heel. Yet, this is not the point of maximum pressure. The second metatarsal head bears the largest degree of pressure in the foot, closely followed by the first and third metatarsal heads and extending up to the great toe (Figure 18-10).[19] The same areas not only bear the peak instantaneous pressure, but also bear the pressure over a relatively longer time than the other parts of the foot. Pressure is on the heel for a shorter period of time. Even though the heel may typically be the first part of the foot to come in contact with the ground, relatively little pressure is encountered when running.

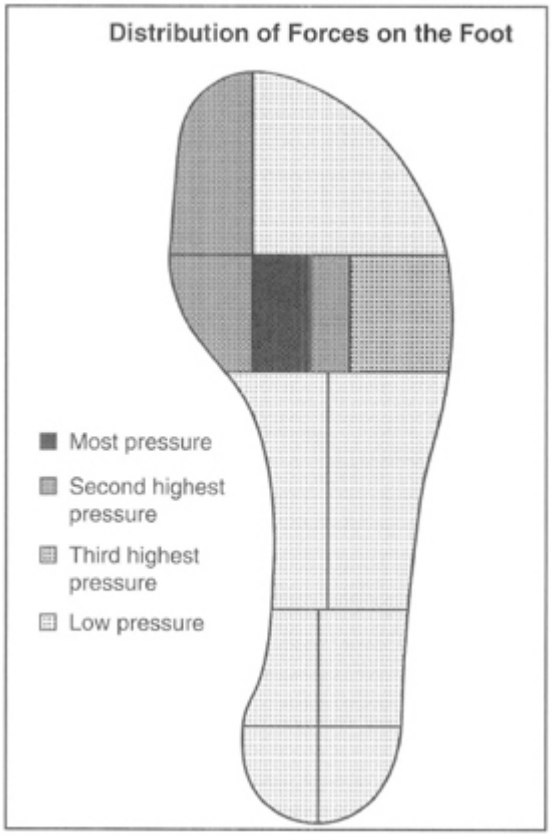

Figure 18-10. Distribution of pressures under the foot while running. (Reprinted from *Running Injuries*, Guten GN, 20-29, © 1997, with permission from Elsevier.)

The fact that the posterior-lateral outsole of a running shoe commonly demonstrates signs of wear appears to contradict the researched results of pressure distribution. The posterior-lateral outsole wear pattern is the result of anterior-posterior shear forces (as opposed to pressure) as the foot contacts the ground.

The peak vertical force in running occurs during the middle of stance.[3,4,14] At this point, the heel is typically off the ground. This observation supports the fact that the highest pressure occurs in the forefoot rather than in the heel.

The calcaneus is a much larger surface area over which to distribute forces than are the metatarsal heads. The fat pad under the calcaneus is relatively thick. The heel thickness of Whites is approximately 17.8 mm and of African Americans is approximately 20.1 mm.[25] These fat pads may decrease the magnitude of peak forces by 20% to 28%.[16] Although the fat pads at the metatarsal

heads have not yet been measured, they are qualitatively less than at the heel. The surface area is smaller at the metatarsal heads, they have less biologic cushioning, and less attention is typically focused on the metatarsal heads in shoe design as well.

Thus within stance, the majority of the muscles are most active during the first half of stance in order to accept body weight and absorb shock. The only muscles (of the 16 tested) that demonstrate their peak stance activity after the COG moves in front of the knee are the BFSH (which is controlling knee extension just after mid stance) along with the iliacus (which is preparing the limb to advance and clear the ground during the forthcoming swing phases).[13,21] The peak vertical force is approximately halfway through stance, and the peak pressures are over the first 3 metatarsal heads.[19]

SWING PHASES

MOTION

With the onset of early float, the ankle immediately begins to progressively reverse the foot's peak position of 30° PF attained in stance toward DF. The ankle advances to 5° DF by the end of late float[20] (see Figure 18-2A). This corresponds to a gradual increase in the TA activity and the relative quiescence of the posterior calf muscles. The exception to that being a small blip in the gastrocnemius as it assists with knee flexion[21] (see Figure 18-3).

The knee moves through an arc of flexion and extension during the swing phases. From a position of 13° flexion at the onset of early float, the knee attains a peak of 103° of flexion by the middle of swing and then reverses into an equally fast rate of extension until a final posture of 10° flexion is reached[20] (see Figure 18-2B).

The hip is still in 10° extension at the end of stance and maintains this position through early float. Then with the onset of mid swing, the hip rapidly reverses into flexion, attaining 30° flexion by the last third of this phase[20] (see Figure 18-2C).

Muscle Control

As swing replaces stance, the flexor muscles of the ankle and knee elevate the limb and ensure foot clearance. At the ankle, the TA rapidly lifts the ankle from its plantar flexed position and is constantly active above 25% MMT throughout swing (see Figure 18-4). Indeed, this muscle is contracting at greater than 20% MMT for more than 85% of the entire running cycle.[21]

Knee flexion in early float is accelerated by increased activity of BFSH, which reduces the resistance of the limb moving forward (see Figure 18-7). The VI and RF increase their action in synchrony with the biceps in order to modulate the rate of knee flexion (see Figure 18-5). These 3 muscles demonstrate a peak of activity at the point that the contralateral leg touches the ground (end of early float) and hip flexion is initiated.[13] At this point, the range of knee flexion is still increasing and becomes, in part, a passive function secondary to the hip flexion.[20] These muscles correspondingly diminish their activity until maximum hip flexion is achieved (ie, middle swing).

The iliacus actively flexes the thigh in concert with the RF (see Figure 18-9) and then begins to turn off once the contralateral foot touches the ground.[13] At the time peak hip flexion is achieved during the latter part of middle swing, all of the hip flexors have ceased activity.

The BFSH muscle again increases its action toward the end of middle swing to compensate for the loss of passive knee flexion afforded by the hip and to modulate the vastii during late float. As the knee extends in preparation for IC, the BFSH once again diminishes its action, while the vastii progressively increase their action.[13] It should be noted that, while not at the same intensity as the TA, the BFSH is active at above 20% MMT or greater for half the running cycle.

The semimembranosus and long head of the biceps femoris are relatively quiet (below 10% MMT) in early float, as is the lower gluteus maximus (see Figure 18-6).[13] Minimal extensor activity is required to maintain the extended hip posture during early float. Following floor contact by the other limb, the small increase in

activity of the tensor fascia lata, BFLH, and adductor magnus suggest the need for frontal plane control. At the end of middle swing, the 2 hamstrings increase their activity as they modulate hip flexion in preparation for stance. Once hip flexion ROM decreases, so does the action of these 2 muscles. The lower gluteus maximus, adductor magnus, and tensor fascia lata increase their action in late float as they stabilize the hip in preparation for IC.

The tensor fascia lata demonstrates a moderate peak of EMG activity in each of the running phases. It is active above 20% MMT for 75% of the running cycle (see Figure 18-8).[13] With this degree of activity, it is easy to see how the tensor fascia lata could become tight if not stretched adequately.

Thus, within the swing phases, a very controlled forward momentum is observed. Forward propulsion in running is provided by hip flexion in early float and middle swing and by knee extension during late float. As the lower extremity advances, there are coordinated eccentric and concentric contractions of the flexors and extensors to accelerate and decelerate the limb.

EFFECT OF PACE

The effect of a running pace has been suggested to have a role in the way the forces are distributed on the foot. Hoshikawa et al[7] found that as the speed of running increased, the stance time decreased. This has been further broken down by Pink et al[20] to reveal that faster-pace runners (8.7 mph) spend a third more time in float and correspondingly less time in single leg stance.

The fact of less time being spent in stance at the faster paces has lead to a widely accepted extrapolation that average (ie, slower) runners are characteristically rear-foot strikers and the elite runners (pace of 12 mph [ie, faster]) generally make IC much farther forward on the shoe.[17,29] Contrary to this thought, we now know that runners tend to select one pattern of IC, which they maintain even when changing paces between a training pace (7.8 miles per hour) and a race pace (9.6 miles per hour).[19]

There are some subtle differences in the distribution of pressure within the foot when running at different paces, however. At faster paces, female runners placed less pressure on the medial midfoot and more pressure on the lateral 4 toes.[19] This may indicate that the foot was in less pronation, or more supination, at the faster pace. Quite likely, the reason that male runners did not show this same pattern is that the group of males had more rigid feet and thus did not have as much pronation into which to move at the faster pace. In female runners, as the stance time decreased (a faster pace), the runners may not have had time to pronate as much.

There are no differences in ankle motion when comparing the 9.1-min/mile runner versus the 6.8-min/mile runner.[20] Two muscles that control the ST joint, the peroneus brevis and the TP, however, appear to be influenced by the running pace. The peak activity for both of these muscles is during stance, but only the peroneus brevis is influenced by pace. This muscle (Figure 18-11) nearly doubles its effort throughout the running cycle when increasing the pace from that of jogging (8.5 min/mile) to a faster pace (5.4 min/mile).[21] During middle swing, both the peroneus brevis and TP increase in intensity when pace quickens in order to provide balanced ST control (Figure 18-12).[21]

Fast-paced running entails considerably larger ROM at the hip and knee during the swing phases.[20] The hip initiates exaggerated motion at the beginning of early float with statistically more extension. The knee follows with increased flexion as a reaction to the momentum from the hip. At the end of middle swing and the beginning of late float, the hip exhibits significantly more flexion at the faster pace as the lower extremity reaches forward for the stride. Thus, the increased range in the hip and knee during the swing phases at the fast pace reflects a chain cycle that begins and ends at the hip with the knee responding to the hip motion.

With faster paces and corresponding increases in ROM at the hip and knee, there is a general trend toward enhanced activity with each of the hip and knee muscles. The pattern of the increased activity, however, is very similar.[13] As an example of this, please see the figures of the BFSH and the VI (see Figures 18-7 and 18-13,

respectively). As the pace increases, the contraction-relaxation of the agonistic-antagonistic muscle groups must occur quickly and precisely. If the muscles lack the precise timing to contract and relax, injuries could result.

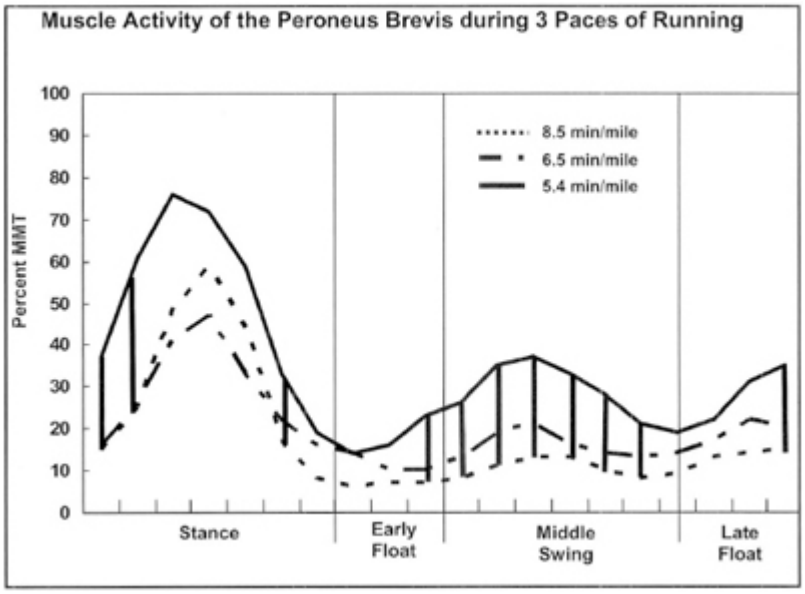

Figure 18-11. Muscle activity of the peroneus brevis during 3 paces of running. Vertical lines note periods of statistically significant differences. (Reprinted with permission from Reber L, Perry J, Pink M. Muscular control of the ankle in running. *American Journal of Sports Medicine.* 1993;21(6):805-810.)

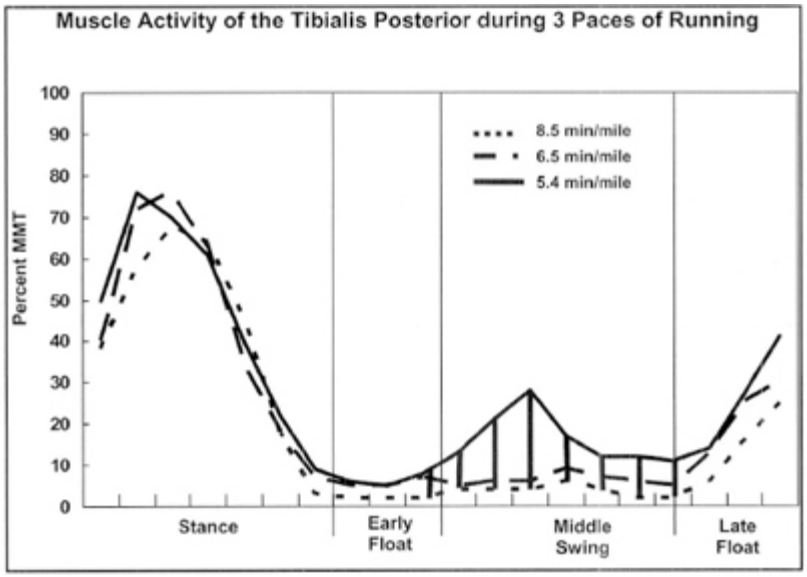

Figure 18-12. Muscle activity of the TP during 3 paces of running at 8.5 min/mile jog pace; 6.5 min/mile (train pace); 5.4 min/mile (race pace). Vertical lines note periods of statistically significant differences. (Reprinted with permission from Reber L, Perry J, Pink M. Muscular control of the ankle in running. *American Journal of Sports Medicine.* 1993;21(6):805-810.)

CLINICAL IMPLICATIONS

The major clinical problems faced by runners are the impact of muscle overuse, the pressures of floor contact, and the mechanics of eccentric muscle contraction. Each has a unique pattern of pathology and a particular functional etiology.

In an epidemiological study by Taunton and colleagues,[26] injuries below the knee in runners were shown to be directly or indirectly related to muscle dysfunction or fatigue. The TP is one of the muscles of concern since it is frequently involved with tendonitis. Also, over-pronation of the foot is a relatively frequent problem seen in runners. This may be due in part to the accentuated ST eversion created by the large impact forces during IC on the posterior lateral heel. Millions of dollars are spent on shoe design and orthoses to help with over-pronation. A clinician would want to consider the quadrupled demand in the TP during the WA period of running compared to walking when presented with a patient having over-pronation. One component of a training regime could focus on strengthening the TP.[9,10]

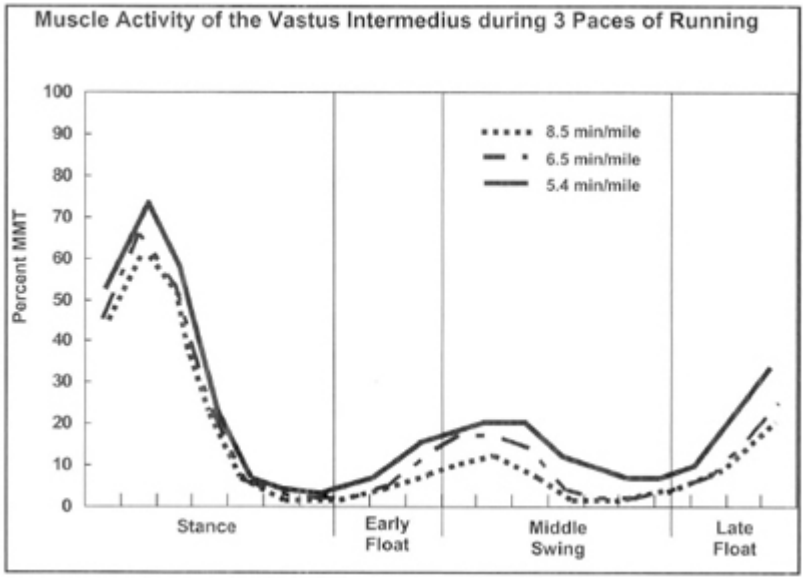

Figure 18-13. Muscle activity in the VI during 3 paces of running. (Reprinted with permission from Montgomery WH, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. *American Journal of Sports Medicine.* 1994;22(2):272-278.)

The body appears to be better designed to accommodate the pressures on the heel, which it performs satisfactorily during walking. During running, however, the heel does not appear to be at risk. The first 3 metatarsal heads and the great toe may be more at risk. A stress fracture of the medial metatarsal head is one injury found in runners. Clinicians would want to explore shoe design for their patients and ensure cushioning in this area.

A study by Monod[12] demonstrated that muscles with sustained contractions at a level greater than 20% of their maximal contraction are susceptible to fatigue overload. Fatigue failure and overload can cause cellular damage, leading to edema and increased compartment pressure. Thus, the TA is more likely to have fatigue-related problems during prolonged running than the posterior calf muscles. TA is active at greater than 20% MMT for more than 85% of the entire running cycle.[21] Previous authors have implicated muscular fatigue as an origin of tibial stress fractures.[11,26] These problems are frequently noted after starting a running program or increasing the mileage. If the power and endurance of the TA muscle can be raised through training, this may allow the muscle to sustain

the high level of activity that is required during running without exceeding its fatigue level. Other muscles particularly vulnerable to overuse are the BFSH, tensor fascia lata, and adductor magnus. These muscles would benefit from special attention in a strength and flexibility program.

Eccentric contractions are frequently implicated in muscle strains and delayed muscle soreness.[1,2,15,23,24,26,27] The muscles that eccentrically contract are the RF, vastii, short and long heads of the biceps femoris, and the semimembranosus. Recent investigations using ultrasonography suggest that the strain occurs in the tendon and aponeurosis during eccentric contractions while muscle contraction is isometric.[6] Further study focused on the muscles involved in running will elucidate this concept. It requires a unique focus in that the BFSH falls into the risk for injury categories of both overuse and eccentric contraction.

As the running pace quickens, the muscles not only increase their eccentric activity, but their tendons and aponeuroses (SEE) must withstand more rapid and severe lengthening (stretch). Therefore, it is important for the recreational runner to follow a graduated training schedule to allow muscles to accommodate to increases in eccentric loading. Strength training in the recreational runner should concentrate on strengthening hip and knee musculotendinous flexors and extensors. Also, the sustained activity of the BFSH, tensor fascia lata, adductor magnus, and TA at all paces, plus that of the peroneus brevis at the faster paces, requires special attention for flexibility and endurance training.

CONCLUSION

This chapter presents ROM, EMG, force, and pressure data on the lower extremity when running at different paces. It is hoped that the clinician is in a better position to understand, evaluate, treat, and prevent injuries in the recreational runner with this as a background.

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Section VI

Quantified Gait Analysis

Chapter 19

Gait Analysis Systems

The complexity of walking becomes very apparent as soon as one considers either referring a patient for objective analysis or establishing his or her own gait laboratory. Immediately, a decision must be made as to what techniques should be employed.

Basically, there are 5 measurement systems. Three of these focus on the specific events that constitute the act of walking. Motion analysis defines the magnitude and timing of individual joint movements. Dynamic EMG identifies the period and relative intensity of muscle function. Force plate recordings display the functional demands being experienced during the weight-bearing period. Each system serves as a diagnostic technique for one facet of gait. Often, data recorded simultaneously using these 3 techniques are synthesized to provide a more comprehensive picture of the deviations impacting gait as well as the underlying causes.

The 2 remaining gait analysis techniques summarize the effects of the person's gait mechanics. One measures the patient's stride characteristics to determine overall walking capability, while efficiency is revealed by energy cost measurements.

There are several choices of technique within each of these 5 basic measurement systems. These differ in cost, convenience, and completeness of the data provided. As there is not a single optimal system, selections are based on the needs, staffing, and finances of the particular clinical or research situation. Some decisions are optional, while others are determined by the type of pathology to be analyzed. Observational gait analysis can be used to guide decision making regarding the particular data (systems) required to elucidate the gait pathology, underlying causes, and functional consequences.

OBSERVATIONAL GAIT ANALYSIS

All professionals involved in the management of the lower extremities use some form of gait analysis. The simplest approach is a generalized screening to note gross abnormalities in a person's walking pattern. The observer is more likely to make the appropriate determinations, however, if the analysis proceeds in a systematic fashion. This circumvents the natural tendency to focus on the obvious events while overlooking other, more subtle deviations that may be highly significant. In response to this need, most organizers of gait analysis courses develop a syllabus as a guide to their students.

To be complete, systematic gait analysis involves 3 steps. First is information organization. The second is an established sequence of observation (data acquisition). Third is a format for data interpretation.

The essential gait events and their classification for observational analysis are demonstrated by the format of the chapters in this book. Normal function is sorted by anatomical area and the phasing of the events in the GC. The list of gait deviations, representing clinical experience with a wide variety of pathology, follows the same anatomical and phasic organization. The observational process is facilitated by having an analysis form that guides the clinician. In addition to identifying the gait deviations, having the form designate the phases in each gait deviation that can occur focuses the observer. A further asset is the differentiation of the more significant gait deviations from the minor occurrences (Figure 19-1).[1] The usual vertical orientation of the recording form from proximal (trunk) to distal (toe) is contrary to the order of analysis, but the result is an anatomically correct summary of the patient's difficulties.

The process of observational analysis (data acquisition) is best performed in 2 stages. First is a gross review to sense the flow of action. Then the analysis should follow an anatomical sequence in order to sort the multiple events happening at the different joints. Clinical experience with multiple approaches has led to the practice of starting at the foot and progressing upward. Floor contact,

ankle/foot, knee, hip, pelvis, and trunk are assessed in this sequence. Familiarity with normal function is developed first. At each level, the direction and magnitude of motion in each phase of gait is noted and imprinted on the observer's memory. An organized awareness of normal function is developed. Pathology is identified from this model as deviations from normal function. At each area, the patient's performance is compared to normal and deviations noted. Regardless of the gross appearance of the patient's gait, the observer should follow the format of sequentially analyzing each anatomical area relative to the gait phases and determining the deviations from normal before moving up to the next segment. Basically, the observer moves horizontally across the gait analysis form within each anatomical area.

The findings are interpreted at 2 levels. Total limb function is identified by summing the gait deviations that occur in each gait phase. In this way, the motions that impair progression or stability are differentiated from substitutive actions. The findings per phase then are related to the basic tasks, and the deterrents to effective weight acceptance and limb advancement are identified. The cause of these functional deterrents is deduced from the physical findings of weakness, contracture, spasticity, sensory loss, and pain. Instrumented gait analysis is recommended when a conclusion regarding the underlying causes cannot be reached.

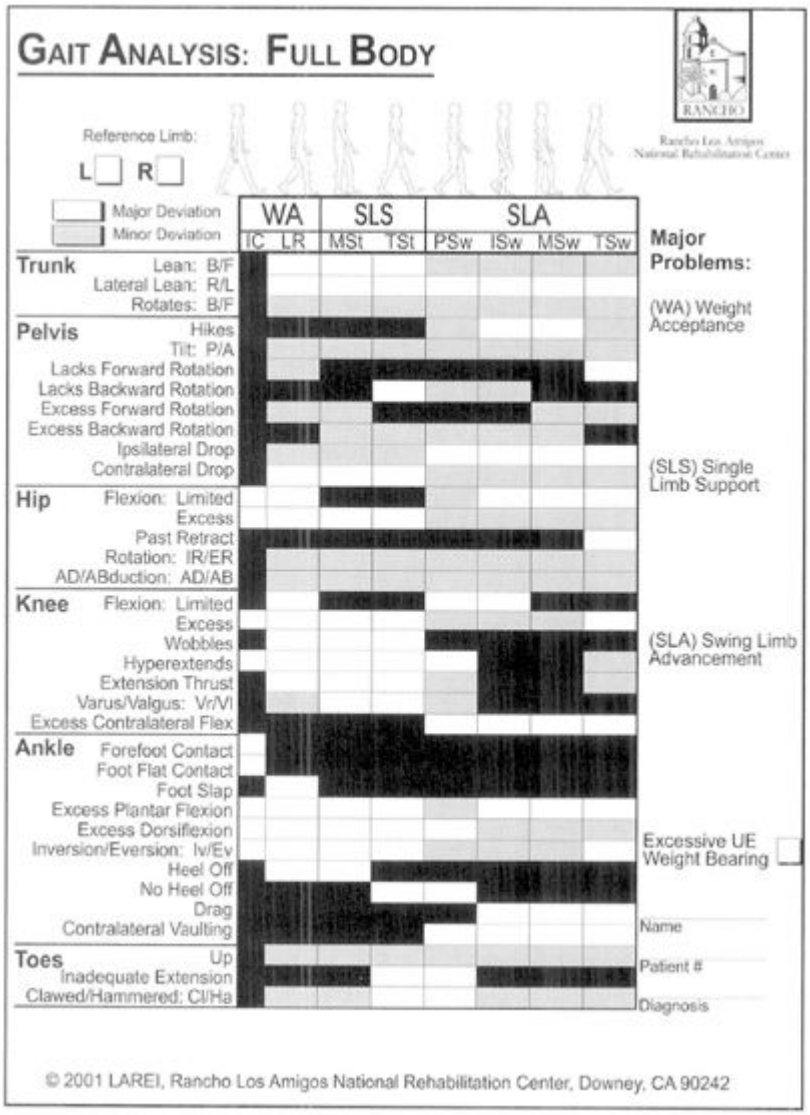

Figure 19-1. Full body observational gait analysis form (Rancho system). Rows = gait deviations; columns = gait phases. Walking dysfunction tabulated by checking the pertinent boxes. White boxes = major gait deviations; gray boxes = minor gait deviations; black boxes = not applicable.

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Chapter 20

Motion Analysis

Because walking is a pattern of motion, diagnosis of the patient's difficulties depends on an accurate description of the actions occurring at each joint. The customary approach has been to carefully observe the patient's gait and make appropriate conclusions. While the use of a systematic method of observation results in more agreement among observers, there still may be disagreement on details. The asynchronous series of changes occurring at each joint of the 2 limbs presents such a maze of data that few persons can assimilate them all. This limitation may result in premature conclusions. An alternate approach is quantified documentation of the person's performance with reliable instrumentation. The indecisions of subjective observation are avoided. Rapid and subtle events are captured. A printed record of the patient's motion pattern is provided. This recording can serve as a reference base for interpreting additional information such as EMG, stride, and force data. It also provides the data necessary for joint moment and force calculations.

Motion, however, is much easier to observe than measure. While the major arcs of joint motion occur in the sagittal plane, there also are subtle actions in the coronal and transverse planes. These deviations from the sagittal plane of progression often are much greater in the disabled walker and they may be of considerable clinical significance. This introduces 2 problems. First is the technical challenge of making the necessary measurements. The second problem is the effect of out-of-plane movement on the amount of sagittal motion that is perceived. For example, a single lateral view will underestimate the amount of flexion present if the limb is significantly rotated because the segments are foreshortened. This

fact is most easily appreciated by comparing the image of a flexed knee with the limb aligned in the sagittal plane to the image of the same limb internally rotated (Figure 20-1). When the limb is aligned parallel to the viewer (ie, in the sagittal plane), the knee appears to be flexed 60°. With internal rotation, knee flexion "appears" reduced. If this were a lateral camera recording of a child walking, the reviewer would say the child lacked the normal swing phase knee flexion.

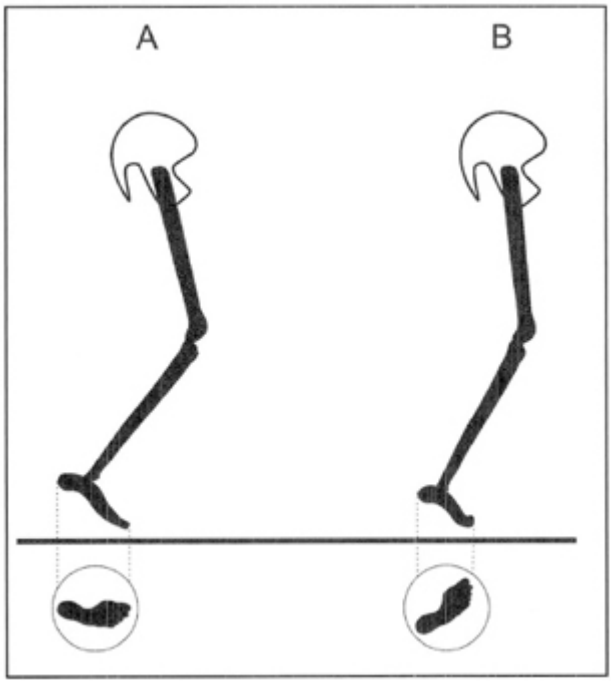

Figure 20-1. Single camera motion analysis cannot accurately identify joint angles when the plane of the limb is rotated relative to the camera. Note that the initial swing knee *appears* flexed (A) 60° when the leg is perpendicular to the camera, (B) but only 40° when the limb is internally rotated.

Two approaches have been developed to avoid this error: strapping a motion-measuring device directly to the limb (eg, an electrogoniometer) or using multiple cameras combined with sophisticated software to determine three-dimensional motion.[18] The technology available varies widely in complexity, capability, and cost.[8] While other techniques also are available (eg, electromagnetic technology,[10,13,24] hand digitizing,[31] or strobe light photography[26-28]),

these are currently infrequently used in the clinical research setting and hence will not be discussed in this chapter.

ELECTROGONIOMETERS

The first goniometer designed to measure knee motion during walking was an electronically instrumented hinge between 2 bars, one strapped to the thigh and the other to the leg.[11,17] While the principle was good, the alignment of the apparatus deteriorated with increasing knee flexion due to differences between the design of the goniometer's axis of rotation (a single pivot point) and the knee (a series of instant centers of rotation that follow a curved path). The curvature of the femoral surface of the knee joint sequentially changes from the broad anterior segment (rocker for a stable yet variable stance) to increasingly sharper curves (rollers for rapid flexion). As the curves become more acute, their radii become shorter and offset the center of rotation.

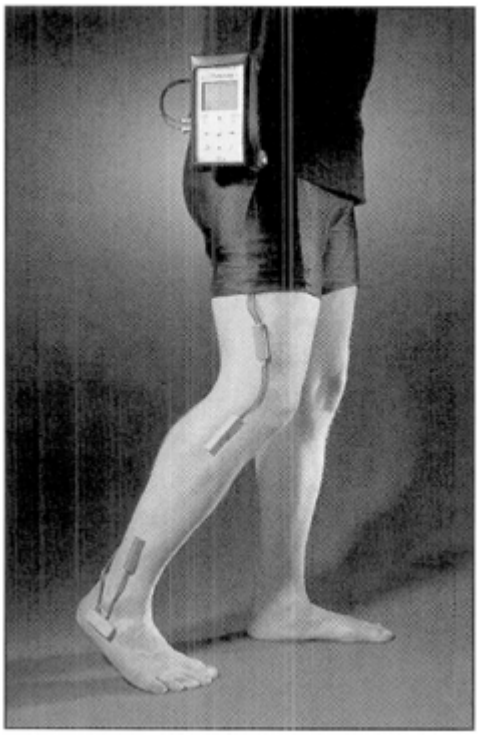

Figure 20-2. Strain gauge electrogoniometer applied to the lateral surface of the knee can be used to provide angular position

information across a variety of activities. (Adapted from photo provided courtesy of Biometrics Ltd.)

Electrogoniometers currently available vary in design, but generally provide an output voltage signal that is representative of the angular change between the 2 surfaces to which the device is attached (Figure 20-2).[8] Movement tracked on the surface of the limb is assumed to reflect actual angular joint changes. For lean patients, this assumption may be appropriate. However, using electrogoniometers to accurately track joint motion may not be possible in patients with significant soft tissue coverage. The advantage of electrogoniometers is that they are less expensive compared to sophisticated multi-camera systems and relatively easy to apply and use to study a variety of activities of daily living.[30]

CAMERA-BASED MOTION ANALYSIS SYSTEMS

When simultaneous study of multiple lower extremity joints or the whole body is desired, cameras provide a noncontact means of recording and reviewing motion. The number of cameras used contributes to the accuracy of the motion recorded and the ease of processing after data are acquired. To appropriately track the body and limbs through space, a minimum of 2 cameras are required. Otherwise sagittal motions will be underestimated when joint alignment is out-of-plane relative to the camera. For precise study of movement patterns during gait, a minimum recording rate of no less than 60 frames per second (hertz) is required to avoid missing peaks of rapid motion change. Ballistic activities (eg, throwing a ball) or other more demanding athletic activities often require higher recording rates (eg, 100 to 1000 Hz) to ensure adequate representation of the movement pattern. Two camera-based methods for describing gait motion patterns will be discussed: single video camera recording for qualitative evaluation and automated three-dimensional, multi-camera approaches for quantified analysis.

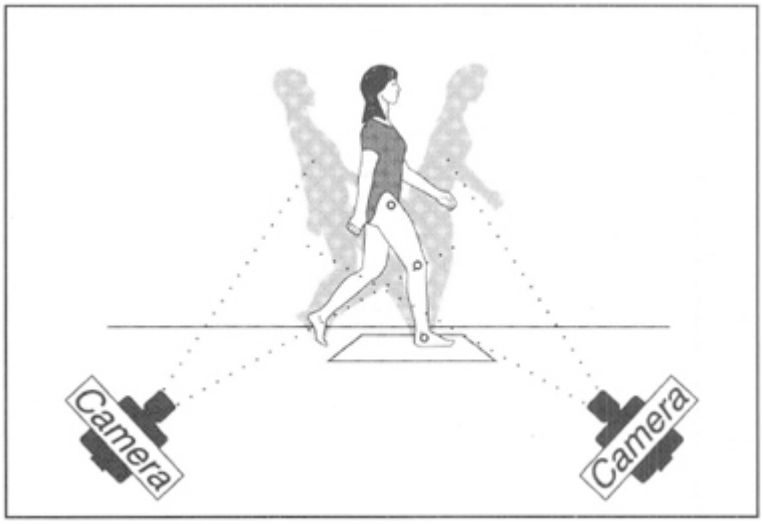

Figure 20-3. Two-camera motion analysis provides a three-dimensional (3D) representation of the limb. Rotation does not alter the joint angles recorded.

SINGLE VIDEO CAMERA RECORDING

The most convenient and least costly means of documenting a patient's gait is a single video camera, which provides lasting images of the patient's gait. If recordings are made from all 4 views (front, back, right, and left), clinically useful information can be obtained by systematic observational analysis even though the data are subjective. For satisfactory clinical review, the playback system should include stop action, slow display, and reverse. The addition of EMG and footswitches to the visible video also gives the investigator a low-budget gait analysis system. It is a way to start a new program until a more comprehensive system is affordable.

It is our practice to make a single camera video record of every patient's walk before applying any instrumentation even though automated motion analysis is to be used. The visual recording has clarified many situations. It provides a cross-check when questions arise. Also, patients occasionally change their mode of walking or have an unexpected response to a testing procedure.

AUTOMATED THREE-DIMENSIONAL SYSTEMS

Three-dimensional motion capture systems use sophisticated hardware and software to acquire and convert images of a person walking to quantified data describing the motions of each joint. Digital data are transmitted directly to the computer rather than providing the usual visual camera display. Cameras track the moving segments by recording the instantaneous two-dimensional coordinates of markers that have been placed over known locations on the limbs and trunk. The positioning of markers is a compromise between anatomical precision and potential marker stability. Data on markers that have been recorded by two or more cameras are integrated by the computer and redefined with their new three-dimensional location (Figure 20-3). Positional information can be stored in the computer or further analyzed to produce motion plots for the joints of interest.

Three-dimensional motion capture technology varies substantially in price depending on the number and type of cameras, the complexity of algorithms used to automatically track anatomic locations and movements across time, and the capabilities of the postprocessing software.[8] The optimal number of cameras to purchase is driven in large part by finances and study design needs. Five cameras are customary for unilateral recording. When simultaneous recording of bilateral data is desired, 6 to 8 cameras should provide sufficient ability to capture motion at typical walking speeds if the markers are easily visible. Ten to 12 cameras are useful when tracking movement across larger areas (eg, multiple strides within a single, over ground walking trial) or when markers are likely to be blocked (eg, with the use of bilateral assistive devices).

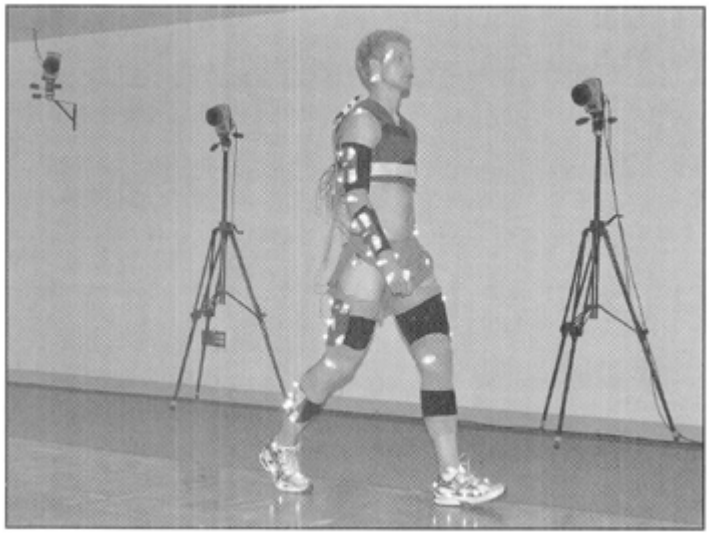

Figure 20-4. Infrared light-emitting cameras are used to track the motion of reflective markers that have been attached over predetermined anatomical locations. Changes in joint angles can be calculated from these data.

The ideal camera layout varies based on the study needs and laboratory space. Cameras can be rigidly mounted from the ceiling or walls to preserve floor space. Alternatively, tripods can be used to position cameras. This latter approach is useful when a variety of studies are being conducted that require different camera set-ups (eg, adult gait, pediatric gait, and a study of hand function). However, tripod-supported cameras are more vulnerable to being “bumped” during a study, which then necessitates recalibration.

Increasing resolution or frame rate capability adds to the camera cost. As indicated earlier, 60 Hz is generally sufficient for analyzing movement during normal walking. Higher speeds are required for faster, ballistic activities in order to adequately describe the motion.

Automated three-dimensional systems commonly use either passive (video-based) or active (optoelectric) technologies. Some systems now allow use of both types of technology. A brief overview of the 2 systems follows.

Passive (Video-Based) System

These systems use light-weight markers coated with a highly reflective material to track motion (Figure 20-4). Each camera lens is surrounded by infrared light-emitting diodes (LEDs) that emit pulses of light. The infrared light reflects off of the markers and back into the camera lens. Filters on the camera help to differentiate the bright spots created by the reflective markers from other areas of brightness in the field of view. The cameras track the moving segments by recording the instantaneous two-dimensional coordinates of the markers on the limb. Marker location is automatically determined by the system identifying the center of the "bright area." Edge detection algorithms can further refine the detection of marker centroids. Coordinate data, describing marker trajectories, are transmitted digitally to the computer rather than providing the usual visual display.[12,15]

Passive marker systems offer the advantage of not requiring wires or battery packs to be worn by the subject during the motion analysis study. However, passive systems are not without challenges. Reflective markers do not contain specific labels that provide the computer with information related to the anatomic location being tracked. Hence, correlating a specific "bright area" to its respective anatomic location requires operator input. Different software techniques have been developed to automate this process so that the operator does not have to repeatedly label each marker. One strategy involves recording a static trial in which the subject stands stationary while the cameras record the location of the markers. The operator then labels the markers, and their relative location is used during subsequent walking trials to automate the labeling process. Another challenge relates to camera resolution, particularly with dynamic activities. Adequate space is required between markers to avoid merging (overlapping) of the "bright areas" with each other. Additionally, other reflective materials (eg, emblems on shoes, shirts, or shorts) can be perceived mistakenly as a marker. Finally, methods for securing the markers need to ensure minimal jiggle as this motion artifact reduces the accuracy of marker locations.

Active (Optoelectrical) Systems

The second approach to automated motion analysis uses active markers to designate the anatomical sites. Each marker is a LED that is activated from a multiplexed power source (Figure 20-5).[3,19,20] Either the power is transmitted by cable from a central source or the subject wears a battery strapped to his or her waist. In addition, a fine cable extends from the power unit at the waist to the individual diodes.

The advantage of an active system is easier marker differentiation by multiplexing. Each marker is activated in a designated sequence so the computer automatically knows (by virtue of pulse timing) which site is being recorded. This sorts the markers for the computer and proximity between markers becomes less of a problem. As a result, a greater number of markers can be accommodated and the recording rate can be much faster. This advantage in simpler data processing has been off-set by 2 limitations. Reflection of light from the floor can prevent accurate recording of the foot. Recent design advancements are said to have corrected this difficulty. Electronic interference with simultaneously recorded EMG is the second limitation. This can be avoided by recording these data separately. Dependency on a power supply and transmitting cables also is restrictive. Otherwise, the general principles of marker placement and data management are similar.

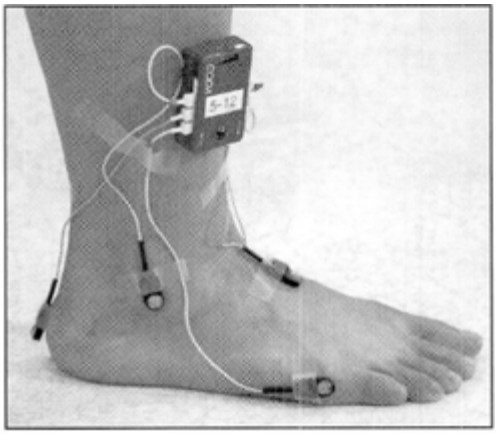

Figure 20-5. CODA active LED markers aligned over predetermined anatomical locations on the foot of a subject. The markers strobe sequentially, enabling the computer software to automatically determine the corresponding anatomic label for each marker. To

power the LEDs, wiring connecting the LEDs to a small battery pack is required.

MOTION MARKER LANDMARKS

All the motion quantification systems depend on defining the arcs and positions of the individual joints numerically. The basic technique consists of placing markers on the skin surface in locations that accurately represent the actions of the underlying joints. These markers are recorded by the cameras and their locations translated into motion data by sophisticated computer programs.

Two objectives for surface marker placement have evolved. Initially, the goal was merely to accurately represent the motion of the limb segments. Today, defining the centers of joint rotation for joint forces calculations is a second aim. This added objective has significantly modified surface marker placement.

Commonly, 3 markers are placed on each body segment so that the three-dimensional motion of the respective segments can be properly identified. There are 2 basic marker systems. The most common technique uses skin markers placed over pertinent anatomical landmarks. These markers designate the critical axes of the joints or serve as the third, out-of-line marker to identify the longitudinal axis of a limb segment. The total number of markers can be reduced by sharing “joint center” markers for adjacent body segments when appropriate. An alternate approach straps a fixture containing 3 to 5 reflectors over the mass center of each bony segment. The size of the apparatus limits use with small children. At present, there are no consistent guidelines for either system.[9]

Many motion capture systems offer software that includes a selection of predefined anatomically based marker sets (eg, the Helen Hayes or Plug-in-Gait marker systems). These frequently rely on the anatomical interpretation of individual investigators or laboratories who designed the marker set. Additionally, users can

develop their own marker system; however, the need to ensure validity of the model is essential.

Sagittal Plane Landmarks

At the pelvis, the clinical landmarks for the sagittal (progressional) axis of the pelvis are the PSIS and ASIS.[25] For gait analysis, the posterior landmark generally is identified by a stick or marker taped over the mid-point of the sacrum between the 2 PSIS. The anterior landmark is the calibrated center of the line between the right and left ASIS. During quiet standing with normal back alignment and a vertical thigh (full knee extension), the sagittal pelvic axis is anteriorly tilted downward approximately 10° from the horizontal (Figure 20-6).[25] This increases the measured angle between the pelvis and thigh (hip flexion) by 10°. Both hip flexion and lumbar lordosis that are greater than normal can proportionally increase the tilt of the pelvis axis. In subjects with pendulous abdomens, the accuracy of ASIS marker placement may be disrupted by the overlying adipose tissue.

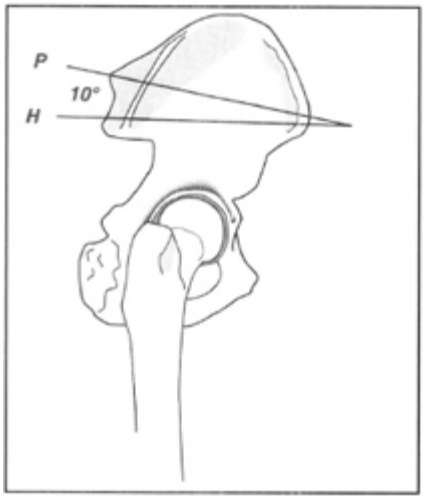

Figure 20-6. Pelvic landmarks are the PSIS and the ASIS. P = pelvic axis aligned with PSIS and ASIS. H = horizontal axis.

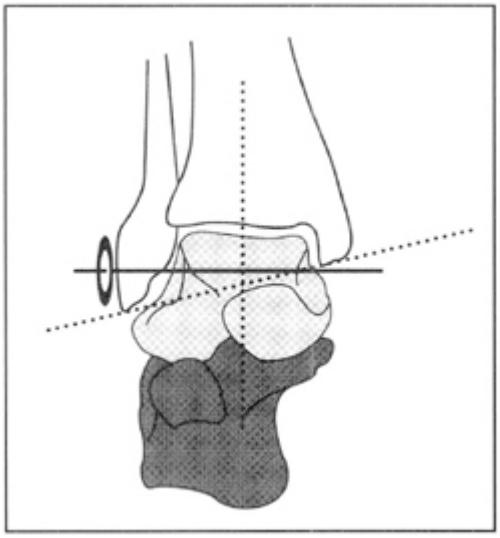

Figure 20-7. Ankle joint axis passes just distal to the tips of the medial and lateral malleoli.

The location of the hip joint center is one of the most difficult anatomic reference points to define. As a ball-and-socket joint, the center of the femoral head also is the center of the hip joint. The head of the femur, however, is located within the acetabulum on the obliquely aligned and tilted lateral side of the pelvis. The only prominent bony landmark on the proximal femur is the greater trochanter. In an unpublished study, marker placement was compared to lateral roentgenograms of the hip, knee, and ankle. Only the anterior, superior corner of the greater trochanter is opposite the center of the hip joint (femoral head); however, the greater trochanter is significantly lateral (see Figure 20-4) and this dimension has not been determined. Alternate approaches have used landmarks on the pelvis as the anatomical reference. The most comprehensive studies primarily made their measurements on x-rays of the pelvis, reserving cadaver specimens for clarification when necessary. The relative difference among the sexes and maturity (adults and children) were determined. A comparative study[5] of the data from 3 investigators (Bell, Andriacchi, Tylkowski) who calculated the relative distance between the pelvic landmarks and the hip joint center has been the most informative. Two reference lines were used: the distance between the 2 ASIS and the distance between one ASIS and the ipsilateral pubic tubercle. For each of the 4 subject

groups, the relative location of the hip joint (distance inferior, posterior, and medial to one or the other 2 reference lines) was determined as a percentage of the reference line length. These criteria have been incorporated in the computer programs designed to locate the hip joint of the individual patient. By combining techniques of 3 investigative teams (Andriacchi, Bell/Brand, and Tylkowski), a mean error of just 1 cm from the x-ray location was accomplished.

Knee joint anatomy is more directly determined. The transverse axis of the knee joint passes between the medial and lateral epicondyles of the distal femur. The long axis of the femur is identified with a third marker on the anterior surface. Because the lateral epicondyle often is not very prominent, the head of the fibula was investigated as an alternate guide. By roentgenographic study, it was determined that the lateral femoral epicondyle lay just above a point 1.5 cm anterior to the posterior margin of the fibular head.[29]

The ankle presents a minor discrepancy between the most stable location for a lateral surface marker (ie, the lateral malleolus) and the center of the joint axis. The axis of the ankle joint axis passes about 5 mm distal to the tips of the medial and lateral malleoli and approximately 8 mm anterior to the tip of the lateral malleolus (Figure 20-7).[14] As this is an area of significant skin motion, the markers are placed over the apex of each malleolus. Thus, the designated axis is slightly proximal but nearly parallel to the true axis. The long axis of the tibia is identified by a third marker on the shaft just below the tibial tubercle.

Coronal Plane Landmarks

Designation of abduction and adduction of the limb segments customarily relies on anterior landmarks. Pelvic alignment is designated by the 2 ASIS. The middle of the patella defines the anterior center of the knee. Similarly, the middle of the distal tibia is the marker for the ankle. These same points can be calculated from the combined medial lateral markers used in a three-dimensional system.

Motion within the foot has generally been ignored as only the ST joint customarily displays more than 5° of motion. Differences in mobility of the heel and the anterior foot, however, are clinically significant. Heel mobility can be designated by the displacement of a low posterior marker relative to the mid-point of ankle axis. The forefoot is designated by markers on the medial and lateral metatarsal heads and a third marker on the dorsum of the forefoot. Other motions within the adult foot are seldom more than a jog of displacement. This is present at the base of the first metatarsal ray (medial) or the fourth and fifth rays (lateral). Motion at the transverse joint of the midfoot may accompany ST valgus. The congenitally deformed infant foot, however, may display significant mid-foot malalignment as well as exaggeration of the other motions.

Transverse Plane Landmarks

Attempts to use just anatomical landmarks have not been successful because the arcs of rotation, even when pathological, are too small for capture by the available camera systems. A mid-segment marker is used to overcome this limitation. The marker is sometimes mounted on a wand to accentuate tracking of the arcs of rotation. The marker may be placed either anteriorly or on the lateral side of the limb (Figure 20-8).

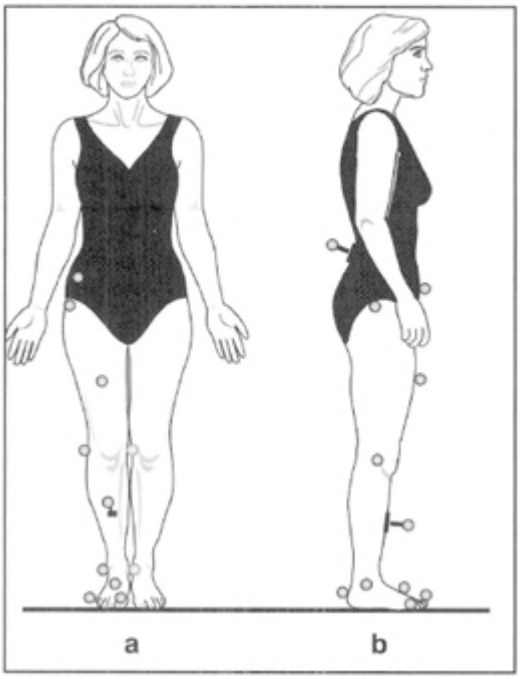

Figure 20-8. Three-dimensional surface marker system for motion analysis of hip, knee, and ankle function. Dark gray balls are the gait markers. Light gray balls are the pre-gait static markers to better designate the limb segment's plane and the axes of rotation. Sticks on the sacrum, mid anterior thigh, and mid anterior tibia facilitate the measurement of rotation by magnifying the arc of motion that occurs.

Factors Influencing Accuracy of Marker Data

Several additional factors reduce the accuracy of motion analysis with surface markers. Obesity hides the bony prominences at the pelvis, particularly the ASIS. Human error in correctly palpating landmarks also leads to inappropriate placement. There can be independent motion of the skin away from the joint centers.[6,21] Laboratories differ in their method of establishing the zero reference position.

Body Anthropometrics

Anatomic and anthropometric variations across individuals can alter the assumed relationships between the placement of surface markers and actual bony landmarks and underlying joint centers. In obese clients, adipose tissue may necessitate markers being offset from the optimal alignment. This can introduce substantial error when calculating joint center locations, particularly at the hip if the abdomen protrudes and prevents placement of the markers directly over the ASIS.

Independent Skin Motion

Bowstringing of the tendons and bulging of the contracting muscles pulling on the elastic skin can contribute to the marker moving relative to the underlying anatomic structure. A radiological study of the knee and mid thigh markers showed progressive displacement as flexion increased.[29] Posterior shift of the mid thigh marker increased from 0.9 cm with 15° knee flexion to 2.8 cm at the 90° position. The skin marker at the knee showed a similar pattern of displacement, being 0.6 cm posterior to the lateral epicondyle at 15° and 4 cm with 90° flexion. In contrast, the tight skin over the head of the fibula moved only 1.7 cm with 90° flexion. There was a corresponding reduction in the knee angle recorded by the skin markers. The loss was 8° with 90° flexion, a difference that is very similar to that reported for the anterior electrogoniometer.

In addition to bowstringing of the tendons, the skin to which markers are attached may move due to accelerations associated with limb movement. This skin wobble can also introduce noise into the motion signal, particularly markers placed on wands as the wands may accentuate the wobble. Reduced tissue stiffness in the proximal compared to distal thigh provides a less stable base for markers.[16] In obese clients, movement artifact arising from skin and soft tissue motion is a key source of error in gait motion data.[32]

To circumvent the difficulties associated with variable movement and location of the skin markers, the use of a mid-segment cluster of markers has been introduced. The purpose is to define the plane of each segment with 3 to 5 markers and then track its movement

through the basic reference planes (Figure 20-9).[4,22,23] Each cluster of markers can be affixed to light-weight, thin, semi-rigid, plastic fixtures that can then be secured to the desired limb segment. Tracking of marker clusters helps to reduce noise and improve accuracy of kinematic data.

The Zero (Neutral) Position

To date, a standard technique for identifying the neutral (zero) reference for each joint has not been defined. Using the subject's quiet-standing position is the easiest method to create a zero reference position but this can introduce considerable variability. Common subtle errors can be introduced. Often it is assumed that the ankle is at 90°, yet a balanced stance requires 5° of DF to position the body vector in the middle of the supporting foot. The knee may be either flexed or hyperextended rather than at neutral. For example, Murray et al's data on men showed the knee never fully extended during walking.[26] This related to a quiet-standing posture of 4° hyperextension that was called zero (MP Murray, written communication, 1964).

Recording the actual standing posture as it occurs helps avoid the current inconsistency. To accomplish this, the anatomical normal (neutral alignment) is the reference posture (Figure 20-10A). When the subject's limb cannot be positioned in normal alignment, the deviation is identified as a postural error and noted as an abnormal "zero" position (Figure 20-10B). The significance of motion marker location was demonstrated by a comparative study of gait motion for the same subjects obtained in 3 different research laboratories that were participating in a collaborative study that included staff travel to the other centers. Results of the analysis showed that the pattern of motion recorded for each subject was the same at the different laboratories but the numerical end points differed markedly.[7]

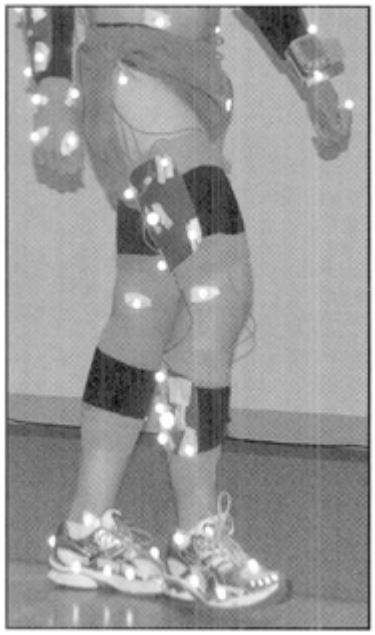

Figure 20-9. Cluster markers on the thigh and lower leg define motion of each segment and assist in reducing the influence of skin motion on kinematic data.

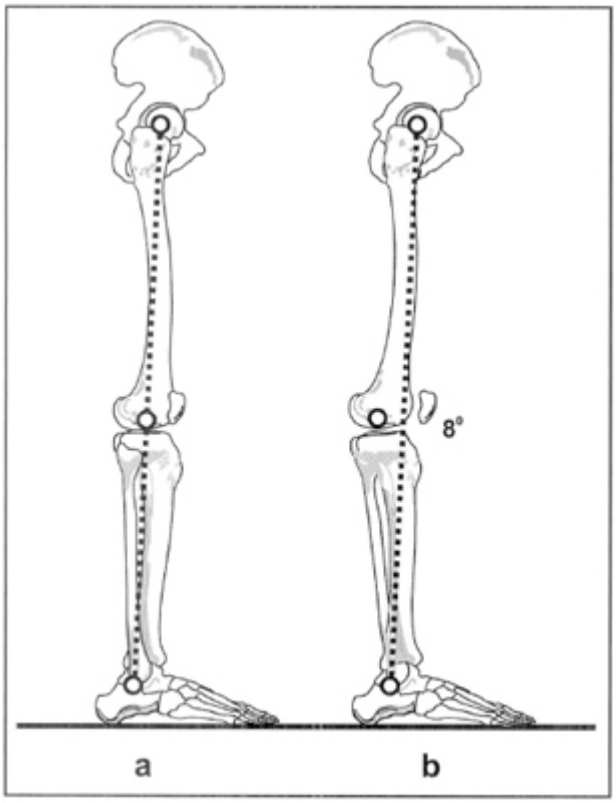

Figure 20-10. (A) The normal vertical alignment of the limb (“zero” position) is a straight line from the hip joint center, through the knee

epicondyle, to the lateral malleolus. (B) Joint postures that deviate from the neutral line should be identified as abnormal. In this limb, the “zero” posture includes 8° of ankle PF and 8° knee hyperextension.

THE RANCHO THREE-DIMENSIONAL LANDMARK SYSTEM

Multiple surface markers are used to permit simultaneous measurement of sagittal, coronal, and transverse motion of the hip, knee, and ankle. The system used at Rancho includes the following arrangement of markers (see Figure 20-8).

The pelvis is defined by the plane between the posterior sacral marker and the 2 ASIS (right and left). Care is taken to align the base of the sacral marker with the PSIS. The plane of the thigh is designated by 2 systems. For the gait recordings, markers are placed over the greater trochanter, anterior mid thigh, and lateral femoral epicondyle. A temporary medial epicondyle marker is added for a quiet standing record to relate the plane these markers define to the sagittal alignment of the limb. A similar system is used to identify the plane of the shank (lower leg or tibia). The gait markers are on the lateral femoral epicondyle, anterior mid tibia (a wand is used), and the tip of the lateral malleolus. Supplementing these is a temporary marker on the tip of the medial malleolus. The plane of the foot is designated by markers on the posterior heel (placed midline on the posterior calcaneus), the lateral aspect of the fifth metatarsal head, and dorsum of the foot. Medially, a temporary marker is placed on the border of the first metatarsal head. Medial markers, however, are threatened with displacement by swing of the other limb. This is resolved by making the medial markers “virtual” or temporary. The medial epicondyle, medial malleolus, and medial first metatarsal markers are initially applied and their positions recorded. This enables calculation of knee and ankle joint centers and the

midline of the foot. Then the 3 medial markers are removed and the remaining markers are used to capture motion patterns during the walking test.

Various modifications of the Rancho marker approach are used by other laboratories. One can identify the joint centers for force calculations as well as measure joint motion by defining the planes of the body segments (pelvis and limb).

REFERENCE SCALES FOR DESCRIBING GAIT MOTION

The numerical scales used to define arcs of motion at each joint are not consistently defined across laboratories or journals. Designation of neutral alignment is the basic area of indecision. Is it 0° or 180°? The clinical standard of using zero was established by the American Academy of Orthopaedic Surgeons many years ago and a reference manual that now is used by many countries.[1,2] Periodically, neutral is called 180° to facilitate reporting the full range of hip, knee, and ankle motion as a continuum of positive values. While this approach simplifies computer management of the data, it makes interpretation difficult because the reader must shift to an unfamiliar reference scale. For optimum effectiveness in information transfer, the data should be stated in the language of the clinician as the basic objective of using motion analysis is to clarify patient care.

Each lower extremity joint is capable of moving in both directions from its neutral (0°) position in each of its planes. Hence, graphically there are positive and negative values in the normal ROM. By custom hip flexion, abduction and internal rotation are represented as positive values (above zero) while extension, adduction, and external rotation are negative values (below zero) (Figure 20-11). The knee motions are similarly represented. At the ankle, there is more confusion as the functional neutral position is a natural right angle between the tibia and foot. Clinicians measure both DF and PF as degrees of deviation from a zero designation of this neutral posture (even though it is a 90° angle) (ie, DF 0° to 30° and PF 0° to

50°). Graphically, DF beyond this anatomically neutral position is a positive value while displacement into PF is negative.

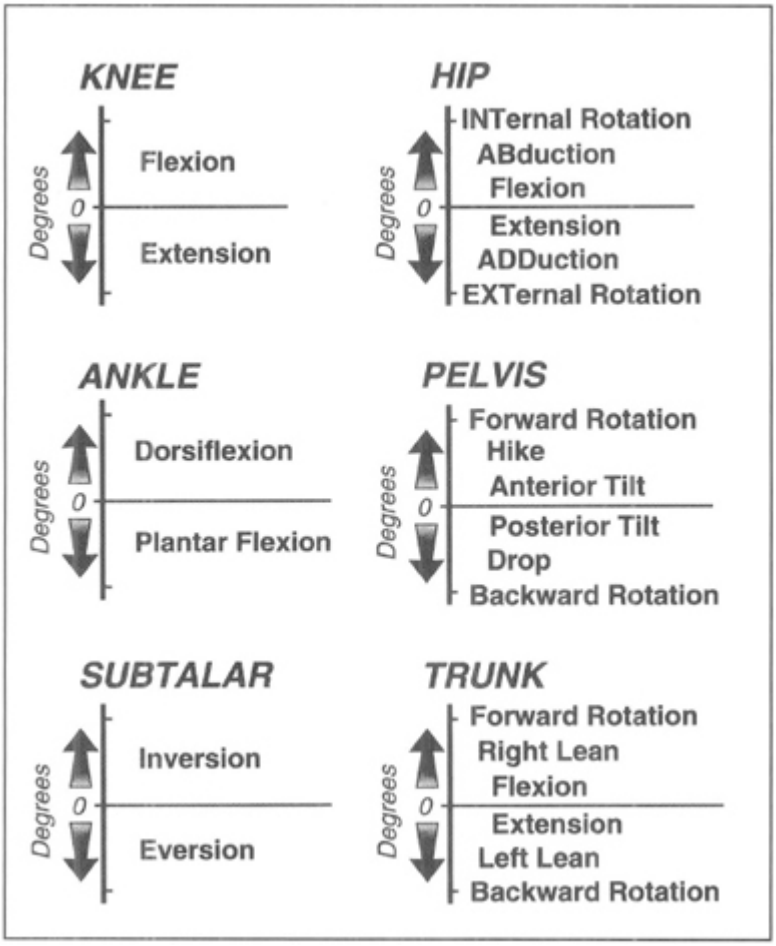

Figure 20-11. Reference scales. The direction for graphing each motion of the individual joints is identified. “0” replaces the neutral position for the motion. Vertical axis indicates magnitude of motion. Arrow denotes increasing values for each direction. Horizontal axis represents the gait cycle time scale.

The three-dimensional motions of the pelvis also have 2 directions of action. Anterior tilt, hiking, and forward rotation of the reference side are presented as positive values. Conversely, posterior tilt, drop, and backward rotation are treated as negative values (ie, below the zero line). For the trunk, the positive side of the graph represents forward rotation, right lean, and flexion. Extension, left lean, and backward rotation are designated in the negative area. It is important, however, to not confuse a clinical discussion with

statements of negative or positive values as they are generic terms. Thus, negative degrees of ankle motion from a graph are still referred to as degrees of PF, etc.

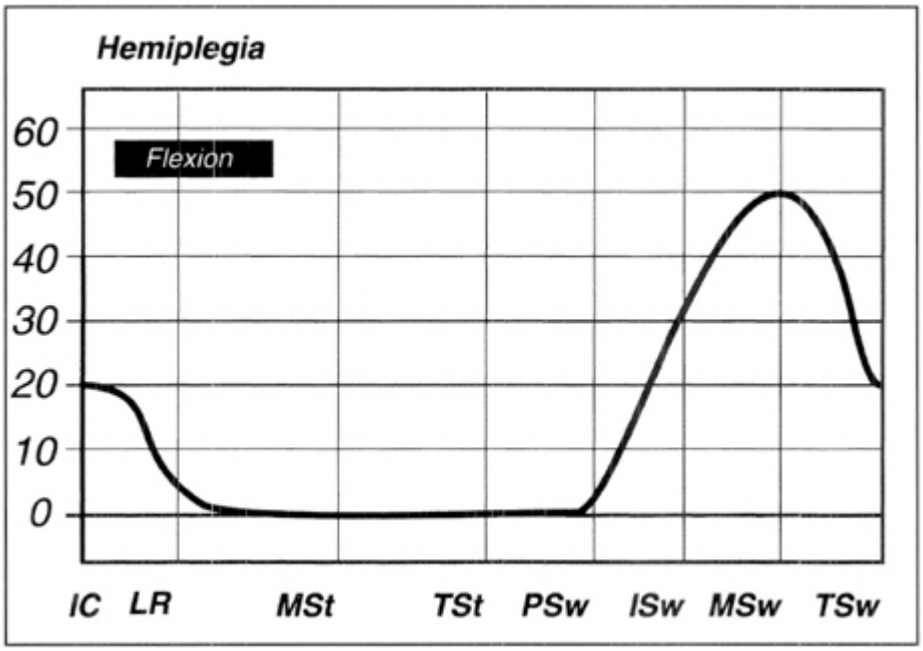

Figure 20-12. Knee motion graph for a client who had experienced a stroke.

MOTION DATA INTERPRETATION

The minimum information available from a motion record is the magnitude of the peak angles (high and low) experienced by the joint being studied. Because the functional demands of swing and stance are very different, the motion patterns of these 2 gait periods should be analyzed separately.

The magnitude of joint motion as an independent item of information, however, may not be sufficient to identify the patient's gait abnormality as the timing of the action may be the critical factor. Technically, identifying the time of the peak actions by the percentage points in the GC is easiest, but this reference does not indicate functional significance. Expanding the analysis to define the motion occurring in each gait phase provides a much better interpretation of joint function. Now each abnormality is identified.

For example, the record of a stroke patient's knee function may identify 20° stance flexion, extension to 0°, swing flexion of 50°, and extension to 20° (Figure 20-12). This implies normal stance action and seemingly minor deviations in swing (flexion 50° versus 60°) and failure to complete terminal extension. In reality, the patient has a significant gait error that is identified only by noting the patient's phasic pattern. Stance phase flexion (20°) occurred at IC (due to inability to completely extend the knee in swing) not as a loading response event. Instead, there was an extensor thrust as loading the limb caused a rapid loss of flexion. Also, this full extension persisted until toe-off. Hence, pre-swing knee flexion was lost. In swing, the presumably good arc of knee flexion (50°) occurred late (ie, in mid swing rather than initial swing). This delay resulted in a toe drag, although the patient eventually advanced the limb.

The third level of analysis involves coordinating the motion patterns of the adjacent joints. Often, this alone will reveal the cause of the abnormality. In the example just cited (see Figure 20-12), there was 15° equinus from terminal swing until toe-off with an associated IC by the forefoot. These findings indicated that the inappropriate knee motions were secondary to excessive ankle equinus, preventing the normal tibial advancement in stance but it did not continue in swing as the limb was advanced by a flexor pattern.

CONCLUSION

Motion analysis defines the person's gait. It does not identify the cause of errors but does delineate their magnitude, timing, and phasic relationships. Through advanced reasoning, which correlates the patient's performance with normal phasic function, the primary deficits can be differentiated from substitutive actions. Single camera evaluation of motion to supplement observational analysis provides one of the least expensive methods of gait analysis. However, quantification of the movement patterns is difficult and out-of-plane errors in motion detection can occur. Although electrogoniometers are more expensive, they provide a relatively easy method for rapidly

recording motion of a single joint during gait. Automated video and optoelectrical systems enable simultaneous tracking of data from the entire body during gait. The latter systems, however, are costly both in equipment (ranging from $150,000 to $300,000) and time invested in data processing.

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Chapter 21

Muscle Control and Dynamic Electromyography

Muscles provide the force needed to activate or restrain joint motion during walking, yet direct measurement of muscle force is denied by several factors. The overlying skin and subcutaneous tissues prevent direct visualization. Muscles do not act alone. Instead they are part of a synergy that controls the 3-D mobility of a joint as well as contributes to the primary function. The force that muscles deliver also varies with the joint position, speed of contraction, and mode of action. EMG provides an indirect method for analyzing muscle activation patterns. New advances in imaging techniques offer insights into the role of the whole muscle-tendon complex as a functional unit during gait.

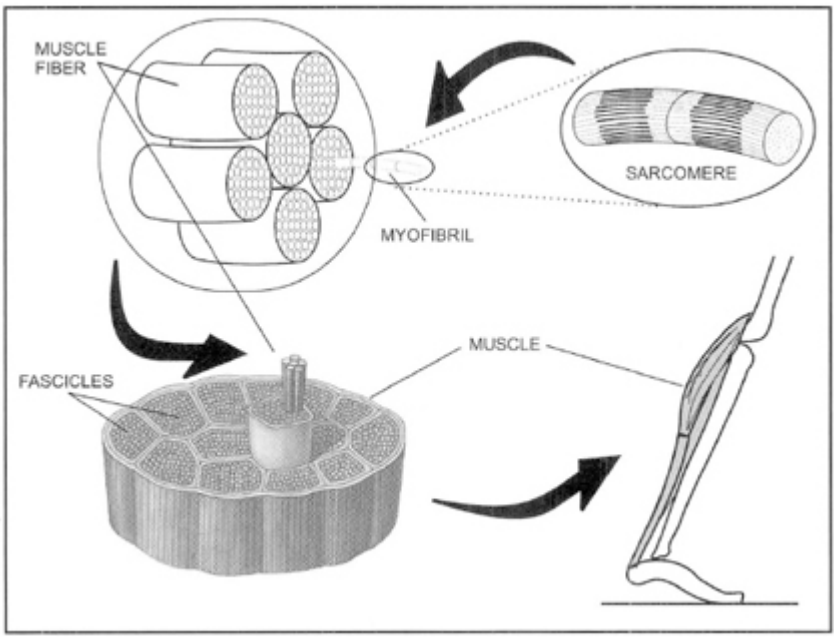

Figure 21-1. Skeletal muscle structure. Sarco-meres, strung together end-to-end, form myofibrils. Parallel strings of myofibrils, enclosed within the endomysium, form a muscle fiber. Clusters of muscle fibers are bound together by the perimysium to form fascicles, which group together to form a skeletal muscle. (Adapted from Lieber R. *Skeletal Muscle Structure, Fun-ction, & Plasticity: The Physiological Basis of Rehabilitation*. 2nd ed. New York, NY: Lippincott Williams & Wilkins; 2002.)

SKELETAL MUSCLE ANATOMY

The basic generator of muscle force is a microscopic structure called a sarcomere with an optimal length of approximately 2 to 3 μm (Figure 21-1).[46] A light microscope reveals the repetitious striated pattern displayed by strings of sarcomeres. A series of sarcomeres, aligned end-to-end, form a myofibril. When visualized with a standard microscope, it is possible to observe clusters of myofibrils enclosed by thin connective tissue sheaths (the endomysium). The grouping of these myofibrils into parallel threads provides the infrastructure for the muscle fiber. Muscle fibers vary widely in length from less than 2 cm in the soleus to over 45 cm in the sartorius.[78] Fascicles, which contain clusters of muscle fibers bound by a slightly denser connective tissue sheath (the perimysium), are visible without magnification.

The following sections contain a brief overview of key structures associated with skeletal muscle anatomy, including the sarcomere, motor unit (MU), and tendon.

THE SARCOMERE

Each sarcomere generates force by the interaction between 2 sets of dynamic protein filaments: thick myosin filaments and the surrounding thin actin filaments (Figure 21-2). These myofilaments

interdigitate to form a hexagonal lattice within the sarcomere. During a muscle contraction, the globular projections and heads of myosin filaments perform a rowing-like movement and repeatedly contact receptive areas on the actin filaments. If sufficient force is generated, the rowing action pulls the actin filaments closer to the center of the sarcomere and shortens the sarcomere. Peak force is attained when all of the cross-bridges on each side of the bare mid-section of the myosin filament are included. Motion reduces the number of contacts as the actin filaments slide along the myosin filaments.

Shortening of the sarcomere causes actin filaments projecting from the 2 ends of the sarcomere to overlap and blocks myosin contact. Conversely, sarcomere lengthening distracts the 2 filaments to the point where some myosin filaments are no longer within range to make contact with the actin filaments. Thus, both forms of displacement from the position of optimal myofilament overlap progressively reduce the isometric force as the contact area is decreased.

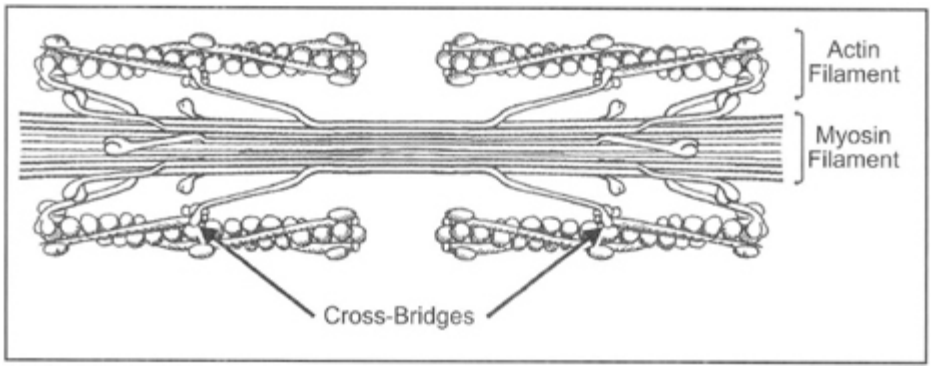

Figure 21-2. Sarcomere: Actin and myosin filaments interdigitate in a hexagonal lattice. Formation of cross-bridges between the actin and myosin contributes to force production during muscle activation. (Adapted from Lieber R. *Skeletal Muscle Structure, Function, & Plasticity: The Physiological Basis of Rehabilitation*. 2nd ed. New York, NY: Lippincott Williams & Wilkins; 2002.)

Excessive distraction is limited by a third giant protein called *titin*, which lies within the myofibril along the longitudinal side of the sarcomere. Titin stiffness apparently is the source of the passive

tension that develops in the strings of sarcomeres as they are lengthened beyond their optimal position.

Joint motion contributes to changes in a muscle's length and theoretically its capacity to generate active and passive force throughout the GC. When the sarcomere within the muscle is shorter than optimal, active tension capacity will diminish due to the overlap of adjacent actin filaments. Additionally, noncontractile elements (eg, titin) will not contribute to passive tension as the structures are not being stretched. At the optimal sarcomere length, the ability to generate active tension will peak, but again, titin will not contribute substantially to passive tension development. In contrast, when lengthened beyond optimal, active tension will rapidly decline while passive tension increases.[31]

Motor Unit

Motor control of the myriad of sarcomere chains (muscle fibers) that provide the force required to move a joint is simplified by having a single motor neuron activate a group of muscle fibers. This cluster of muscle fibers, their controlling neuron, and the motor nerve cell in the anterior horn of the spinal cord is called a MU (Figure 21-3). Despite the wide distribution of a MU's muscle fibers throughout a fascicle, all fibers in a MU contract simultaneously due to the shared innervation.[21]

Just one MU, however, does not produce sufficient force to create useful motion, although it might produce a visible twitch. To increase force production within a muscle, the nervous system uses 2 strategies. The firing frequency of MUs already recruited can be increased to help maximize force production within a unit (ie, temporal recruitment). Recruitment of additional, larger MUs also enhances force production (ie, spatial recruitment).[28]

Tendon

Tendons, which provide the muscle's bony attachments, are composed of dense fibrous connective tissue (mainly collagen). Anatomically, the tendon is continuous, with the less dense fibrous tissue sheath enclosing the muscle fascicles (epimysium). Biomechanical studies of tendons confirm their stiffness. Under loaded conditions, tendons stretch (technically called strain) approximately 3% to 5% of their initial length.[84] Previously, the major function of the tendon's flexibility had been defined as a means of lessening the impact of abrupt tension from vigorous activity. More recent research using dynamic ultrasound imaging has revealed that the tendon's strain (stretch) capability may reduce the demand for sarcomeres to lengthen during muscle activation.[8,16,25,29,30,33,34,43,44,50] This concept will be discussed in greater detail later in the chapter in relation to eccentric contractions.

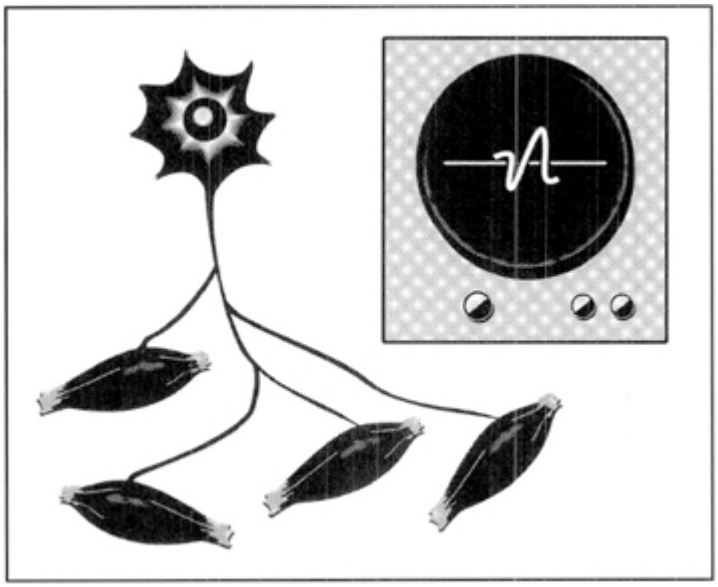

Figure 21-3. An MU. Left: The basic elements are the anterior horn cell, axon, and branches to its muscle fibers (4 displayed in this diagram). Right: A typical single MU EMG record display on an oscilloscope.

FUNCTIONAL POTENTIAL OF A MUSCLE

While an in-depth review of muscle morphology and architecture is beyond the scope of this chapter, 2 key areas will be highlighted due to their impact on walking function. These areas include the impact of a muscle's size (ie, physiological cross-section area [PCSA]) on force production and the influence of fiber length on muscle excursion.

Muscle Size

The dimensions of a muscle define its functional potential. The cross-sectional area measured perpendicular to the muscle fibers' line of pull represents the number of sarcomere chains (myofibrils) in parallel. This is called the PCSA, and it identifies the muscle's maximum tension capability. Sarcomeres, and thus muscle fibers, respond to stimulation with a maximal contraction (ie, "all-or-none"). This allows the cross-sectional area perpendicular to the muscle fibers' line of pull to define the muscle's maximal force potential.

Fiber Length

Maximum velocity of the muscle contraction is determined by the muscle fiber length (ie, the number of sarcomeres in a series). Longer muscle fibers have more sarcomeres in their chain. This reduces the amount that each sarcomere has to shorten to attain a designated velocity with a minimal loss of force.

Table 21-1

Motor Unit Content

Muscle	*Muscle Fiber Size (μ)*	*Motor Units Per Muscle*	*Average Muscle Fibers Per Motor Unit*	*Total Fiber Count*
Tibialis anterior	57	445	609	271,450
Gastrocnemius	54	579	1784	1,030,620

The length of muscle fibers in leg muscles varies almost 20-fold, from as short as 20 mm (soleus) to as long as 455 mm (sartorius).[78] Muscles with longer muscle fibers such as the semitendinosis, gracilis, and sartorius generally allow greater and more rapid joint excursions due to the summative contribution of each serially aligned sarcomere's change in length.[46,78] In contrast, when fibers are shorter (eg, soleus, TP, FHL) excursions are limited and the speed of movement is curtailed.[46,78]

Influence of Muscle Size and Fiber Length on Functional Potential

Detailed analysis of 2 typical leg muscles shows the significance of MU size, PCSA, and fiber length.[22] The medial head of the gastrocnemius (MG) had a 28 cm^2 cross-section. This area contained 579 MUs with 1784 fibers in each MU (Table 21-1). The smaller TA muscle with a 13.5 cm^2 cross-section contains 445 MU with 609 muscle fibers each. Thus, the number of sarcomere chains in parallel would be approximately 1 million for the medial gastrocnemius and 270,000 for the TA. The potential force of the gastrocnemius is approximately 6 times that of TA. The length of the TA muscle fibers (77 mm), however, is more than twice the 35 mm fiber length of the medial gastrocnemius and, thus, double the number of sarcomeres in its series. During equivalent arcs of joint motion, the TA could shorten twice as fast as the gastrocnemius. The functional demands on these 2 muscles during walking are consistent with their sarcomere populations. The medial gastrocnemius provides a major source of weight-bearing stability during terminal stance, while the primary role of the TA is rapid DF for clearing the foot during swing.

The size of these muscles enables individuals without pathology to walk at the customary speed of 84 m/min without fatigue. The level of exertion is grade 3 by manual muscle testing. This represents a 15% effort by the plantar flexors and the dorsiflexors.

MODES OF MUSCLE ACTION

The term *contraction* means to shorten. A muscle contracts in response to its activating signal (EMG). The magnitude of the opposing resistance determines the muscle's mode of action. Three potential patterns have been defined: concentric, isometric, and eccentric (Figure 21-4). All 3 modes of muscle contraction are used during each stride of walking.

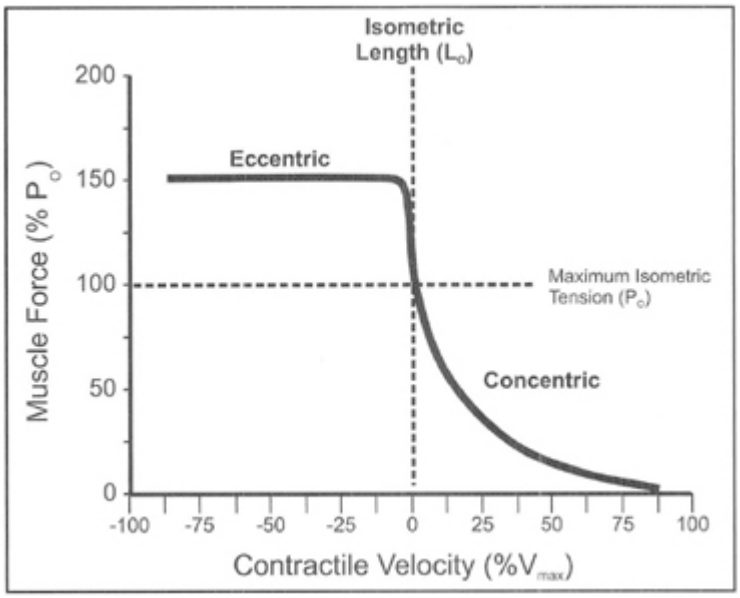

Figure 21-4. Relationship between contraction velocity and muscle force for 3 different modes of contraction. (A) Concentric: Faster muscle shortening (ie, more rapid contractile velocity) associated with lower force production. (B) Isometric: Contractile velocity = 0. (C) Eccentric: Force rapidly increases as muscle lengthens (negative contractile velocity). (Adapted from Lieber R. *Skeletal Muscle Structure, Function, & Plasticity: The Physiological Basis of Rehabilitation*. 2nd ed. New York, NY: Lippincott Williams & Wilkins; 2002.)

CONCENTRIC

When the muscle's force is greater than the resistance, the contracting muscle fibers shorten and motion is initiated. The contraction pulls the origin and insertion of the muscle toward each other and the joint moves. Increasing the rate of motion, however, reduces the muscle's ability to generate force (see Figure 21-4A).[24,46] Animal experimentation has demonstrated that the reduction of muscle force generation begins with the onset of movement. At 1% of maximum contraction speed, muscle force was reduced 5%. Motion at 17% of maximum speed dropped muscle force to 50%.[46]

The relationship between the rate of a concentric contraction and the generation of force follows a hyperbolic curve (see Figure 21-4A). The physiological basis of this relationship is pertinent to the gait of persons with muscle weakness. The weaker the muscle, the slower will be its rate of use.

Isometric

The rate of muscle contraction has a zero velocity (see Figure 21-4B). This occurs when the muscle force and the resistance are equal in intensity or the opposing structure is immovable. There is no motion. Consequently, an isometric muscle contraction is a special stage of concentric muscle action.

Eccentric

When the resistance is greater than the force being generated by the muscle, the muscle is lengthened. This action has been defined as an eccentric contraction because the ends move away from each other. A common synonym is a "lengthening contraction," which is a contradictory term. "Contraction" means shortening, yet the muscle is made longer.

Graphically, the tension in the muscle abruptly rises to a plateau with a significantly greater force than the muscle's maximum

isometric capacity (see Figure 21-4C). Once the plateau is achieved, the force is not influenced by the rate of velocity change.

The source of the additional tension had not been identified until recent experiments used portable ultrasound instrumentation to isolate the muscle from its tendon during weight-bearing activities.[33] The ultrasound head was strapped over the medial gastrocnemius muscle during dynamic joint motion. The investigators measured changes in fascicle length during terminal stance and found that the length of the active muscle fascicles had not changed. However, the calculated length of the whole tendon-muscle complex had increased. Thus, the length changes in the 2 tissues differed. The muscle contraction (accompanied by strong EMG) was isometric. In contrast, the tendon and aponeurosis stretched. Also tension increased. With the onset of pre-swing, the abrupt transfer of body weight to the other foot released the tightly stretched calf structures and the ankle rapidly plantar flexed. The abrupt shortening response was identified as elastic recoil because the rate of motion exceeded the limits of muscle contraction and there was no EMG. This identified the mechanics of the large push-off power burst, which has been a topic of debate for years. The source of this event is the accumulated tension in the series elastic component of the Achilles tendon as the ankle dorsiflexes an additional 5° during terminal stance in response to the body's forward fall over the supporting foot.

"Eccentric" muscle action also has been identified in the TA and the vastus lateralis during normal walking.[16] The TA's reaction to initial heel contact is vigorous contraction and high EMG to maintain a neutral ankle, yet a 6° arc of PF occurs during the first 6% of the GC.[29] A similar contradiction is evident in the quadriceps during loading response.[16] Vastus lateralis fascicle length did not change despite vigorous EMG activity in the same muscle and flexion of the knee following initial heel contact.[16]

Thus, ultrasound analysis has demonstrated that "eccentric muscle contraction" can actually arise from a combination of isometric muscle action and tendon stretch. Also, during pre-swing, the secondary reaction of abrupt shortening by both the tendon and

muscle that followed the intense terminal stance stretch had the characteristics of elastic recoil. The motion was faster than the muscle can contract and it was not accompanied by EMG. Thus, the tendon and muscle acted independently. Only the tendon was lengthened, while the muscle contraction remained isometric.

Hence, concentric muscle activity is the predominant mode of muscle contraction. An isometric muscle contraction is the zero rate of motion, and thus, represents a specific stage of concentric muscle function. Ultrasound studies have shown that many contractions previously thought to be eccentric are actually isometric, with lengthening occurring from the tendon stretching.[8,16,25,29,30,33,34,43,44,50]

ELECTROMYOGRAPHY

EMG is an indirect means of identifying the timing and relative intensity of muscular function by recording the signals of activation. In response to the neural stimulation of the myoneural end plate, intramuscular electrical signals are generated to activate the force, producing sarcomeres (Figure 21-5). With appropriate instrumentation, these myoelectrical signals can be recorded as they spread through the muscle and adjacent soft tissues.

Muscle Activation

Prior to a visible muscle contraction, an action potential is propagated down the motor neuron to the neuromuscular junction (ie, the border between the nerve and the muscle). At the neuromuscular junction, acetylcholine (a neurotransmitter) is released from the motor neuron, travels across the junction, and binds to special receptors on the postsynaptic muscle fiber membrane. This causes the muscle fiber membrane (or sarcolemma) to rapidly depolarize. Branching internally from invaginations in the sarcolemma is the transverse tubular system, which swiftly carries the action potential throughout the muscle fiber

(2 to 5 m/s).[46,49] Rapid spread of the action potential is accompanied by the transverse tubular system signaling the sarcoplasmic reticulum to release packets of calcium. The calcium is necessary to free the actin filaments to repeatedly form cross-bridges with myosin, leading to the dynamic generation of muscle force.[46] All of the sarcomeres in the muscle fiber virtually contract at once,[49] which is commonly called an "all-or-none" reaction.[54] It is the electrical signal produced by the muscle fiber during muscle activation that is recorded by EMG. When the neural impulses stop, calcium reuptake and restorage in the sarcoplasmic reticulum allow the actin and myosin to stop interacting and the muscle to relax.[46]

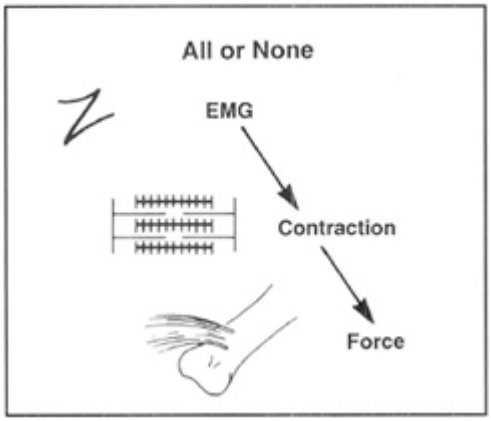

Figure 21-5. "All-or none" sequence of muscle activation. Electromyographic signal, sarcomere contraction, force output.

SIGNAL MANAGEMENT

The EMG signal is evidence that the muscle fibers are generating a force to either initiate or inhibit motion (see Figure 21-5). The EMG of one MU is a complex waveform because the wide dispersion of its fibers throughout the muscle results in slightly different activation times. In adults without pathology, the activation times vary by approximately 20 μs.[21] A single MU signal, however, is not seen during normal function as the resulting force would be too small to have any effect (less than 0.2% of a moderate muscle's capability).

Even the clinical level of trace muscle strength is at least 1%. Voluntary isolation of a single MU is possible as a learned effort.

The typical kinesiological (dynamic) EMG is an asynchronous series of electronic waves (action potentials) of varying amplitude and durations, which represents the activation of multiple MUs (Figure 21-6). The resulting electromyogram, called an interference pattern, is a composite of the 2 mechanisms used to increase muscle force. More MUs may be activated or the rate of MU stimulation is increased.[28] Both timing and relative intensity of muscle effort are important factors in quantified gait data.[63,72,79,80]

Timing

The period of muscle action is defined by the times of onset and cessation. These data can be determined directly from the raw EMG. Either visual inspection of the raw printed EMG record or computer analysis may suffice.

Defining the onset and cessation points of the muscle's activity remains a subjective decision. Most recordings contain some single spikes or short bursts of extremely small signals, which are functionally insignificant. The experienced observer filters these out by sight.

For computer analysis, minimal criteria for both intensity and duration were established. Minimal signal intensity was set at 5% of the maximum manual muscle test EMG.[6] This value approximates the clinically ineffective grade 2 (poor) level of muscle action.[3] The standard minimum duration of muscle action was set to at least 5% of the GC.[6,65]

Variability in subjective determination of the EMG endpoints was investigated by comparing an experienced investigator to a rule-based computer analysis. The computer showed 100% repeatability, while the ability of the investigator to select the same EMG onset time after a 1-week interval was 51% (intrasubject repeatability).[20] Consistency in selecting the same EMG onset by 3 experienced raters was 23% (intersubject variability).

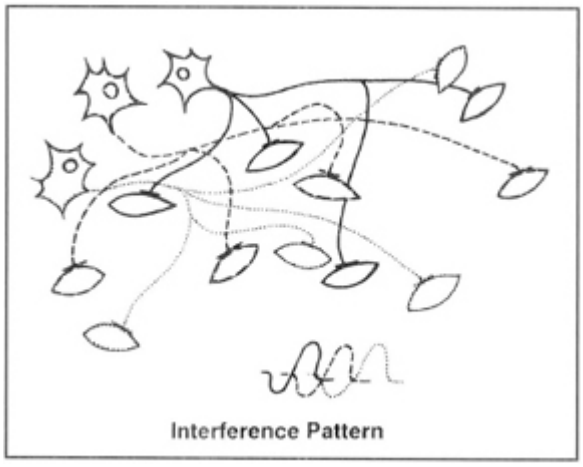

Figure 21-6. Representation of the normal “interference” EMG pattern. Three MUs, each with 4 muscle fibers, are illustrated. Below is the EMG created by their overlapping signals.

Time Adjusted Quantification

Individual strides show minor differences in timing of muscle action even though their intensity profiles are similar (Figure 21-7). These timing differences must be considered when combining the individual EMG data into an accurate mean intensity profile (see Figure 21-7).[7] Otherwise, the mean data will have a duration longer than that of any single stride because they will start with the earliest onset and continue to the last cessation. This error is avoided by calculating the mean values for onset and cessation of the series of strides recorded. The onset and cessation times of the individual quantified records are adjusted to match the mean timing. Then the mean profile is calculated. The result is a time-adjusted mean profile (TAMP) (see Figure 21-7, bottom row).

Normalization

The magnitude of the raw EMG signal represents 2 variables: the location of the electrode relative to the muscle’s MUs and the intensity of muscular action. Even with great care, no 2 electrode applications produce the same quantified data.[15] Anatomical factors contributing to this situation include the small size of the muscle

fibers (50 μ), a varying mixture of slow and fast fiber types, wide dispersion of MUs, the fibrous tissue planes separating the muscle fiber bundles, and variations in the contour of individual muscles. Consequently, the intensity of 2 muscles cannot be compared unless the EMG difference due to the individual variations in the number and type of MUs sampled by the electrode are excluded by normalization.

The normalization technique involves treating the functional data from each electrode as a ratio (%) of some reference value generated with the same electrode. For persons with normal neural control, the most convenient reference for the normalization process is the EMG registered during a maximal manual muscle test. The result is expressed as a percent of the reference value (ie, % MMT) (Figure 21-8A) or % MVC (Figure 21-9) if the maximum effort is measured with a dynamometer. While the latter is more accurate, the technicalities of aligning the limb and the machine require considerably more time. Both of these functional references differentiate the intensity of effort among muscles as the peak value will be relative to maximum ability. Repeated “spontaneous” maxima vary about 10%.[57]

Because subjects differ considerably in the way they reach their maximum effort, the common practice is to use a 4- or 5-second isometric test. Within this record, the mean value for the highest 1 second of data is selected as the reference value. This time interval is sufficiently short to avoid fatigue yet long enough to average out fluctuations in subject performance. Some investigators have found better consistency using a submaximal effort (50% max) as a defined target rather than the spontaneous maximum ($r = 0.83$ versus 0.68 for 100% max).[75,81,82] This may relate to having a consistent target.

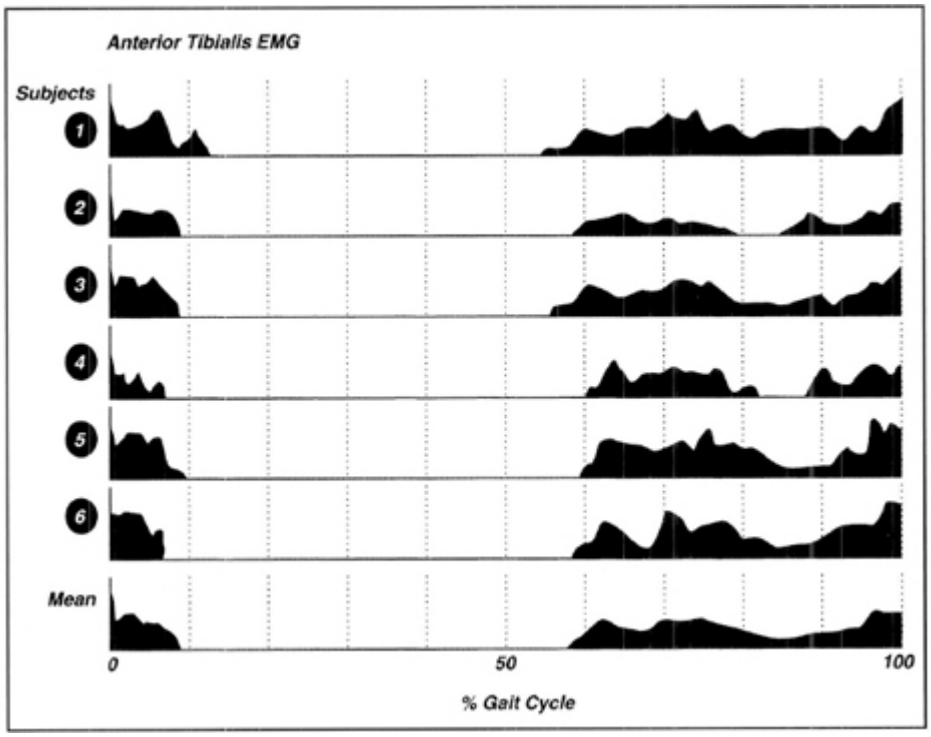

Figure 21-7. Time-adjusted mean EMG profile for the group (bottom). Individual records of mean EMG of 6 subjects showing differences in onset/cessation times and details of amplitude pattern. Group mean amplitude profile calculated after individual records were adjusted to mean timing.

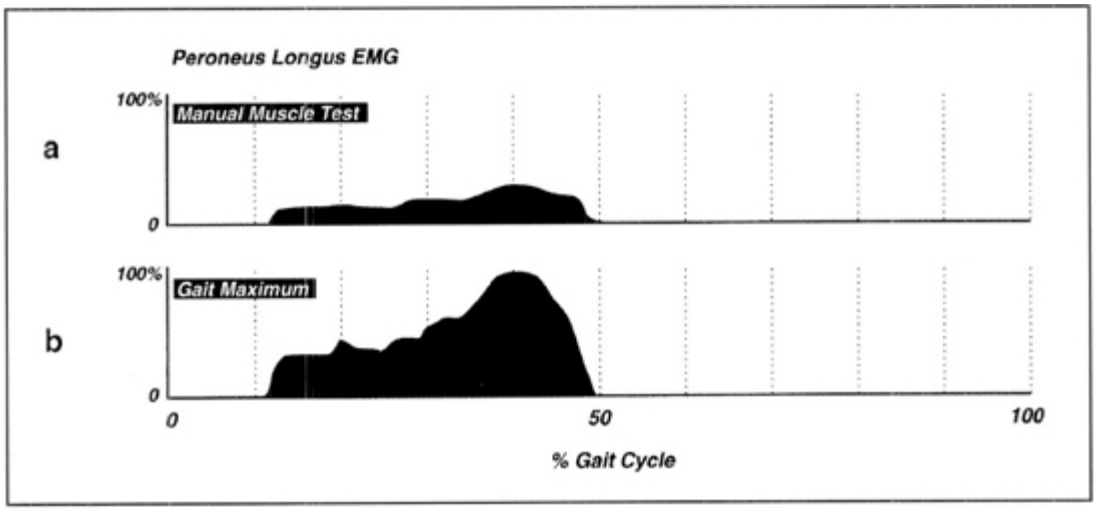

Figure 21-8. EMG normalization standard. (A) 100% = Maximum manual muscle test value (% MMT). (B) Maximum EMG in the stride.

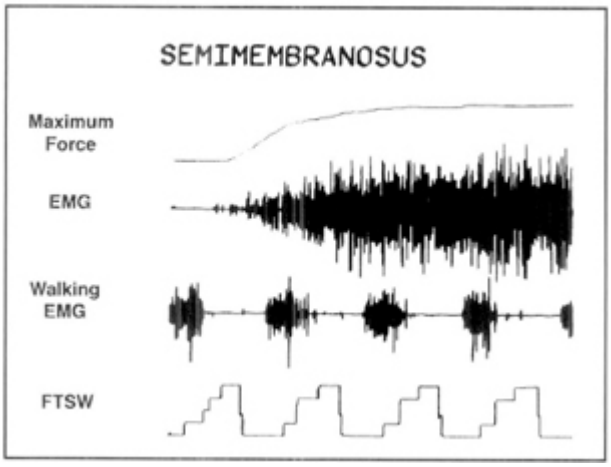

Figure 21-9. Normalization technique using maximum voluntary effort for the semimembranosus data recorded from a subject without pathology. Maximum force = isometric torque recording; EMG = muscle action during torque test; walking EMG = record of four strides; FTSW = footswitch records showing stance (staircase) and swing (baseline).

During rapid forceful effort, the brief EMG may be greater than 100% of the reference value. An in-house analysis of the waveforms in each situation showed the amplitude of the highest potentials were the same, but the number of such peak potentials was greater with the rapid, forceful effort than in the sustained maxima test. This apparently represents a trade-off between the need to preserve MUs for endurance and mass MU activation for instantaneous force.

If an inaccurate single effort is likely, the EMG sum for all the tests with that electrode may be used as the baseline reference value. Low-effort levels have greater signal variability, probably as a result of greater MU trade-off. This makes the normalizing base less consistent than using a strong or maximum effort.

A third technique that is often used in gait compares the EMG of interest to the peak value obtained with the same electrode during the gait stride (100%). This approach has the advantage of being applicable to all test situations and it is convenient.[56,81,82] The disadvantage is that the peak values of both weak and strong muscular activity are defined as 100% (see Figure 21-8B). This approach should be limited to patients with neurological lesions that impair voluntary control such as the more severe spastic disabilities

of cerebral palsy, stroke, or brain injury (ie, in persons who cannot reliably produce a maximum effort for the normalizing reference).[39,67]

Alternatively, for clients lacking sufficient volitional control to produce a significant EMG signal during strength assessment, a minimum threshold normalization value can be assigned to represent the muscle's maximum voluntary contraction.[58] This value is then used for subsequent normalization efforts during walking. In the Pathokinesiology Laboratory, we have assigned 25 digitized units as the normalization value when the maximum volitional muscle effort failed to produce 25 digitized units (61 mV) of signal during a 0.01-second interval.[58] This minimum normalization value reflects approximately 20% of a full interference pattern.[58] Use of this approach prevents the artificial inflation of muscle effort during gait that can occur when normalizing to very low numbers.

The conclusion is that normalization is an essential step before comparing activity among muscles. Calculating the functional EMG relative to that obtained with a maximum strength test provides the most informative data when the subject can cooperate with the baseline test.

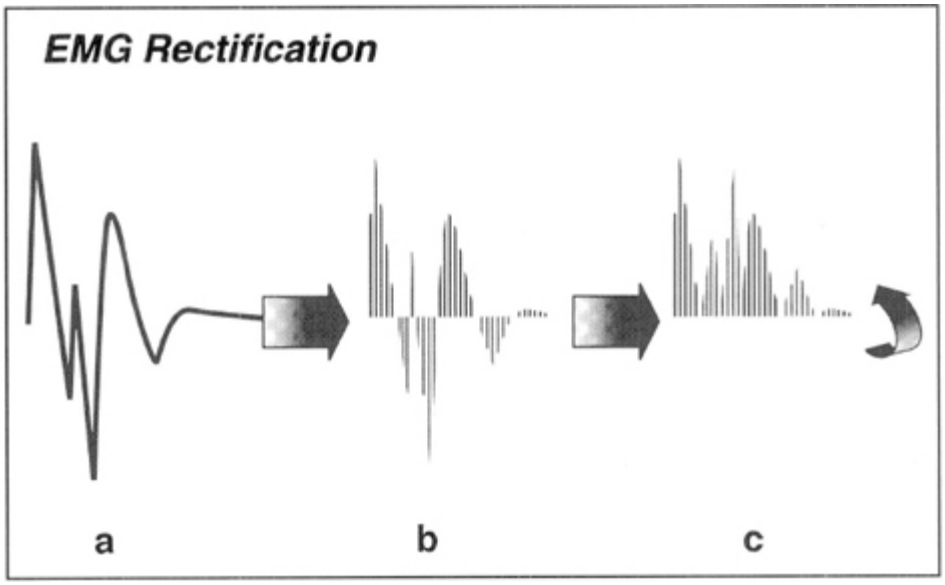

Figure 21-10. Rectification of EMG. (A) Raw EMG. (B) Negative waves delineated. (C) Negative waves assigned equivalent positive values and added to prior positive values (full-wave rectification).

Quantification

The first step is digitization, which transforms the analog EMG signals into numerical values. The Nyquist frequency rule indicates full data capture requires that the recording capability be 2 times the peak frequency of the signal of interest to avoid some loss. This necessitates sampling the data at 2500 Hz, a requirement readily accommodated by today's computers.

An adequate sampling rate is demonstrated by the ability to reproduce a signal pattern, which is virtually indistinguishable from the original raw analog record. One sign of an inadequate sampling rate is the inability to reproduce the clonus seen in the raw record of a spastic patient.

Following digitization, the signal is full-wave rectified by transposing all the negative signals to the positive side of the zero line (Figure 21-10). This avoids having positive and negative values cancel each other in the subsequent processing.

Integration consists of summing the digitized, rectified EMG signals over a time interval that is appropriate for the clinical function being tested. Interval duration is based on the expected rate of change in the activity being performed. For a static situation such as an isometric test, the interval can be as long as 0.25 sec. Gait analysis requires much shorter data intervals to be compatible with the rates of joint motion and muscle function. An interval of 0.01 second (approximately 1% GC for normal free gait) has been found to best correlate with the onset and cessation times displayed by the raw EMG. Reproducibility studies, however, show less variability when a 3% interval is used, but this is not compatible with the fast joint motion of normal gait.[37]

Presentation of the quantified EMG may be in absolute values (millivolts) or as a percent of a standard normalization. The absolute measurement (millivolts) is the most convenient, but it does not identify the clinically significant information of how hard the muscle is working (ie, relative effort).

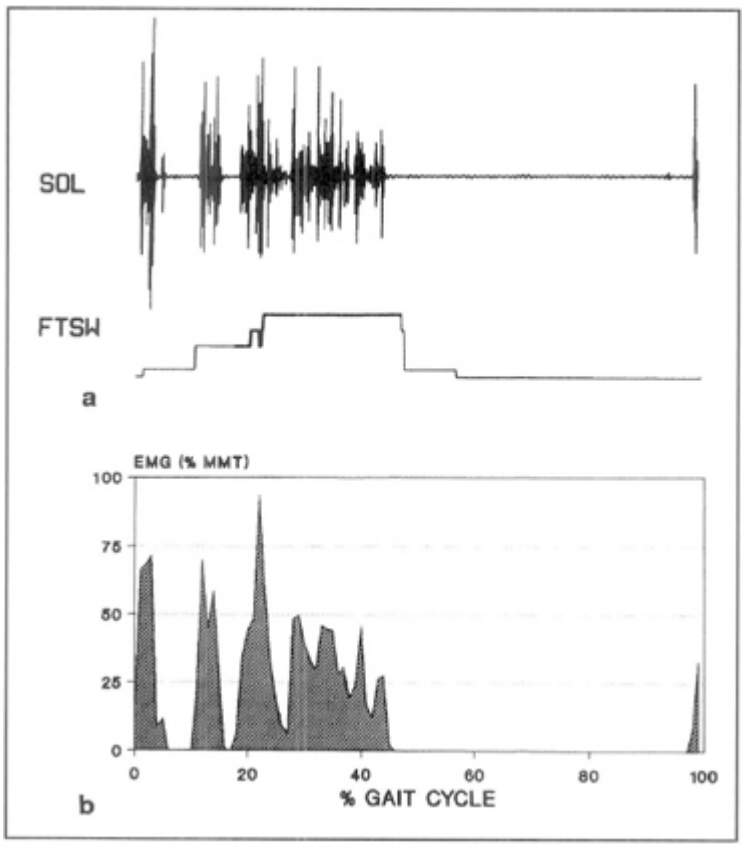

Figure 21-11. EMG representation of soleus muscle action during walking. (A) Raw EMG. (B) Quantitated EMG.

ELECTROMYOGRAPHY INTERPRETATION

Appropriate interpretation of the timing and/or intensity of effort can identify the functional effectiveness of muscular action. Several clinical questions can be answered. When is the muscle active? How hard is the muscle working? How does the effort of one muscle compare to that of others? What is the quality of the neural control? The dynamic EMG record, however, is not a direct measure of muscle force.

Timing of muscle action in gait may be defined by 3 different reference scales. Percentage of the GC is the simplest

determination, but the designated percentage points carry no functional significance. The minimum correlation with functional significance is the onset and cessation times of the EMG relative to the stance and swing periods. This has the advantage of technical simplicity by requiring only IC and toe-off signals. Using the 8 gait phases as the reference base for the EMG interval provides the most functional significance for the data on muscle activity.

Differences in the EMG amplitude of a single muscle represent varying levels of effort. Additional MUs are added as more muscle strength is required. Visually, the EMG record becomes denser and taller. When quantified, this translates to a numerically greater value (Figure 21-11).

Muscle Force

Muscles generate the force needed to provide the joint stability and motions used in walking and other physical activities. Dynamic EMG identifies the amount of muscular effort used during these activities but does not specify the actual force as several factors modify muscular effectiveness. The type and speed of contraction and fiber length determined by joint position directly define the force the muscle fibers can produce. In addition, the muscle force used for a particular torque varies with the lever arm available at each joint position. The intensity of the target muscle's involvement in an activity also is modified by the contributions of synergistic muscles.

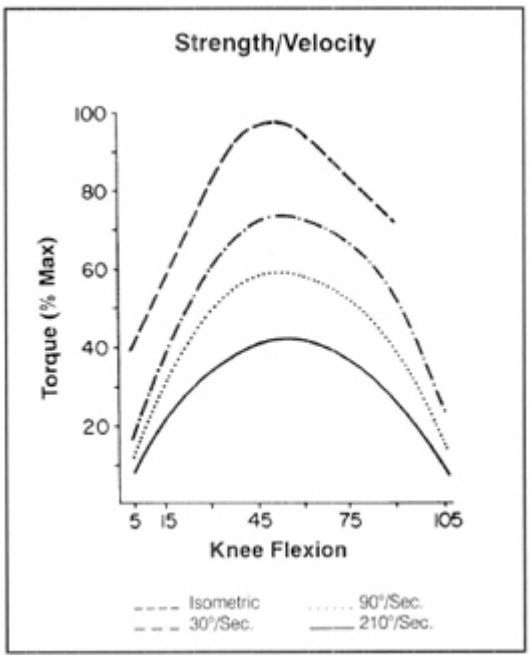

Figure 21-12. Maximum quadriceps strength (isometric torque) during isometric and isokinetic knee extension.

TYPE OF MUSCLE ACTION

Muscle force varies as the stability of the myosin-actin bonding within the sarcomere and fibrous connective tissue (FCT) tension changes. Isometric contractions, by allowing no motion, have a stable sarcomere length and fixed FCT tension. This form of muscle action has long been the basic means of testing strength. An eccentric or lengthening contraction presents similar sarcomere stability and potentially greater FCT tension. Total muscle strength becomes the sum of the sarcomere force and the fibrous tissue tension. The relative strength of maximum eccentric effort has been reported both as equal to isometric[70] and exceeding it by 10% to 20%.[42,74] Concentric muscle action shortens the muscle. The repeated changes in actin-myosin bonding result in approximately 20% less force than that generated by an equally strenuous isometric effort.[61,62,70] The strength loss varies nonlinearly with the speed of the effort.[62] Consequently, the same EMG value could represent a force proportion of 1:1 (isometric or eccentric), 1.2:1 (eccentric), and 0.8:1 (concentric).

The mode of increasing MU participation also may change during an activity. For example, the first dorsal interosseus muscle in the hand uses recruitment for the first 15% of force production and then employs increased frequency.[52-54]

Contraction Speed

The faster a muscle contracts, the less force can be attained for the same effort (Figure 21-12). Quadriceps activity, for example, shows a 38% decrease in maximum force between isometric (zero velocity) and isokinetic action at 150 deg/sec.[62] The EMG is relatively constant during maximum efforts at any speed even though the forces differ (Figure 21-13). Consequently, the force implied by the EMG differs with changes in speed.

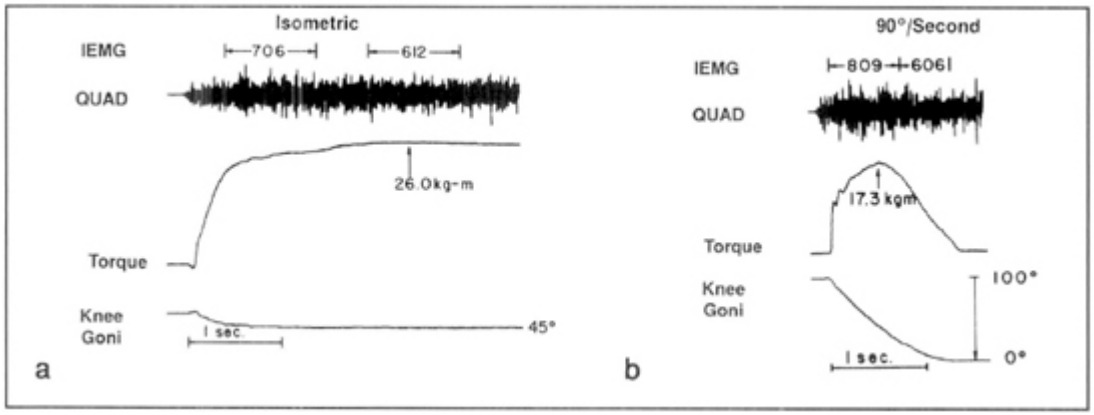

Figure 21-13. EMG and isometric torque during maximum knee extension. (A) Isometric with knee at 45° flexion (Knee Goni). (B) Isokinetic (90° to 0° arc at 90°/sec). IEMG = Integrated EMG and mean intensity over 1 second measured at 2 intervals; QUAD = raw EMG, test duration; Torque = Cybex measurement (kg•m).

Joint Position

Both muscle fiber length and bony leverage can change as joints move. Either factor can alter muscle torque. Shortening or lengthening the muscle from its optimum sarcomere setting leads to

a corresponding reduction in force. Strength of the quadriceps, for example, is markedly altered by changes in joint position (see Figure 21-12).[45,47,49] While the intensity of the EMG remains the same, strength declines 50% between 50° and 10° flexion.[26]

SUMMARY

In each situation, the ratio between EMG and force is altered. This results in variability in the muscle's force generation ability without a change in the number of active MUs (ie, the EMG of maximum muscular effort in each situation remains a virtual constant, F_{EMG}). The extent to which the optimum isometric force has been modified is reflected in the following equation:

$$F_{EMG} = F_{IM} - (K_1C + K_2L_M + K_3L_T + K_4V + K_5H)$$

where F_{EMG} = force inferred from EMG; F_{IM}= isometric force; and C, L_M, L_T, V, and H represent the contraction type, muscle fiber length, tendon length, velocity of contraction, and recent muscle contraction history, respectively. $K_{1\text{-}5}$ are proportional constants.

The significance of these variables is the finding of a linear relationship during isometric efforts of increasing intensity,[32,55] yet the slope of the data line differs with the joint position tested.[5,11,51] Dynamic studies show a differing ratio of EMG to force with variations in the speed of action.[23] When either velocity or force is kept constant, the change in the other variable is a linear relationship.[55,57] Others have reported a curvilinear relationship.[12,77,86]

The electromechanical delay is an additional variable in defining the relationship between the EMG and muscle force. That is, the electrical response precedes the mechanical reaction. The delay is assumed to arise from the action potential's propagation across the muscle, the excitation-contraction coupling process, and the stretching of the series elastic components within the muscle by the contractile component.[85] Initially, Inman et al identified an 80-ms delay.[32] More recent studies found the interval between the EMG

and resulting force to be no more than approximately 40 ms.[17,48,68,76,85] The fact that the EMG signal of a single MU lasts only 1 to 3 ms[49] reaffirms muscle mechanics as the source of the delay.

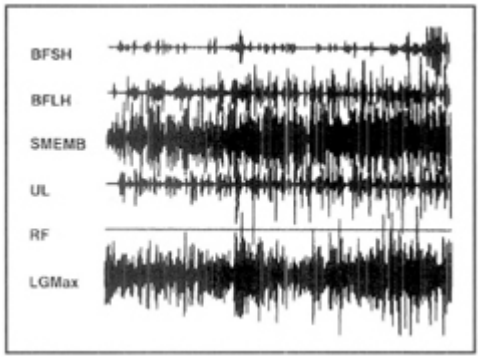

Figure 21-14. Synergistic action during a standard manual muscle test of strength. Lower gluteus maximus (LGMax) was the muscle being tested. Simultaneous action occurred in the other hip extensors, biceps femoris long head (BFLH), and semimembranosus (SMEMB). Quadriceps (VL) participation implies use of mass limb extension. Biceps femoris short head suggests a low synergy with the other biceps head. Lack of rectus femoris (RF) action indicates it is a flexor and not part of the extensor synergy.

Synergy is the final concern. Muscles generate force as part of a group (synergistically) rather than in isolation (Figure 21-14) to produce rotation about a joint (torques or moments). Subtle differences in alignments across the joint also give most muscles another function so their participation in a particular activity varies with the functional demand. Thus, muscle synergy makes it impossible to accurately assign the measured torque to a particular muscle as advocated by some investigators.[10] While designating a representative muscle simplifies the calculations, the total source of the muscle force has not been considered.

The versatility of today's computers makes it possible to include all the modifying factors in the calculations of muscle effectiveness.[60] When these mechanical values for each of the muscles within the synergy of an activity are correlated with their quantified EMG's (defining the relative intensity of effort), the torque pattern can be

reproduced (Figure 21-15).[59] Hence, EMG can imply force by indirect means.

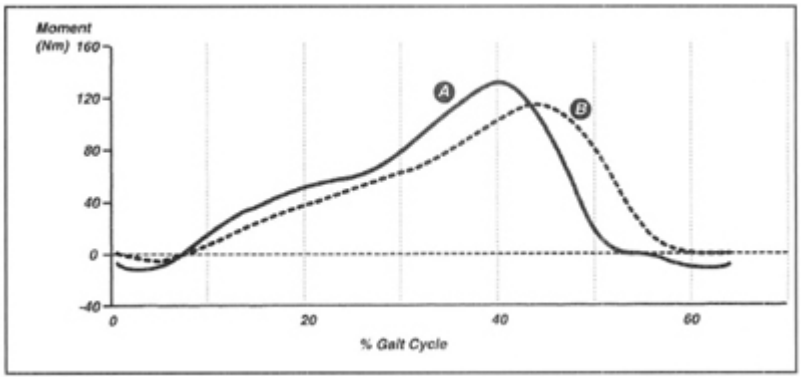

Figure 21-15. Time and intensity relationships of ankle muscle action and torque during walking. (A) Action of the ankle plantar flexors is defined by the product of their quantified EMG and their mechanical qualities (cross-sectional area, passive elasticity, etc). (B) Torque (moment arm multiplied by vertical ground reaction force).

ELECTROMYOGRAPHIC ANALYSIS OF PATHOLOGICAL GAIT

Neurological control errors, muscle weakness, voluntary substitutions, and obligatory posturing to accommodate pain or deformity lead to an abnormal EMG recording during walking. Both timing and intensity may be altered either for a particular phase or over the entire cycle.

Abnormal Timing

Relative timing of muscle action compared to normal function is highly significant in the interpretation of pathological gait.[4] Seven classifications of abnormal activity are used: premature, prolonged, continuous, delayed, curtailed, absent, and out-of-phase (Table 21-2

and Figure 21-16). The additional period of muscle action indicated by premature and prolonged EMG patterns has functional significance when it involves another phase of gait. This activity may represent dynamic obstruction of desired function or it may be appropriate support for an abnormal joint posture. Conversely, EMG patterns that are curtailed, delayed, or absent imply the lack of desired activity (see Figure 21-16). Sometimes abnormal limb posture makes the muscle action unnecessary. Continuous muscle activity throughout the stride is always undesirable. Out-of-phase EMG, however, may signify a useful substitution. Obstructive versus accommodating muscle action is differentiated by correlating the EMG with the accompanying motion patterns of the limb. Often, one must consider adjacent joints as well as the one being controlled by the muscles in question.

Table 21-2

Electromyography Timing Errors

Deviation	*Definition*
Premature	Action begins before the normal onset
Prolonged	Action continues beyond the normal cessation time
Continuous	EMG uninterrupted for 90% or more of the GC
Delayed	Onset later than normal
Curtailed	Early termination of EMG
Absent	EMG of insufficient amplitude or duration
Out-of-phase	Swing or stance time reversed

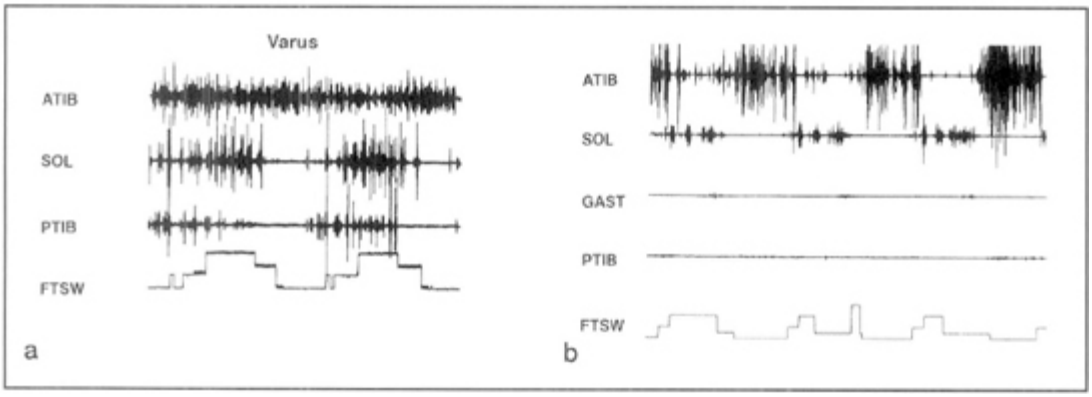

Figure 21-16. Abnormal EMG patterns occurring in clinical tests (timing). (A) Continuous anterior tibialis (ATIB); premature soleus (SOL), onset in terminal swing rather than loading response;

curtailed posterior tibialis (PTIB), terminal stance action missing; footswitch (FTSW) differentiates stance (steps) and swing (baseline) periods in each stride displayed. (B) Prolonged anterior tibialis (ATIB); premature soleus (SOL); no significant activity in gastrocnemius (GAST) and posterior tibialis (PTIB); FTSW = footswitch of reference limb.

Rancho defines the timing of normal function as one standard deviation from the mean. While this standard excludes 32% of the “normal” population, correlations with gait motion indicate the inappropriate EMG represents inefficient muscle action and should not be a standard for normal function.

Abnormal Intensity

Pathological function is identified as excessive, inadequate, or absent. The normal range is one standard deviation above or below the normal mean value (Table 21-3 and Figure 21-17). By visual analysis, excessive activity is difficult to identify. The analyst must carefully relate the magnitude of the observed EMG to the muscle test values. Inadequate signal is more apparent. Abnormal function also can modify the shape of the EMG pattern by clonus in spastic patients and MU enlargement from lower motor neuron disease.

Technically, the simplest measure is mean intensity throughout the muscle’s period of action. This, however, may overlook a major difference in function during a part of the muscle’s period of activity. Assessing the intensity of muscular effort according to each phase of action imparts far more clinical significance as functional demands continually change. The associated EMG identifies the appropriateness of the muscle action at that time. Rapid changes in amplitude or differing peak values within the phases also should be compared. Even a continuous EMG pattern should be analyzed for intensity variations to differentiate dominant from lesser action. These details further contribute to understanding the patient’s stability and mobility problems. The significance of the EMG intensity

depends on the motion that is occurring for reasons similar to those identified for deviations in muscle timing.

Motor Control Variations

Selective control displays an EMG response that is proportional to the demand in both timing and intensity. The relative intensity of the EMG increases as the demand torque becomes greater and then the EMG also diminishes as the demand lessens (Figure 21-18). This may be reflected in a rising ramp of myoelectric signals or steps of different magnitudes. Cessation of normal stance phase muscle action tends to be abrupt, while a descending ramp in swing is often seen. This sensitivity to functional need results in each muscle having its own timing and intensity pattern.

Primitive mass pattern control displays simultaneous onset and termination of the basic extensor muscle groups in stance and a similar synergy among the flexor muscles in swing (Figure 21-19). With reduced sensitivity to local demand, the EMG of each muscle tends to have a relatively uniform intensity during its period of action. The EMG amplitude among the muscles, however, generally differs.

Rapid elongation of muscles with a hyperactive stretch reflex (sometimes referred to as spasticity) stimulates an abrupt muscle pattern of unchanging intensity. Body weight, antagonistic muscle action, or phase reversal can be the stimulus. Consequently, stance phase muscles may display spasticity in swing and vice versa (see Figure 21-19). The EMG pattern varies with the rate of stretch. Slow stretch introduces a continuous pattern of EMG signals with uniform amplitude. Quick stretch elicits a sequence of evenly spaced bursts (clonus). As the spastic response dwindles, the amplitude of the clonic bursts decrease. Both types of stretch reactions tend to be called *spasticity*, although the slow continuous response is technically rigidity and the fast action is true spasticity. The EMG pattern can be very irregular when spasticity and voluntary muscle action signals are both stimulating muscle action.

Table 21-3

Relative Intensity Errors

Deviation	*Definition*
Excessive	EMG value greater than the normal band
Inadequate	EMG value less than the normal band
Absent	EMG insufficient to identify functional significance

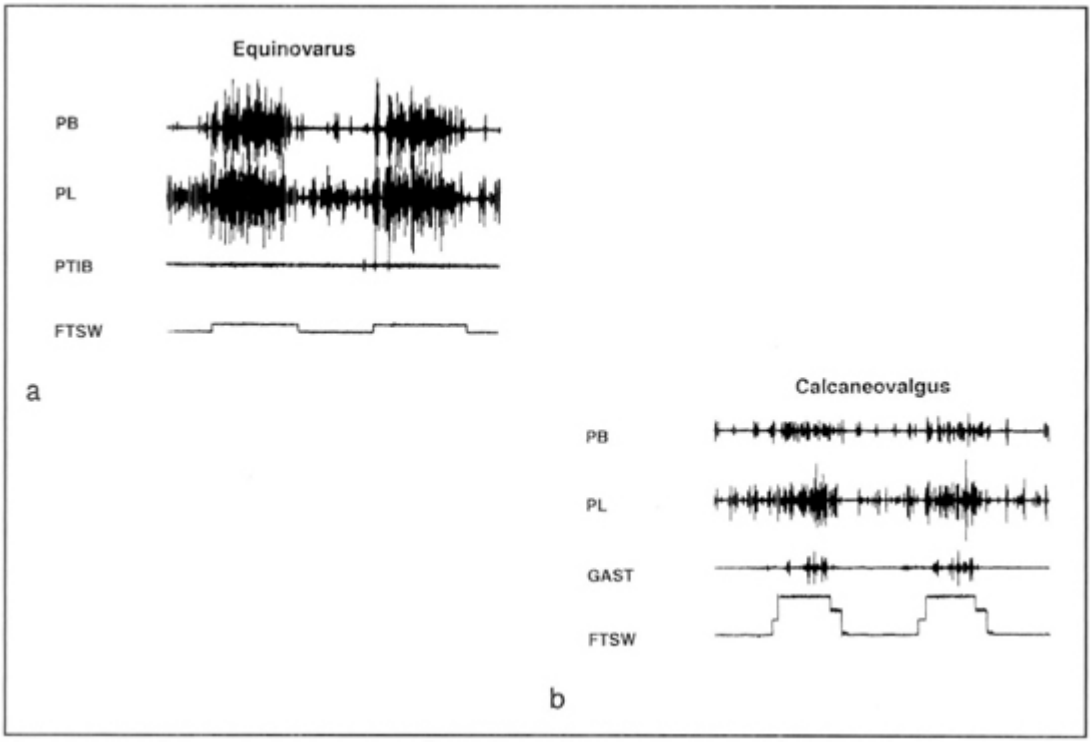

Figure 21-17. Abnormal EMG patterns (intensity). (A) Excessive peroneus brevis (PB) and peroneus longus (PL), amplitude, and density indicate greater MU activity than customarily needed; absent posterior tibialis (PTIB). The continuous peroneus longus (PL) record demonstrates low levels of action, appropriate, although excessive, stance phase EMG, and out-of-phase swing action at a lesser but functionally significant intensity. Footswitch (FTSW) identifies fifth metatarsal foot support. (B) Inadequate intensity and delayed onset (GAST); except for a single early burst in mid stance, EMG amplitude is insufficient for meaningful dynamic support of the ankle. Peroneus brevis (PB) and peroneus longus (PL) show phasic,

though premature, action in stance and clonic bursts in swing. Footswitch (FTSW) shows foot flat (H-5-1) is dominant.

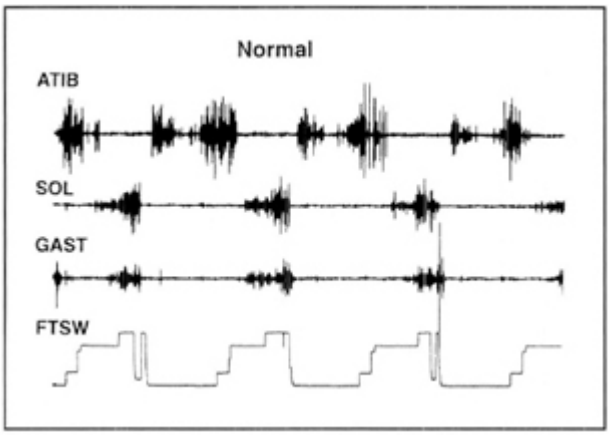

Figure 21-18. Selective control. Both the intensity and timing are proportional to the functional need. Anterior tibialis (ATIB) has 2 intervals of intense action: initial swing to pick up the foot and terminal swing/loading response to control the heel rocker. Soleus (SOL) and gastrocnemius (GAST) action controls the tibia. The low EMG in mid stance indicates the low demand of the ankle rocker. The marked amplitude increase in terminal stance correlates with the heel-off support (high demand of the forefoot rocker). Footswitch (FTSW) shows a normal sequence with a long mid stance (third step, H-5-1) interval followed by heel rise (top step, 5-1).

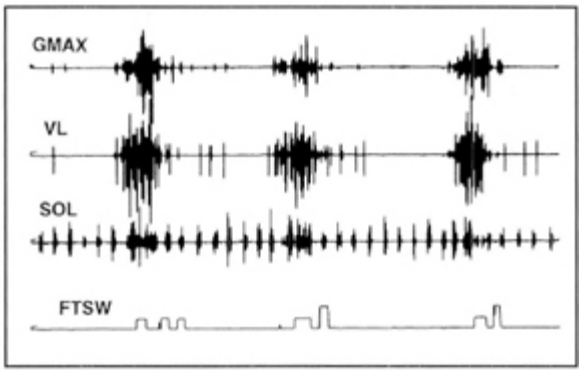

Figure 21-19. Primitive locomotor extensor synergy. Simultaneous control timing for the 3 muscles is indicated by the dense EMG segments: gluteus maximus (GMAX) hip extension, quadriceps (VL) knee extension, ankle plantar flexion (SOL). Spasticity (clonic beats)

throughout swing in soleus (SOL). Footswitch (FTSW) shows unstable stance on the fifth metatarsal only.

ELECTROMYOGRAPHIC INSTRUMENTATION

The EMG record is determined by 2 factors: 1) the intensity of the muscle action and 2) the quality of EMG instrumentation. The technical factors that influence the EMG are types of electrodes and signal management (amplification, filtering, and transmission). In addition, the ease of interpreting the record is dependent on the method of data processing and display. Computers currently available provide an excellent means of recording and processing EMG.

ELECTRODES

It is possible to record the myoelectric signals that spread through the local muscle and adjacent soft tissues with 3 types of electrodes: needle, surface, and wire. Needles are too insecure and uncomfortable for use in gait analysis. Both surface and wire electrodes are in common use. Their major differences are level of skill required and selectivity in defining muscle activation patterns.

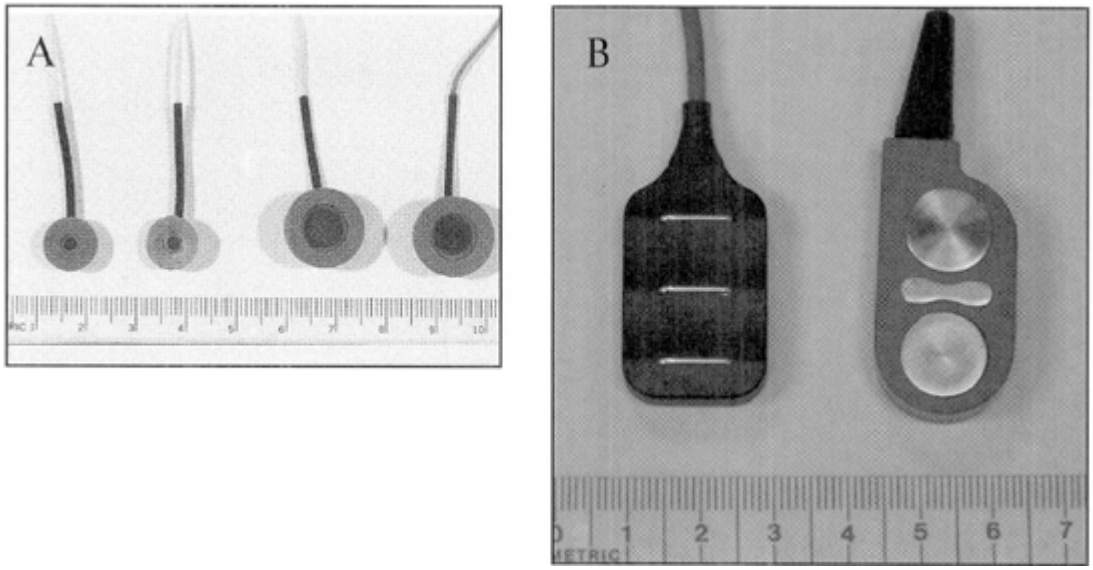

Figure 21-20. Surface electrodes. (A) Passive. (B) Active.

Surface Electrodes

Convenience and comfort are the primary advantages of surface electrodes. Any interested investigator may apply surface electrodes as the requirements are easily met; just identify the location of the muscle of interest and then tape a pair of electrodes to the overlying skin (Figure 21-20).[9,18,63]

Surface electrodes, however, have 2 significant disadvantages. First, they lack the ability to isolate the signals of the designated muscle from those produced by adjacent muscles. This becomes particularly troublesome when attempting to record from muscles located deep to a more superficial muscle. For example, differentiating EMG activity recorded from the TP from the more superficial soleus is not possible using surface electrodes. The second disadvantage of surface electrodes is that thickness of the subcutaneous fat can be a major barrier to the signals reaching the surface.

Two types of surface electrodes are in use today, passive and active (see Figure 21-20A). The passive surface electrodes are a pair of shallow, cupped, silver/silver chloride discs of varying sizes ranging from approximately 7 mm to 20 mm. The silver chloride provides a stable skin-electrode interface by diminishing the

polarization,[27] while the central cup holds a saline gel for better signal transmission.[19] Electronic grounding of the system is gained by a large remote ground plate.

Each passive electrode is a separate sensor; hence spacing between pairs can be tailored based on the specific muscle's architecture and the desired conduction volume. Small electrodes applied close together are said to best localize the signals to the target muscles. The user can define the separation between pairs of sensors.[9] Large electrodes with greater spacing detect more signal but also amplify the noise.

Active surface electrodes have built-in amplifiers and integrated circuitry to provide optimum impedance (see Figure 21-20B). Active electrodes provide preamplification of the signal at the electrode site. This reduces the relative amount of noise compared to EMG signal, thus providing a cleaner (less "noisy") signal. Electrode application is simpler because all the elements are often combined in a single unit, including the ground plate.[9] Neither gel nor skin preparation is needed.[1] For some clients, skin hair may need to be removed in the region where the electrode will be applied to ensure firm contact between the skin and the sensors.

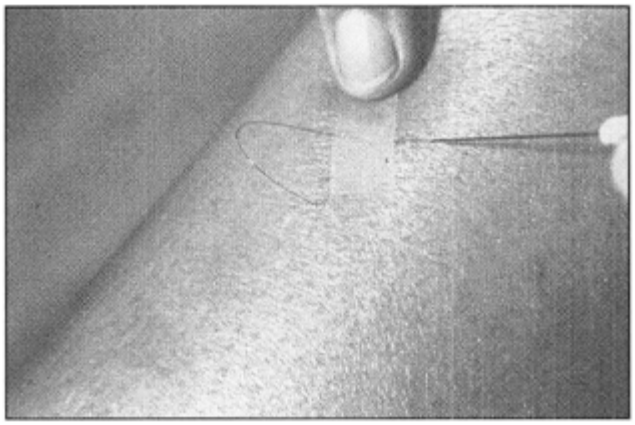

Figure 21-21. Wire electrode. Skin insertion and tension relief loop shown.

Wire Electrodes

Selectivity is the significant advantage of wire electrodes. The myoelectric signals of adjacent muscles can be isolated as they are recorded directly from the muscle (Figure 21-21). The disadvantage of wire electrodes is the need to penetrate the skin with a hypodermic needle to place the wires in the designated muscle. This limits the application of wire electrodes to a certified clinician.

The preferred electrodes are a pair of nylon (or Teflon)-coated, nickel-chromium alloy wires (50-μm diameter). The increased flexibility of 25-μm wire allows the hooks to yield with small amounts of tension. The end of each wire is bent into a hook and their 2-mm bared tips are staggered to avoid an electrical short.[38] For sterilization, these units are packaged in small envelopes in a way that avoids tangling the wires on removal.

Basmajian et al's technique of inserting both wires with a single 25-gauge hypodermic needle is the preferable method.[2] Insertion into the designated muscle is accomplished with a quick motion while the skin is stabilized. After the needle is removed, the accuracy of electrode placement is confirmed by light electrical stimulation through the electrode wires to generate a muscle contraction in combination with palpation and observation of the target muscle or its tendon.

Radiographs have demonstrated that wire electrode displacement can occur with motion,[35] generally at the time of insertion. Moving the muscle through a few passive ROM as well as having the client exert some vigorous contractions ensures early wire fixation. To prevent pulling on the intramuscular site, a 3-cm loop is formed just proximal to the fixation tape. Now wire displacement is an infrequent event. Whenever any muscle fails to display the expected action and an interim muscle test confirms the loss of signal, a second set of wires is inserted. If an insertion site is painful, then a gentle "tug" on the wires can often subtly reposition the wires to a more comfortable position. If unsuccessful at alleviating the discomfort, then a second insertion should be done as pain will inhibit normal function. Mild local bleeding at the insertion site appears to be the cause of the discomfort.[36] Following the gait study, a quick tug unbends the hook and frees the wires for removal. The client should be asked to relax

so that muscle contractions do not hinder sliding of the wires during removal.

Research using MU glycogen depletion to isolate its muscle fibers demonstrated that they are widely dispersed throughout the muscle and their immediate neighbors are from different MUs.[14] In the cat gastrocnemius, the MU territory covered as much as one-third of the entire volume of the muscle (Figure 21-22).[14] For the TA, the MU territory equaled 12% of the muscle's cross section.[13] The distances between individual fibers of the same MU in the biceps brachii range between 0.5 and 6 mm.[71] A single MU may be in contact with 50 or more other MUs. These data support the clinical experience that fine wire electrodes do not miss existing muscle action even though their contact area is small.

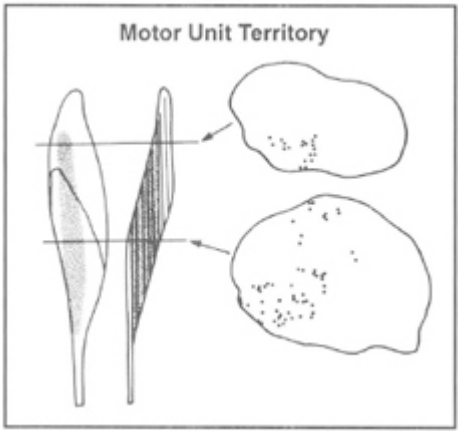

Figure 21-22. MU dispersion in a muscle. Gray areas indicate the territory of the MU. Cross sections identify the dispersion of the MU's muscle fibers. (Adapted from Burke RE, Levine DN, Saloman M, Tsairis P. MUs in cat soleus muscle: physiological, histochemical and morphological characteristics. *J Physiol*. 1974;238:503-514.)

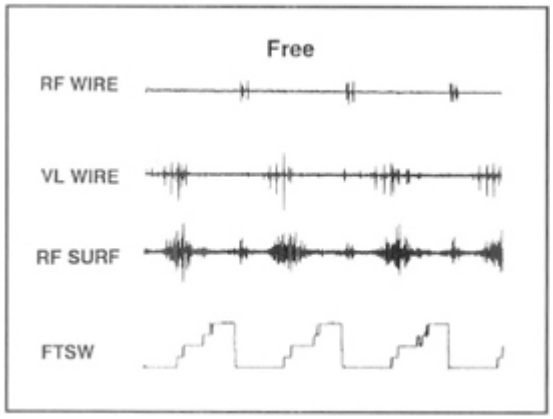

Figure 21-23. EMG cross-talk during a gait record. Rectus femoris surface (RF SURF) EMG shows 2 intervals of action that correspond to the EMG displayed by the rectus femoris wire (RF WIRE) and vastus lateralis wire (VL WIRE). Footswitch (FTSW) shows a normal sequence of foot support.

ELECTRODE SENSITIVITY

Dispersion of the myoelectric signals through the tissues by volume conduction is a normal phenomenon.[18,19,40,41,66] The signals enter adjacent muscles as well as reaching the skin surface. Passage of the myoelectric signals through the tissues progressively lowers the frequency content of the EMG as the tissues act like a low-pass filter and attenuate the higher frequencies. Because muscles function in synergy, signals from more than one muscle arrive at the same surface area, although with different intensities. Hence, the EMG record from surface electrodes generally represents action of more than the designated muscle (Figure 21-23). This is muscle cross-talk.[15,19,73]

CROSS-TALK

The ability to differentiate adjacent muscles depends on the frequency content of the EMG signals (Figure 21-24). This means the EMG spectra are different for wire and surface electrodes.

The wire data frequency range is 10 to 1000 Hz (mean = 350 Hz), while the surface electrode data range is 10 to 350 Hz with a mean frequency of 50 Hz. For both electrode systems, the peak intensity approximates 100 Hz. The lower frequency content of signals arriving at the wire electrodes from adjacent muscles can be filtered out using a high-pass filter.

Surface electrode cross-talk from adjacent muscles has been confirmed by studies using simultaneous wire and surface electrode recordings of adjacent muscles.[19,40,41,66,73] A comparison of relative electrode sensitivity to the activity of the ankle plantar flexor muscles (gastrocnemius, soleus, and TP) showed a significant difference in the EMG captured.[66] The intensity of surface and wire EMG for each muscle was compared for each of 3 standard muscle tests (Figure 21-25). Single stance heel rise with the knee flexed tested the soleus. Single stance heel rise with the knee extended was the gastrocnemius test. The TP was tested manually by the examiner providing resistance to foot and ankle inversion and PF.

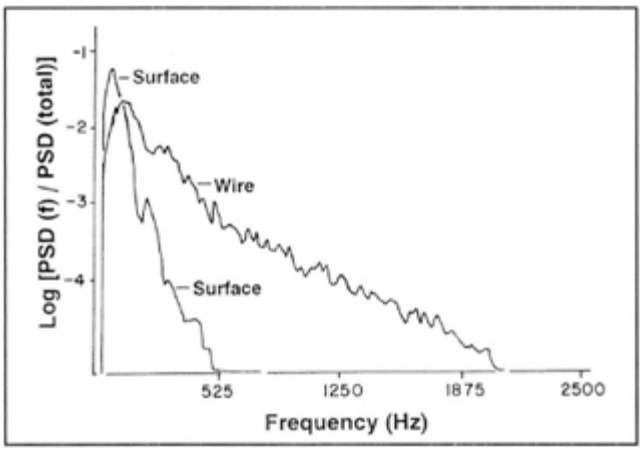

Figure 21-24. EMG power spectrum of wire and surface EMG. Vertical axis is a logarithmic scale of EMG intensity. Horizontal scale is the signal frequency.

The data showed all 3 muscles participated in all of the tests (see Figure 21-25).[66] The soleus test produced the highest wire EMG of all 3 muscles. Soleus wire EMG also was twice the magnitude of the soleus surface electrode data. The gastrocnemius test resulted in

equal height of wire and surface EMG. The posterior tibialis test produced more surface EMG than wire EMG.

These findings identified several significant events.[66] The soleus was the dominant ankle plantar flexor of the ankle. There was strong soleus activity during both heel rise tests (knee flexed or extended), while flexing the knee markedly reduced the action of the gastrocnemius. Even during the manual test of the posterior tibialis, the soleus also was a participant. The conclusion was that the wire electrode data represented the relative intensity of the 3 muscles, while the surface electrodes included considerable cross-talk.

Two other studies of cross-talk have used electrical stimulation to track the spread of the EMG signals. Stimulation of the anterior tibialis muscle generated EMG potentials in the surface electrodes over the peroneus brevis and soleus muscles. The intensity reached 16% of a primary contraction.[19]

Cross-talk also was demonstrated in the thigh by stimulating the quadriceps through its femoral nerve.[40] This generated EMG signals in both the lateral and medial hamstrings. The intensities with single differentiation were 17% and 11% of voluntary maximum effort. This dropped to 7% and 4% with double differentiation.

The differences in surface and wire electrode selectivity define the appropriate uses of each system. Surface EMG should be limited to studying group muscle action and areas without prominent obesity. Also, the possibility of cross-talk should be recognized whenever antagonistic muscle action accompanies moderate- to low-level EMG for the agonist muscle (10% to 15%). The existence of cross-talk blurs the onset and cessation times of the muscles. Similarly, cross-talk in surface electrode systems can imply cocontraction when it is not present.

Wire electrodes are necessary whenever accurate knowledge of individual muscle action is critical to the clinical or research decisions.[18,19,66] Wire electrodes should be chosen in situations where it is important to differentiate the action of adjacent muscles or to make finite judgments on patients' abnormal function.

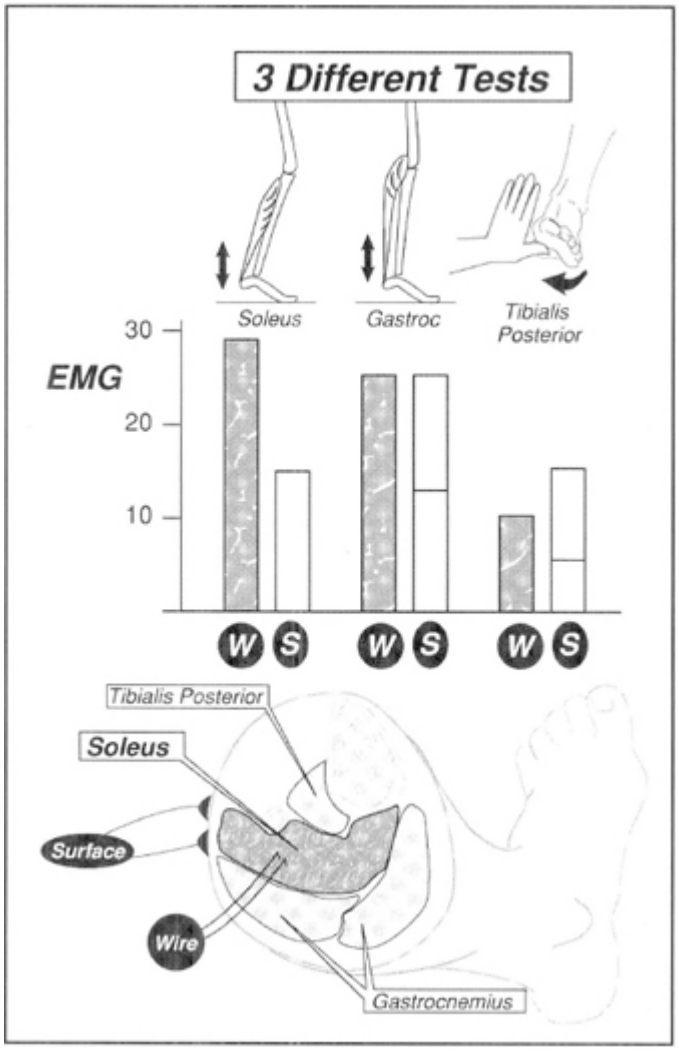

Figure 21-25. Cross-talk recorded by the soleus surface electrodes during 3 muscle tests. Top row: The 3 muscle tests performed were soleus = flexed knee heel rise; gastrocnemius = extended knee heel rise; and TP = manual resistance to inversion. Middle row: Quantified EMG data. W = wire; S = surface. During the soleus test, wire EMG recorded greatest EMG, surface recorded half as much. When evaluating the gastrocnemius, both the wire and surface EMG recorded equal quantities (gray bar delineates surface EMG in excess of normal surface-to-wire ratio). During TP testing, surface EMG recordings were higher than fine wire (gray bar = cross-talk). Bottom row: Anatomic diagram shows the cross section relationship of the muscles tested and the location of the electrodes (wires and surface).

Amplifiers and Signal Filtration

EMG signals are too small (300 μv to 5 mv) for direct interpretation without amplification. Wire electrode data are clear and stable when amplified by a factor of 1000. Generally, surface electrode data require 2 or 3 times greater amplification. A differential amplifier with a high common mode rejection ratio (80 db or higher) should be used. This type of amplifier reduces the interference of signals from common sources such as electric power lines. The input impedance must be high, at least 1 M-ohm to adequately pick up the low-level EMG signals.

The frequency content of the signals must be considered when selecting an amplifier. The basic spectrum of myoelectric signals includes frequencies between 10 and 1000 Hz. Higher frequencies have been identified but they represent less than 0.01% of the spectrum.[64] To preserve the quality and quantity of the signal, the amplifier selected must have a frequency bandwidth that covers the frequency range of the myoelectric signals obtained with the selected electrode system. For wire electrodes, a high frequency response of 1000 is more than adequate (as 90% of the energy is below 700 Hz). For surface EMG, the high end of the amplifier's bandwidth should be 350 Hz. The low-end frequency response of the amplifier could be as low as 10 Hz for both electrode systems but signals of 10 to 15 Hz and even 25 Hz are generated by the tissue displacement from muscle contraction and floor impact. These artifacts are excluded by limiting the low end of the frequency response to 40 Hz.[1] A notch filter excludes the 60 Hz noise from the environment (eg, lights, motors).

For wire electrodes, the Rancho system uses a 150- to 1000-Hz bandwidth to exclude the myoelectric signals representing cross-talk from adjacent muscles. This system was designed to exclude the signals of the surface EMG as by definition they represent spread, making wire electrodes quite selective for just the target muscle. The surface electrode bandwidth used at Rancho is 40 to 1000 Hz.

Loss of the clean baseline indicates the EMG system needs service. With visual analysis, the contaminated baseline can be

partially accommodated by conscious exclusion of the faulty signals. Automatic analysis, however, will include the noise as small levels of muscle action unless these signals are filtered prior to processing. A simultaneous blast of signal on all channels usually is indicative of electronic “noise” experienced during loss of signal transmission in telemetry systems. It needs to be excluded before the data are quantified or interpreted. Another source of noise includes loose connections/plugs. These can be identified by gently wiggling the connectors and lightly shaking the recorder pack while recording to reproduce the aberrant signal. Also, surface electrodes must be solidly affixed over a muscle.[69] If not, a rocking motion can occur when underlying muscles contract. This can cause a break in contact between the skin and a portion of the electrode, leading to noise in the signal. Use of compressive wraps, in addition to tape, is useful for securing sensors over muscles of the lower extremity.

Transmission of the myoelectric signals from subject to recorder can be by either cable or telemetry. Small cables are easiest but they introduce a drag that has been proven to slow the patient’s walking speed.[83] Suspending the cable overhead avoids this restraint. Telemetry was considered the ideal system because it offers complete subject freedom. The high need for service has lessened its popularity. The frequency used for telemetry transmission needs to differ from channels that others in the area may be using for broadcasting. As some communication devices used on construction sites use similar frequency ranges, there is a potential for noise interference when transmitting on the same channel.

Analysis Systems

A number of commercially available computer software packages enable real-time monitoring of EMG data during collection. Postcollection, the dynamic EMG data can be normalized to either the maximum voluntary contraction or to the maximum EMG recorded during gait depending on the software purchased. Additionally, some software packages permit the operator to use footswitch or kinematic data to define stride events and to relate the

EMG activity to the GC. Many labs have also developed their own customized software to automate the processing of EMG data.

Care must be taken when using automated programs to process EMG due to inevitable presence of “noise” within dynamic EMG recordings (ie, unwanted signals arising from tissue motion and environmental influences such as nearby lights and motors). Despite automated approaches to filtering noise (eg, band pass filters and notch filters), a trained eye should still screen the EMG to confirm the integrity of the signal prior to submitting it to automated processing programs.

CONCLUSION

Kinesiological EMG provides a means for determining the source of kinematic gait abnormalities. Variations in timing of muscle activity and intensity of effort can be a source of deviations (eg, overactivity of the ankle plantar flexors and invertors leading to an equinovarus foot). At other times, these differences may reflect a compensatory mechanism to accommodate for the presence of other impairments (eg, prolonged activity of the vastii during stance to stabilize a knee that is prevented from fully extending due to the presence of a knee flexion contracture). Information derived from EMG recordings can be used to guide clinical treatment interventions (eg, strengthening, stretching) as well as surgical interventions (eg, muscle transfers and releases). Selection of fine-wire versus surface EMG should be driven by the clinical or research question and the availability of personnel with the prerequisite skills.

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Chapter 22

Kinetics of Gait Ground Reaction Forces, Vectors, Moments, Power, and Pressure

The pattern of limb motions and selective muscle control that characterizes walking also includes a sequence of force interactions generated to catch, lift, and propel the body along the desired path of progression. Three measurements commonly used to describe the kinetics (or forces) influencing gait are linear force, dynamic force, and fast dynamic force. The functional equivalents of these measurements (listed in the same order) are GRF, moment of force, and power.

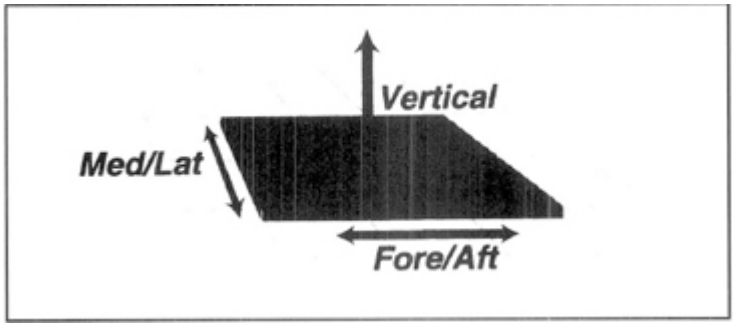

Figure 22-1. Force plate and direction of forces recorded. Vertical; Fore/Aft = forward and backward horizontal shear force (plane of progression); Med/Lat = medial and lateral horizontal shear force.

GROUND REACTION FORCES

There is no means of directly measuring the forces muscles generate during walking. While the timing and relative intensity of muscle activation can be identified by EMG, the absolute force that is generated cannot be noninvasively measured. Muscles are hidden by overlying tissues and their function is complex. Each change in joint position modifies the muscle's leverage, fiber angle, and tendon alignment. Muscle force also differs depending on the mode of contraction. A concentric (shortening) contraction generates less force than is attained during an isometric or eccentric contraction that includes the series elastic components. The difference may reach 20%.

Newton's third law of motion provides a means for indirectly estimating the forces muscles generate. As body weight drops onto and moves across the supporting foot, vertical and shear (anterior-posterior [AP] and medial-lateral [ML]) forces are generated. The immovable floor reacts with forces of equal intensity but opposite direction to those being produced by the weight-bearing limb. A force plate mounted in the floor can be used to measure and quantify the GRF as vectors with both magnitude and direction. By relating the reaction forces to the joint positions and movements at each percentage of the GC, muscle activity and demand can be inferred. EMG, however, is required to confirm specific muscle activation patterns.

MEASURING TECHNIQUE

Measurement of the GRF is accomplished with a force plate set into the center of the walkway.[14] It consists of a rigid platform suspended on piezoelectric or strain gauge transducers. In the piezoelectric-based force plate system, each supporting corner has 3 sensors set at right angles (orthogonal) to the others. The vertical load and horizontal shear forces in the fore-aft and ML directions are measured directly (Figure 22-1). In strain gauged-based force plates, the 4 load transducers are located between a top plate and a base plate. The forces are measured as they are transmitted from the top plate to the base plate through the load transducers.

Force data are gathered by merely having the subject walk across the plate but several technicalities must be observed to ensure an accurate outcome. It is important that the subject spontaneously load the force plate as a natural event during the course of moving along the walkway. Deliberate stepping onto the plate (called *targeting*) alters the data by slowing the gait velocity and introducing artificial limb motion. Targeting is avoided by having the plate camouflaged, mounted even with the floor surface, and covered with the same material as the rest of the floor. In addition, the subject's awareness of the plate is minimized by focusing his or her attention on the far wall.

To obtain accurate data, it is essential that the test foot completely contacts the plate while the other foot remains clear of the plate (Figure 22-2). This latter requirement often means repeating the test several times before the proper foot contact pattern is attained. While force plates are now available in many sizes, the area of the standard commercial force plate (approximately 40 x 60 cm) leaves little room for error during a walking trial. The use of 2 to 4 plates in series gives greater walking freedom and facilitates capturing bilateral activity. This is a major investment, however, as the costs of the force plates plus the supporting electronics are correspondingly multiplied.

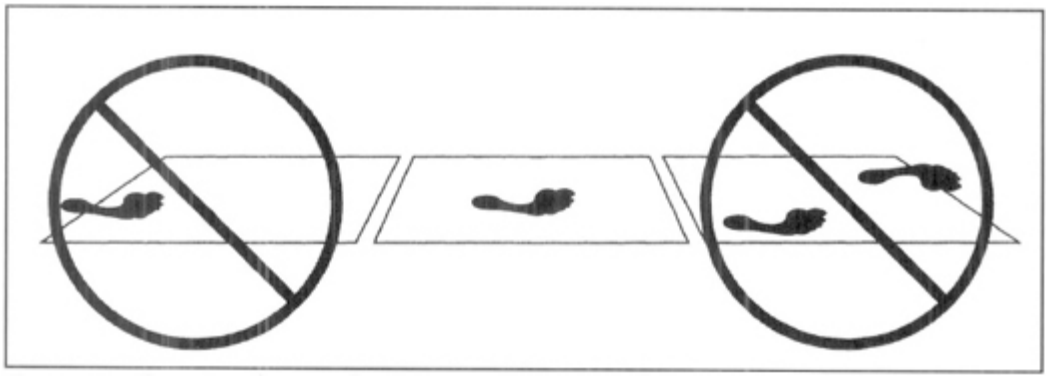

Figure 22-2. Correct and wrong foot contact patterns on a force plate. Left: Partial foot on plate (wrong). Center: Total contact by one foot on plate (correct). Right: Other foot also striking plate (wrong).

As body mass advances across the supporting foot, sampling the GRF each percentage of the GC provides the basic information

needed to determine the force of the controlling muscles and the linear strain imposed on the joints. The GRF characteristics that are most clinically relevant to walking are vertical load, horizontal shear, vector alignment, and COP.

VERTICAL LOAD

During gait, the magnitude of the vertical force varies with the changing limb position. In the sagittal plane, the sequence of the normal forces generated during stance forms 2 peaks separated by a valley (Figure 22-3A). At the customary gait velocity (82 m/min), each peak approximates 110% of body weight while the valley force is about 80% of body weight. Commonly these peak vertical forces are designated as F_1, F_2, and F_3 (Figure 22-4). The first peak (F_1) occurs at the transition between loading response and mid stance (12% GC) in response to the challenge of WA and SLS. The peak magnitude is increased above body weight by the addition of acceleration from the rapid drop of the body mass and muscular forces stabilizing the joints. The mid stance valley (F_2) is created by the rise of the COG as the body rolls forward over the stationary foot. The second peak (F_3), occurring in late terminal stance, is modified by the push of ankle plantar flexor muscles against the floor in addition to the downward acceleration of the COG as body weight falls forward over the forefoot rocker.

Normal gait also generates a sharp impact force at initial contact (called a heel transient, F_o or F_i). The impact intensity ranges from 50% to 125% of body weight and has a brief duration (1% to 2% GC).[24,27] Depending on the rate of recording, it is seen as an independent spike or as an overlay on the ascending arm of the first peak (F_1).

The magnitude of the vertical force changes as gait speed is varied.[12,18] Walking at a slower velocity reduces the vertical acceleration, which results in a corresponding decrease in the height of the peaks and depth of the valley. The normal slow stroll (60 m/min) generates a flat plateau at a level equal to body weight

(Figure 22-5).[22] Pathology that limits the rate of limb loading also lowers the vertical GRF peaks.[4] Conversely, fast walking speeds induce higher peaks and lower valleys. Running (see Figure 22-5) registers peaks almost 2.5 times body weight.[17,18] Hence, the rate of loading the limb is the determinant of peak load, and this rate is influenced by gait velocity.[21,25]

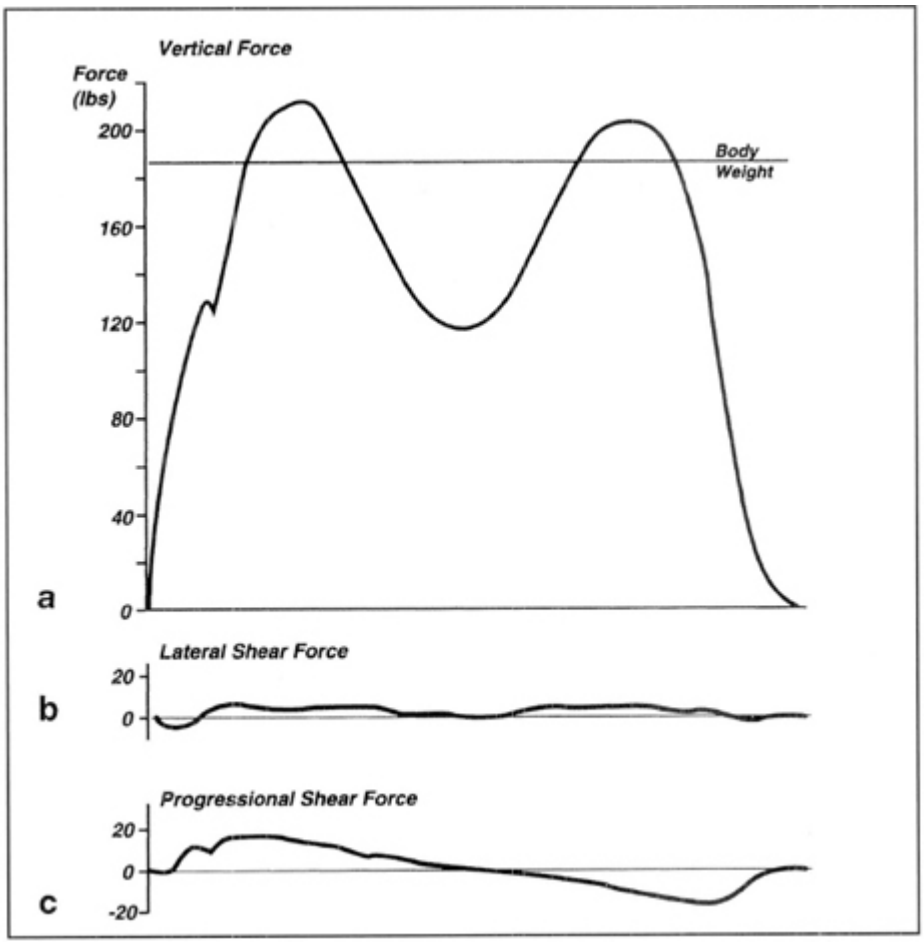

Figure 22-3. Normal ground reaction force pattern during stance. (A) Vertical force. (B) Lateral shear (medial-lateral). (C) Progressional shear (fore-aft or AP).

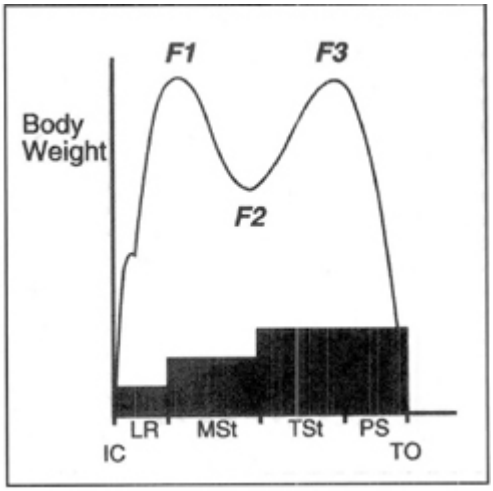

Figure 22-4. Vertical ground reaction force pattern. F1 = loading response peak; F2 = mid stance valley; F3 = terminal stance peak. Shaded base indicates foot contact pattern by step height (heel, H-5-1, 5-1). LR = loading response; MSt = mid stance; TSt = terminal stance; PS = pre-swing.

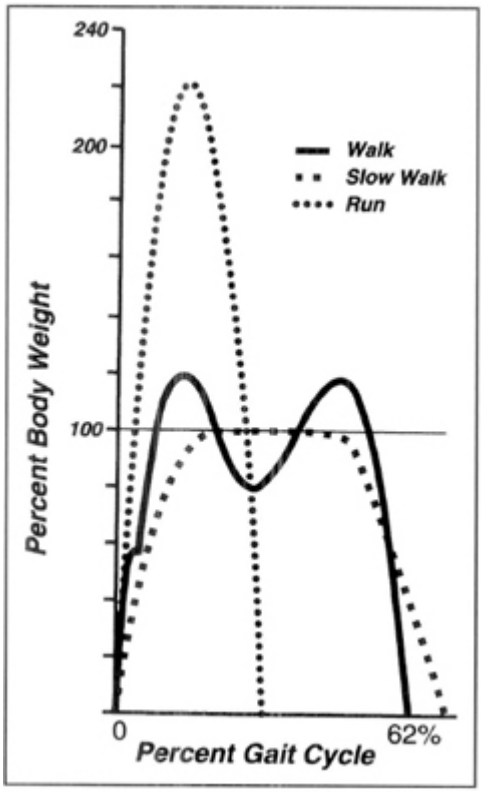

Figure 22-5. Vertical force variations with changes in velocity during running, walking (80 m/min), and slow walking (60 m/min).

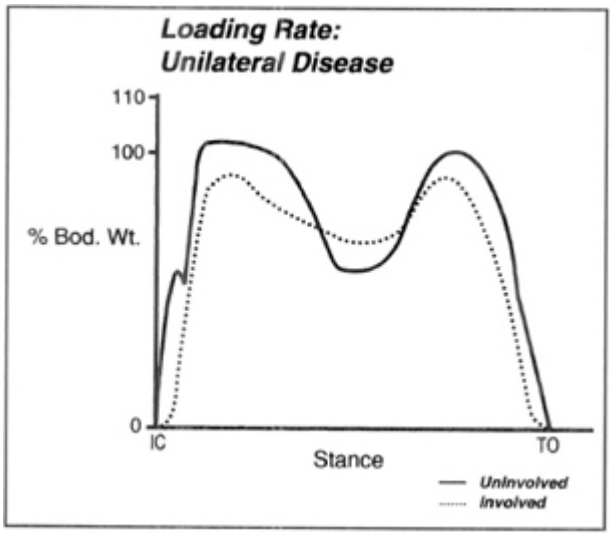

Figure 22-6. Asymmetrical vertical force pattern with unilateral hip pathology.

Pathology also impacts the vertical GRF pattern.[1,11] Individuals with hip osteoarthritis demonstrated a reduced peak vertical GRF and reduced rate of vertical loading compared to control subjects walking at a similar speed (Figure 22-6). It is possible that pain, a common finding in persons with osteoarthritis, may contribute to this altered GRF pattern. In addition, protective mechanics such as rapidly lifting one's arms may prevent the highest peak from equaling body weight. Consequently, vertical load is not a reliable clinical measure when disability is severe.[10] Better functional measures are gait velocity and single limb stance time.

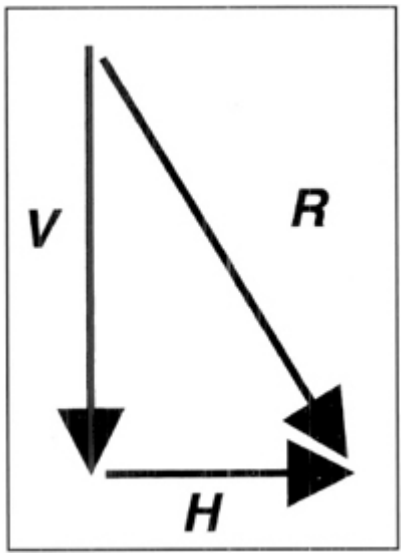

Figure 22-7. The force vector (R). A result of the simultaneous vertical (V) and horizontal (H) forces in one plane (sagittal or coronal).

HORIZONTAL SHEAR

The forces generated parallel to the walking surface are called shear. Horizontal forces in the AP and ML directions are evidenced by the GRF vector deviating from absolute vertical. Without adequate friction at the foot/floor interface, these shear patterns would result in the foot sliding and potential threats to stability.[6-8]

The magnitude of the shear force is small compared to the vertical component of the GRF (see Figures 22-3A and 22-3B). Peaks occur in early stance and late stance in the AP direction (also called fore-aft).[6-8] In early stance, the foot imparts an anteriorly directed force onto the ground at the end of loading response (peak of approximately 13% BW).[3,13] Mid stance is an interval with minimal shear in the sagittal plane. Just before heel rise, the foot starts to generate a posteriorly directed shear force that increases throughout terminal stance to a final peak of 23% BW. In the frontal plane, the peak medially directed shear force (5% BW) generated by the body occurs in the middle of loading response. The laterally directed shear reaches a peak of 7% BW in terminal stance.[3]

VECTORS

The GRF can be represented by a single vector that combines the simultaneous vertical, AP, and ML components recorded from a force plate. This vector has both direction and magnitude (Figure 22-7). The magnitude of the vector is equivalent to the hypotenuse of a right triangle with the arms being the vertical and horizontal forces (see Figure 22-7). The slope of the vector is equivalent to the ratio of the vertical force to horizontal shear. The vector intersects the force plate at the location of the instantaneous COP. The GRF vector represents the summation of all the net muscle, gravitational, and inertial forces acting on the body at a given instant in time.

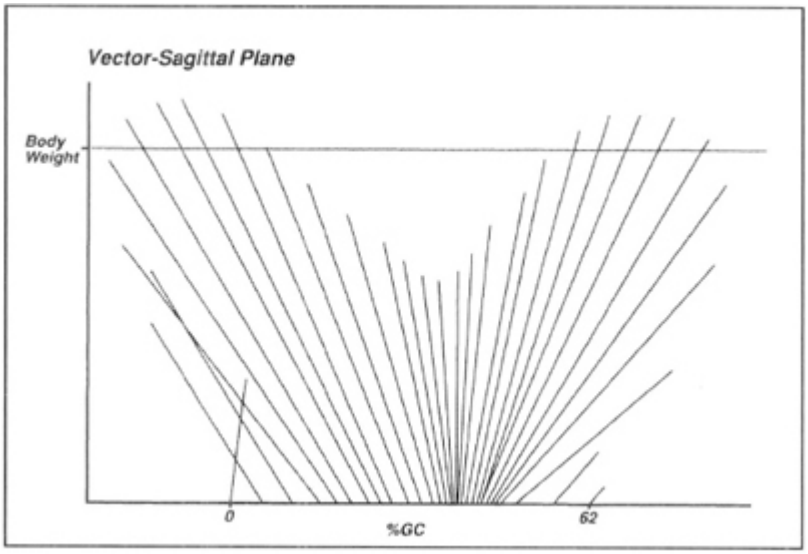

Figure 22-8. Normal pattern of sagittal vectors during a normal stride (5% GC intervals). The vectors form 4 patterns: impact, loading response, mid stance, and terminal stance.

Body weight is transmitted to the ground by the supporting foot. At any instant in the GC, there is a postural relationship between the location of the body's COM and the area of foot contact in both the sagittal and frontal planes. Sagittal plane vectors represent the resultant of the vertical and AP GRF components. Frontal plane vectors provide insights into the relationship between the vertical and ML components of the GRF.

Sagittal Plane Vectors

Between initial heel contact and toe-off, the COP advances across the length of the foot, from the heel to the heads of the metatarsals. At the same time, the COM advances from a trailing position 20° behind the contacting heel to a forward position 20° beyond the forefoot. When sampled at each 1% stance interval, these differences in length of travel create a vector alignment that resembles the ribs of a fan. The vectors can be subdivided into 4 patterns (Figure 22-8). Initial contact creates a momentary vertical force without shear. The lack of any tilt has been considered to represent inertia of the proximal body mass.[26] A second possibility is that the high speed of the normal heel strike creates a moment without shear as body weight first drops onto the floor. This last interpretation is consistent with the isolated high frequency vertical spike recorded with a highly resonant force plate.[24,27]

Subsequent vectors during the loading response reflect development of an anteriorly directed shear at the foot-floor interface. The effect is a posterior slope of the vector, which become more vertical as the limb load increases. The base of the fan is the supporting heel (see Figure 22-8).

Mid stance is an interval of nearly vertical vectors. Ankle DF allows the base of the vector (COP) to advance across the plantigrade foot in parallel with the progression of the body's COM. During this phase, the small anterior shear disappears as increasing amounts of body weight are transferred to the forefoot. In terminal stance, the forefoot becomes the dominant area of support. The rocker mobility of the forefoot allows the heel rise to progress and the development of an increasing posteriorly directed shear with a greater anterior tilt of the vector. The vector pattern is similar to the terminal edge of a fan, with each vector having a greater forward slope.

Coronal Plane Vector

The ML shear force is small (typically less than 10% BW). Consequently, the vertical force dominates the coronal vector pattern. The 2 nearly vertical peaks are very similar in timing to the sagittal plane vector pattern.

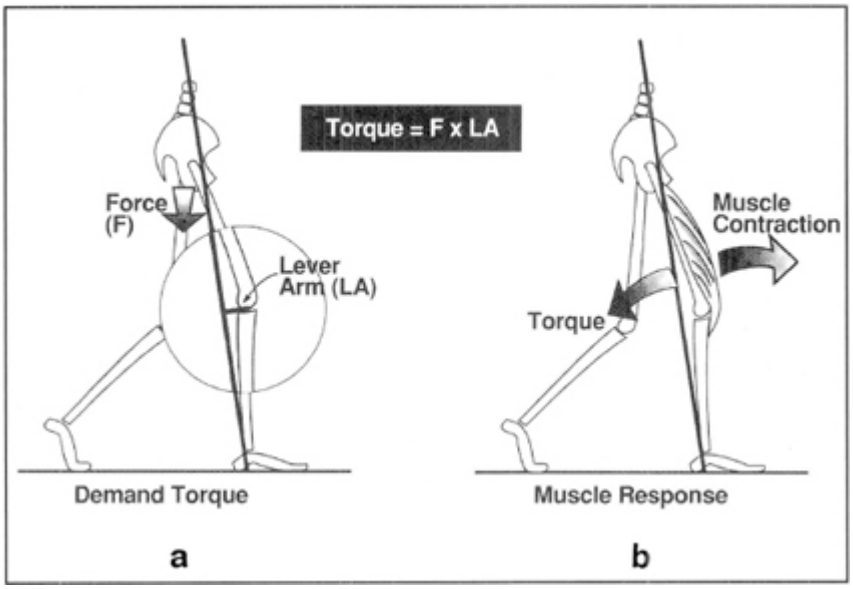

Figure 22-9. Sagittal vector at the knee. (A) Demand torque: Vector alignment behind the knee creates a flexion torque. (B) Response moment: Joint stability is preserved by the vastii, creating an equal extensor moment.

MOMENTS

During stance, the location of the mass center of the “passenger unit” (HAT) changes relative to the joints of the supporting lower limb and this influences joint stability. The perpendicular distance between the line of the body vector and the center of the joint serves as a lever, which causes the HAT to rotate the joint. The magnitude of this dynamic force approximately equals the product of the GRF (minus the limb mass distal to the joint) multiplied by the length of the lever (moment = force x lever arm). Walking adds 2 modifying factors: gravity and inertia. Gravity pulls on the proximal segment of the joint. Inertia is the resistance to change. The sum of these 3 factors constitutes a dynamic force that can be interpreted as either

external energy for motion (threatens postural collapse) or internal energy for muscle control (preserves weight-bearing stability of the joint).

Comparison of joint moments calculated with and without the effects of gravity and inertia have varied in the magnitude of difference. No difference was found at the ankle but the data for the hip and knee had intervals in the GC where the simple vector data registered higher values.[3,19,26] Bressler and Frankel noted a brief increase (approximately 20%) in both the knee and hip values at the beginning of loading response and a similar difference in terminal stance for the hip.[3] Mikosz and colleagues noted the exaggeration of the sagittal knee and hip torques (expressed in units of percent body weight x stature) was 1% each.[19] These 2 groups of authors concluded that use of the unmodified vector data was adequate for most situations.[3,19] Wells, in contrast, felt the omission of the gravitational and inertial components was a grave error. His multifactorial calculations for the hip, however, differed markedly from the results of the other studies.[26]

Two technical terms are used to identify dynamic force: *moment* and *torque*. They have the same meaning but were adopted by 2 different professions, engineering and kinesiology. Engineers focused their efforts on deriving the internal forces from external measurements. *Moment* is their preferred term (Figure 22-9). Kinesiologists focused first on motion and then integrated independent measurements of motion and muscle control. The term they prefer is *torque*. Moment and torque are both acceptable words and the choice is optional. Increasing collaboration between engineers and the clinical professions (physicians, physical therapists, orthotists, and prosthetists) behooves all groups to become familiar with both terms as they are well established in the literature.

Pathology complicates the functional picture. Muscle action may be excessive, inadequate, or abnormally timed. Soft tissue tightness may be a passive force. Identifying just the controlling force complicates the interpretation. By clearly designating the 2 stages, postural demand and muscular response, the functional situation is

made clearer and therapeutic planning is simplified. For example, during loading response, the body mass is behind the flexed knee (see Figure 22-9A). This places the vector posterior to the knee joint, creating a flexion demand, and stabilization is provided by an extensor response (ie, quadriceps action or its equivalent), which may be an orthosis or forward trunk lean to advance the body vector anterior if there is a flexion contracture.

A moment is expressed using the units Newton meter (N•m). Differing subject sizes are normalized by relating the absolute moment value to each subject's weight and height (ie, N•m/kg•m).

POWER

Rapid bursts of dynamic force are called *power*. Their magnitude is the product of the joint moment multiplied by the joint's angular velocity. Bursts of power most frequently occur at the time the mode of muscle contraction changes from eccentric to concentric activity. For example, the eccentric quadriceps moment that resists the shock of limb loading during loading response becomes a burst of concentric power at the onset of SLS. The reason for the change has not been determined. It could represent a boost of extensor power at a critical instant. Another interpretation could be an accommodation for the lower force productivity of concentric muscle action. The first interpretation is suggested by the large burst of power by the ankle plantar flexors in pre-swing. Power is expressed as watts per sec/kg•m. Positive power values indicate energy generation and are commonly associated with concentric contractions, while negative power values signify energy absorption and are frequently associated with eccentric contractions.

FUNCTIONAL SIGNIFICANCE OF MOMENTS AND POWER

While the moment and power forces during walking customarily represent contracting muscles, this is not always true. Passive tension of ligaments and sheets of fascia save energy by replacing muscle action. The lack of EMG from the designated muscles confirms the absence of muscle action.

Normal occurrences of this situation are found at the hip and knee. Dense ligaments limit extension of both joints. Throughout terminal stance, the flexor moment at the hip progressively increases, yet the hip flexor muscles are not active. An abductor moment is found in pre-swing as the COM falls toward the other limb. This is a desirable move limited only by passive tension of the fascia lata rather than abductor muscle action. The knee flexes 40° in pre-swing in response to a burst of ankle plantar flexor power. Yet neither the hip nor knee flexors registers any notable EMG.

Moments and power calculations only identify the dominant force, yet they are routinely named for the pertinent muscle group. This is justified by the volume of reference data confirming the relationship of muscle action to the moment. In normal function, there are few occasions where 2 antagonistic forces are in serious competition. Pathology, however, can create such situations. Then clarification of such a conflict requires confirmation by EMG or other clinical techniques. Remote motion also may follow the action of an adjacent segment. For example, the knee flexor moment in pre-swing is a passive reaction to the PF at the ankle. There is no EMG activity in the knee flexors. Another example is the hip flexor moment at the end of terminal stance. Passive tension of dense anterior ligaments is the opposing force.

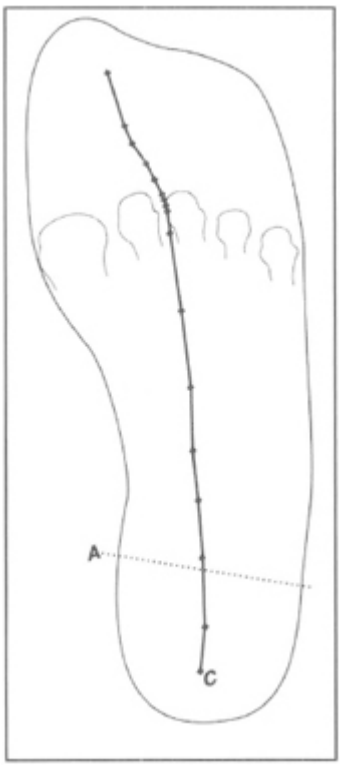

Figure 22-10. Center of pressure pattern during a normal stride. Longitudinal line (c) = path of calculated mean pressures; ♦ = gait cycle timing of mean pressure calculations; foot outline = tracing of subject's foot. Horizontal line A identifies ankle joint axis. (Data from Rancho's Pathokinesiology Laboratory.)

CENTER OF PRESSURE

The base of the GRF vector lies within the foot as this is the body segment in contact with the floor. This point is called the COP. By tracking the path of the instantaneous COP during stance, the patient's pattern of progression is determined. While the word *pressure* is in common use, it is not correct as the area of contact is not considered in the calculation. Center of support would be more accurate.

Each COP point represents the mean of the vertical forces registered on the 4 instrumented supporting posts of the force plate. By also defining the location of the foot on the force plate, an anatomical correlation with the COP can be made (Figure 22-10).

The COP is related to the plantar outline of the foot but its location does not necessarily identify the portion of the foot receiving the greatest pressure. For example, during mid stance, the location of the COP can be misleading. Support is shared by the heel and forefoot and the mean of these forces generally falls in the mid foot area, but this area may never contact the floor.

Varying technology has been developed to quantify the pressures experienced by the foot at the foot-shoe interface as well as during barefoot walking. In-shoe pressure measurement systems enable identification of potentially deleterious pressure conditions that could lead to tissue damage in those lacking protective sensation (eg, individuals with a diabetic sensory neuropathy).

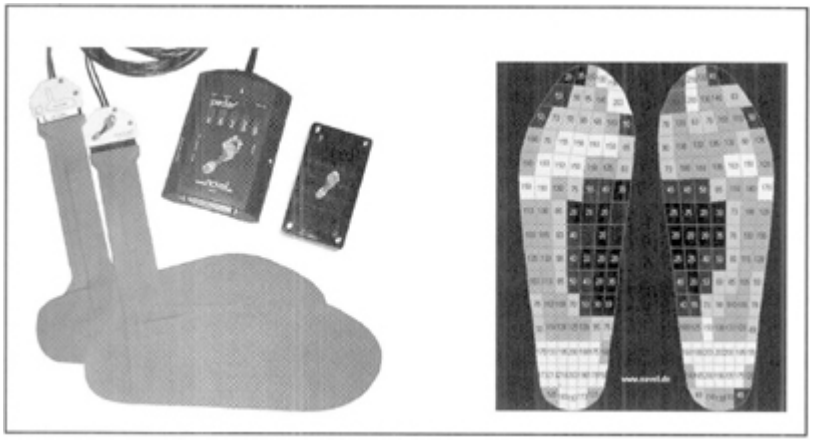

Figure 22-11. Individual foot pressure sensors allow localization of foot pressure data into discrete anatomic regions for use during clinical and research studies.

PLANTAR PRESSURE

As body weight drops onto the supporting foot, its compressive force is imposed on the plantar tissues. Relating the force to the area of contact identifies the pressure (force per unit area). This is a useful clinical measure.

To determine the pressures experienced by the different structures within the foot, there must be a means of limiting the recording to discrete anatomical areas.[2] Two approaches are currently in use:

segmented force plates and insoles with an array of individual pressure sensors.

The simplest segmented “force plate” is a Harris mat with a surface of multiple small projections that, when covered with ink, imprints an overlying sheet of paper. Primarily designed for static measurement, the relative pressure is indicated by the density and size of the inked area.[15,23] The data are qualitative yet provide an inexpensive clinical method to examine relationships between body load and anatomical foot structure on foot pressure patterns.

Instrumented insoles are the most recent development. The insoles typically contain an integrated set of sensors.[9,16] Each sensor records an independent vertical force that allows discrete localization of the pressure (force/area of the sensor) within the foot. The sensors coupled with computer technology can be separated into distinct foot areas such as heel, medial and lateral arch, each metatarsal head, and the toes. Different insoles, corresponding to different shoe lengths and widths, are required to ensure accurate pressure mapping for the entire foot. The numerical and color print records (Figure 22-11) that are produced can be used for research and clinical purposes. The systems are generally costly and can exceed $100,000 for the hardware and software.

Peak plantar pressure values vary across the foot, are influenced by the use of shoes, and change across walking speeds.[5] At a comfortable walking speed of 80 m/min, peak pressures during shod walking exceeded 200 kPa under the great toe (270 kPa), central metatarsals (250 kPa), heel (230 kPa), medial metatarsals (220 kPa), and lesser toe (210 kPa) regions in a group of healthy older adults (mean age 70 years).[5] The lowest pressures were recorded under the lateral metatarsals (150 kPa), lateral arch (110 kPa), and medial arch (100 kPa). While walking more slowly (57 m/min) resulted in a significant decrease in peak pressure under the heel, great toe, lesser toes, and medial and central metatarsals, walking more briskly (97 m/min) significantly increased pressure under the heel and great toe.[5] Barefoot walking also resulted in significantly higher pressures under both the heel and central metatarsals compared to shod walking.[5]

Terrain also influences pressures.[20] In a group of young, healthy adults, peak pressures during barefoot walking were significantly greater beneath the greater and lesser toes when traversing concrete versus either grass or carpet. The use of shoes, however, eliminated the peak pressure differences across terrains.

CONCLUSION

Loading the limb in stance initiates significant force on the floor in all 3 directions (vertical, AP, and ML). As the body realigns itself over the supporting limb, the magnitude and direction of these forces change. Analysis of these forces can significantly contribute to the understanding of the mechanics of gait. Joint torques can lead to potential threats to postural stability at a joint, or alternatively can enhance stability while minimizing muscle demands. The vertical GRF can contribute to deleterious regions of high pressure if not adequately distributed across the foot-floor interface.

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Chapter 23

Stride Analysis

The natural mix of joint mobility, muscle strength, neural control, and energy leads to a customary walking speed, stride length, and step rate. These time and distance factors, in combination with the swing and stance times, constitute the person's stride characteristics. They represent the individual's basic walking capability.

Velocity (or walking speed) is the fundamental gait measurement. Walking speed defines the person's rate of travel by identifying the time required to cover a designated distance. Technically, the term *velocity* is the more exacting measurement as the direction of travel is a factor. The combination of direction and magnitude classifies "velocity" as a vector physical quantity. Usually this is an insignificant point as functional testing is routinely done in the forward direction, although getting a child to walk continuously in a straight line is a challenge. The term *speed* is a numerical (scalar) value independent of direction.

By strict scientific rules, walking speed is expressed in meters per second (m/s) to follow the International Standards of Measurement. Many clinicians, however, prefer to express walking speed as meters per minute (m/min) in order to be compatible with the more understandable designation of cadence (steps per minute) and the custom of relating energy cost units to meters traveled. Persons without pathology, while able to voluntarily modify their gait velocity as needed, also have a spontaneous rate that is called either *free* or *customary walking speed* (CWS).[30] This free rate of travel designates the optimum functional balance of the person's physical qualities.

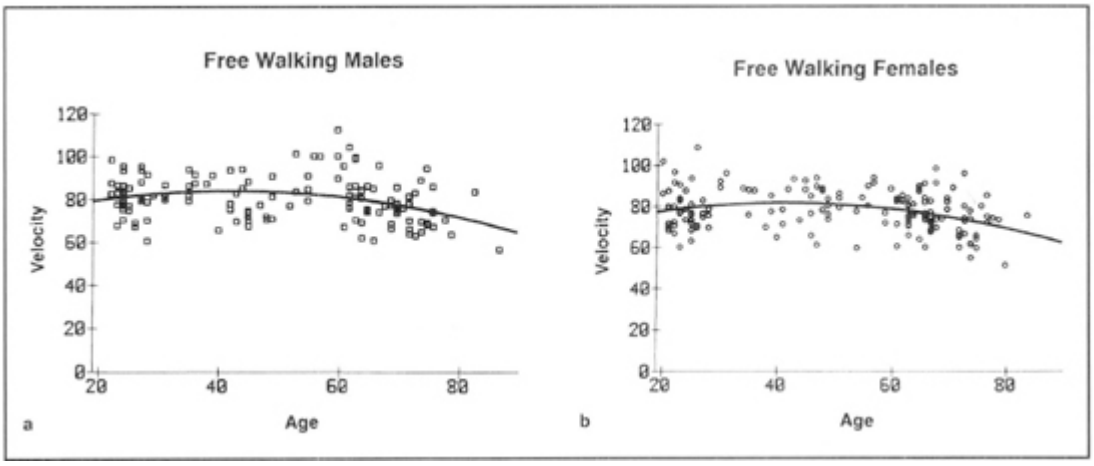

Figure 23-1. Normal velocity during free pace walking. (A) Males (N = 135). (B) Females (N = 158). Vertical scale = meters/minute; horizontal scale = age (20 to 85 years).

Normal free gait velocity on a smooth level surface averages 82 m/min for adults. Men are 5% faster (86 m/min) than the group mean (Figure 23-1A). Women's walking velocity (77 m/min) is 6% slower (Figure 23-1B). These laboratory values are similar to those of Murray[20,22] and 2 studies involving the covert observations of pedestrians.[8,10] The range of mean values among these studies was 80 to 91 m/min for the men and among the women it was 73 to 81 m/min. Stride analysis during 5-minute energy cost measurements on an outdoor, 60-meter track led to similar results (mean 80 m/min, men 82 m/min, and women 78 m/min).[30]

The primary determinants of gait velocity are the length and repetition rate of the person's stride.[1,5,15,20,28,29] This relationship tends to be linear and relatively consistent for individual subjects. In practice, steps rather than strides are counted. Step rate commonly is called *cadence*. The relationship between walking velocity and its determinants is calculated as follows:

$$\text{Velocity} = \text{Stride Length} \times 0.5\ \text{Cadence}$$
$$(V = SL \times 0.5C)$$

Stride length for persons without gait pathology averages 1.41 meters. Men have a 14% longer stride length than women. The men average 1.46 m and women 1.28 m (Figure 23-2). Children have a

significant increase in stride length for each year of growth until they reach 11 years of age. After this, the changes are minor.[2]

Cadence (step rate) of women (117 steps per minute) is faster than that of men (111 steps/min) (Figure 23-3). This nearly compensates for their shorter stride length. The mean adult cadence (men and women) is 113 steps/min. Children reduce their cadence with age.[2]

NORMAL VARIABILITY

Normal adults show a moderate variability in their free walking velocity. One group of 60 subjects ages 20 to 65 years showed a 7% standard deviation during indoor testing following pretraining to a common cadence.[20] Another 111 persons ages 20 to 80 tested on an outdoor track displayed a 4% deviation.[30] Two identifiable sources of this variability are age and height (or limb length).

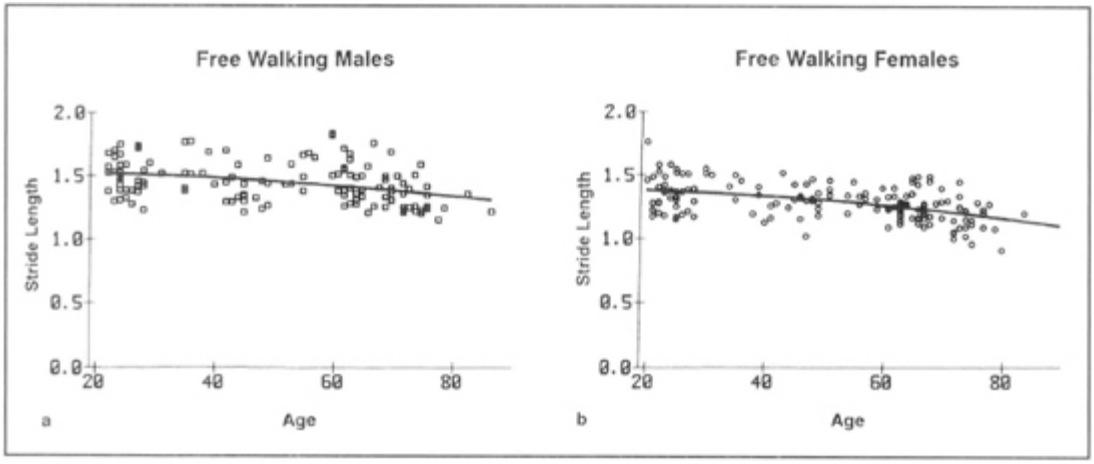

Figure 23-2. Normal stride length during free pace walking. (A) Males (N = 135). (B) Females (N = 158). Vertical scale = meters; horizontal scale = age (20 to 85 years).

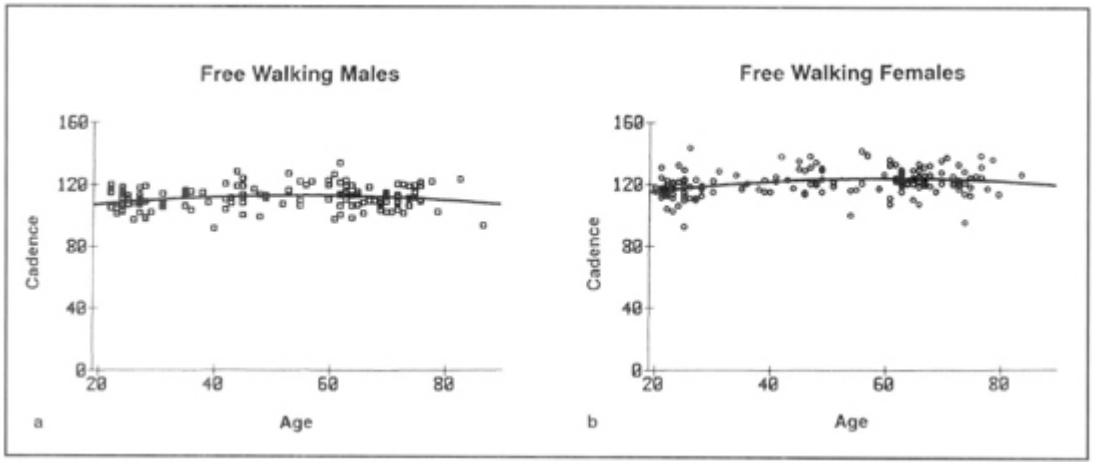

Figure 23-3. Normal cadence during free pace walking. (A) Males (N = 135). (B) Females (N = 158). Vertical scale = steps/minute; horizontal scale = age (20 to 85 years).

AGE

Studies of aged gait in adults show a notable difference in velocity (14%) when arthritis and other disabilities are allowed as natural events.[9] In healthy adults, gait characteristics such as velocity and stride length remain relatively unchanged until the seventh decade of life.[14] After 60 years of age, reductions in velocity have been documented[4,14,16] and become more pronounced with age. The decrease in mean velocity was just 3% for a 60- to 65-year-old group.[21] This increased to 9% when the study group included ages 60 to 80 and 11% with the upper age limit of 87 years.[5] The Rancho study of 247 persons ages 20 to 84 showed that the significant decline in walking ability began after age 70 years. The reduction in gait velocity arises, in large part, due to decreases in stride length of approximately 7% to 20%.[13,16,20,21,23,31]

Alterations in stride characteristics in older adults may arise in part from muscle weakness. In a group of sedentary older adults (mean age 75 years), maximum isokinetic strength of the hip extensors served as a key predictor of walking velocity, stride length, and cadence, accounting for 37% (r = 0.61), 35% (r = 0.59), and 12% (r = 0.34) of the total variance in these variables, respectively.[4]

The consistency of gait characteristics across steps also changes with age. For example, greater variability in stride-to-stride width was found in healthy older adults compared to younger adults.[11] Interestingly, as stride-to-stride width variability decreased, the likelihood of a fall occurring during locomotor activities increased in one prospective study of older adults.[19]

Limb Length

The increase in leg length in the growing child is obvious. Between the ages of 1 and 7 years, the mean leg length increases 194% (31.6 cm to 61.5 cm, r = 0.95).[29] Variability also doubles (standard deviation increasing from 2% to 4% of the mean). There is a corresponding increase in stride length and walking speed. The ratio between stride length and leg length progressively increases between ages 1 and 4 (1.36 to 1.48) and then stabilizes (age 7 = 1.57). Within this age range, the correlation between leg length and stride length is strong (r = 0.95).

A similar relationship between stature and stride length has been assumed to exist in adults[7,12] but the supporting data are weaker. Grieve and Gear found a modest mean ratio between stature and stride length (r = 0.53), consequently one can attribute only 28% (r^2) of the change in stride length to stature.[12] Men, women, adolescents, and older children showed similar correlations (r = 0.51 to 0.59). Murray found a 4% difference (r = 0.46) in the group means of tall, medium, and short men (40 each). The relationship was stronger during fast walking. This is consistent with the higher correlation (r = 0.71) found during treadmill endurance running. Endurance during running also was found to decrease 7% when the stride length was altered (tests used SL = +0.60 and -0.80 leg length).[26] These studies thus have identified that leg length can influence stride length but the relationship is weak during walking (r^2 = 0.21 to 0.28) and moderate in an endurance run (r^2 = 0.49). Hence, the recommendation that stride length should routinely be defined as a ratio of stature lacks a sound factual basis.[12,32] Das et

al also oppose this recommendation because relative values do not present a clear picture of the distance covered.[6] Thus, the standard measurements should be the basic data with relative anatomical values added if they contribute to the topic.

VOLUNTARY VARIABILITY

Normal persons have a wide range of safe and relatively comfortable walking speeds. One study of men showed a 45% increase in gait velocity with equal gains in stride length and cadence (18% each).[21] Similar testing of women registered a 35% increase in walking speed while voluntary slowing reduced the velocity by 41%. Again the changes in stride length and cadence were about equal.[22] A Rancho effort to get the slowest normal velocity resulted in a 50% reduction. Slower trials led to a disruption in the walking rhythm.

The standard deviations for normal free walking are approximately 10% of the mean value.[20,30] The preceding data indicate 4% of this deviation is related to leg length. Age has no significance until the person is past 60 years. Thus, the larger factor appears to be spontaneous variability.

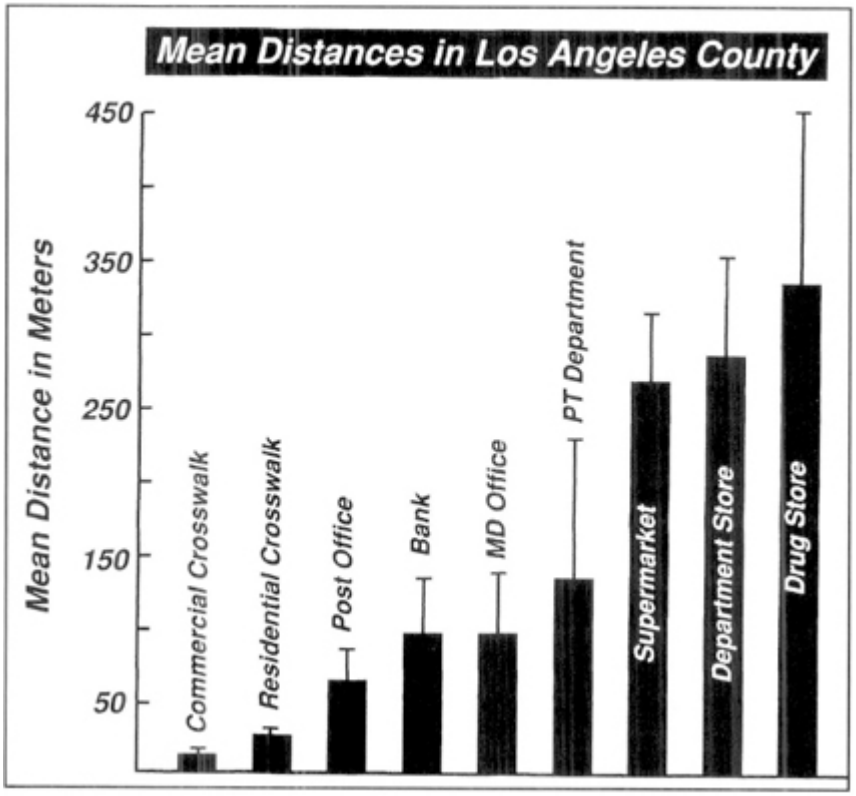

Figure 23-4. Mean walking distance in Los Angeles from available parking to different common destinations.

RANGE AND DURATION OF CUSTOMARY WALKING

Functional ambulation requires the individual to traverse a certain distance to perform a specific activity. The average distances for various daily living activities were measured in a cross section of different urban areas in Los Angeles, California. Most activities such as going to the post office, doctor's office, supermarket, or department store necessitate less than a 300-meter walking distance from available parking (Figure 23-4).[18] Assuming automotive transport is available and the average walking speed is 80 m/min, it follows that most daily living activities require less than 4 minutes of

walking for an individual capable of walking at a normal speed. For people with limited walking capacity, many daily living activities will require greater walking times. For example, it would take an individual who is only able to walk at 25% of normal velocity (~20 m/min) approximately 16 minutes to traverse from a parking lot to a drug store (see Figure 23-4).

When maximum walking speed slows, it may become difficult to safely negotiate some community environments. Langlois and colleagues,[25] in a study of community-dwelling older adults living in New Haven, Connecticut, found that less than 1% of pedestrians aged 72 years and older walked at a customary speed that would enable them to cross a street from curb to curb in the time allotted (required walking speed = 73 m/min). Even in sections of the city where signal changes were slowed to accommodate a more moderate walking speed (ie, 55 m/min) due to the greater presence of older adults, the speed requirements still exceeded the self-selected comfortable walking speed of approximately 93% of those studied.[25] Hence, for the majority of these individuals, crossing a street in a timely manner required increasing walking speed above their customary rate. While many were able to adjust their speed to meet the task requirements, approximately 11% of those studied reported difficulty crossing the street.[25]

STRIDE-MEASURING SYSTEMS

Several techniques now are available for measuring a person's stride characteristics. There are both indirect and direct approaches. The indirect techniques consist of measuring the stride characteristics from the motion recording. A particular foot or ankle marker is tracked for its pattern.

Direct techniques use the foot contact pattern with the floor. Both time and distance are significant to the direct measurements. A transparent force plate will display the relative contact times of the different foot areas as the skin changes color and shape with weight bearing. The analysis of these data is by subjective observation.

Foot pressure systems automatically provide the timing data from which stride characteristics can be calculated. Footswitch systems allow measurement of the stride characteristics and floor contact pattern in either the laboratory or clinical environment with less comprehensive equipment.

Stop Watch

The simplest means of measuring a person's walking ability is with a stop watch. Either cadence or velocity can be determined in this manner. Velocity measurements require a designated walking distance as well as timing. To minimize the effect of the examiner's reaction time, the measured walking distance should be at least 50 feet (31 m). An additional 10 feet (3 m) should be available before and after the measurement areas to exclude the variability of starting and stopping. Counting the number of steps taken during the timed interval provides cadence. If one has both cadence and velocity, then stride length can be calculated. One can use footprints on a walkway for direct step or stride measurements.

Automated stride analysis instrumentation offers more precision in the measurements. Several types of stride analysis systems have been designed, including footswitch systems and instrumented walkways.

Footswitch Systems

Commercially available footswitches consist of either a set of individual sensors or instrumented insoles. Many laboratories also fabricate their own foot switches. The in-house systems commonly include only 1 or 2 sensors (heel with or without the toe) with the limited purpose of differentiating stance and swing or just the GC timing.

Individual Sensor Systems

Sets of small (1.5 cm^2) discs designed as pressure sensors also can serve as footswitches to identify the floor contact times of the heel, individual metatarsal heads, and great toe (Figure 23-5). Thin sensors (~0.5 mm thick) are taped to the sole of the foot over the bony prominences of the heel, metatarsal heads, and sometimes the great toe. The parameters calculated vary with the system. Basically, the duration of floor contact and the stride characteristics are identified. Portability depends on the type of the recorder and analyzer used. The advantage of using individual sensors is that different-sized insoles are not required to fit inside the range of shoes commonly worn by patients. The disadvantage, however, is that collectively, the individual sensors typically take longer to apply and require basic anatomic knowledge to palpate correctly for the bony landmarks.

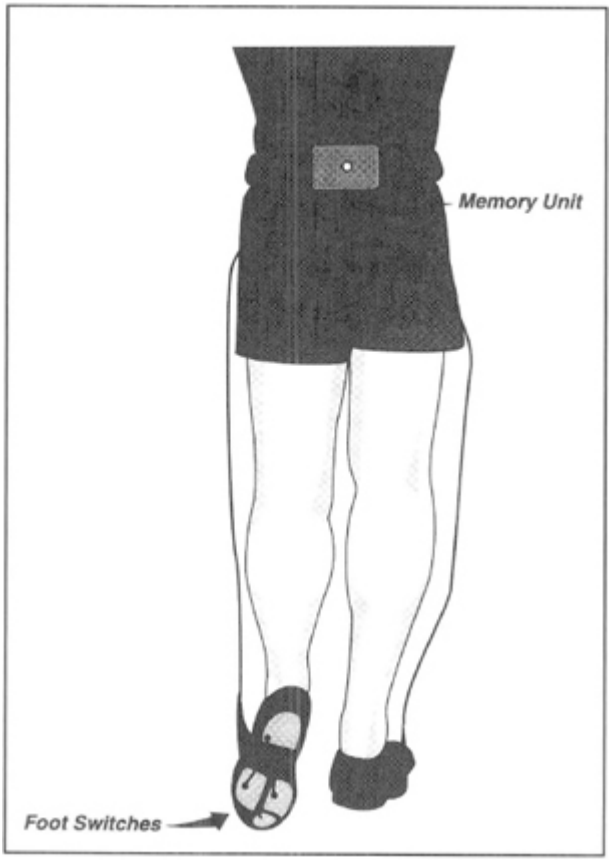

Figure 23-5. Insole footswitch system. The bottom of the left foot shows a diagram of the compression switches that are contained within an insole. The footswitches can be applied within a shoe or on

the sole. Cables from footswitch insoles plug into a memory unit. Memory unit stores footswitch signals and timer. (Adapted from Perry J. Integrated function of the lower extremity including gait analysis. In: Cruess RL, Rennie WRJ, eds. *Adult Orthopaedics.* New York: Churchill Livingstone; 1984.)

Insole Footswitch System

Each insole typically contains large compression-closing sensors in the areas of the heel (4 x 6 cm), fifth and first metatarsal heads (3 x 4 cm), and great toe (2 x 2 cm). As force (body weight) progresses across the surface of the insole during each step, the individual sensors are activated and provide an electrical signal that can be used to differentiate when each portion of the foot is in contact with the ground (see Figure 23-5). One may either slip the insole footswitch assembly into the shoe or tape it to the bare foot. There are standard-sized insoles for both adults and children. The heel and forefoot sections are separated to allow for minor length adjustments to accommodate variations in fit across shoe designs.

To avoid inadvertent activation by shoe pressure, footswitch sensitivity is approximately 8 psi (4 psi for children). This introduces an average 2% GC delay in the registered onset of stance and a correspondingly premature cessation compared to simultaneous force plate recordings (Figure 23-6). To determine the true stance and swing periods, the computer program includes a 2% correction factor to allow for the switch closure/opening delay. Reapplication assessment of the footswitches did not show a variation in the gait measurements beyond that found with simple repeated testing (unpublished data).

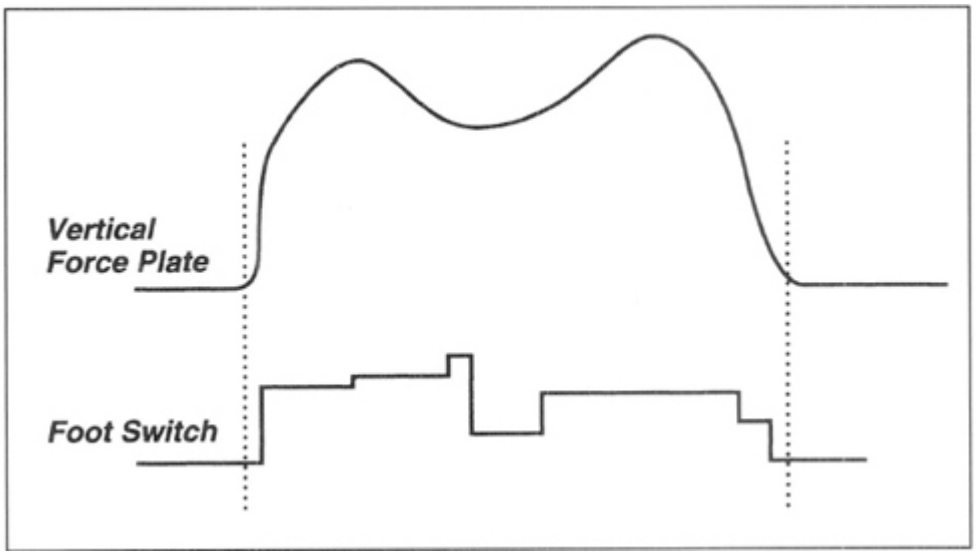

Figure 23-6. Footswitch timing compared to a simultaneous force plate record. Footswitch sensors have a 2% GC delay in onset and cessation. Horizontal scale = GC. Vertical scale not significant.

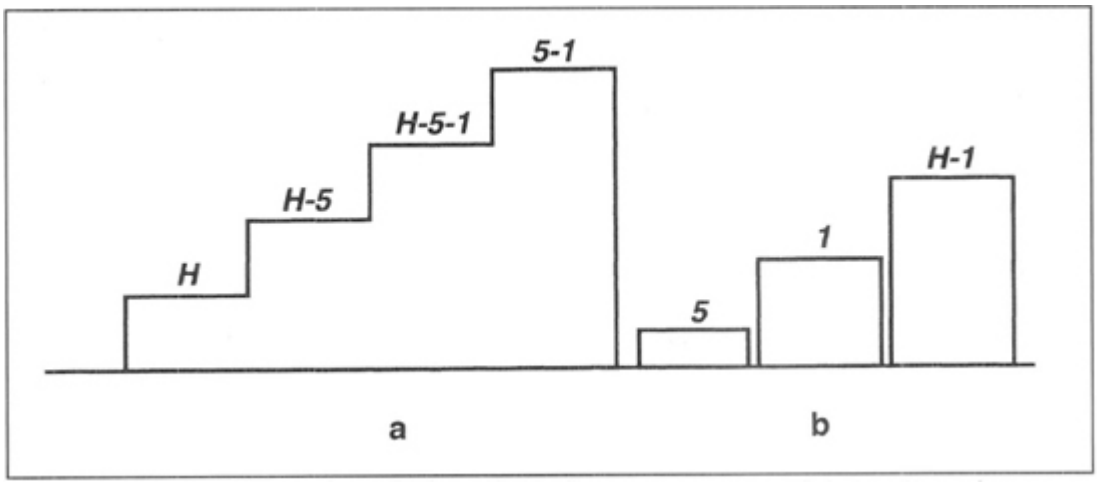

Figure 23-7. Diagrammatic scale to display individual sensor sequence and timing. Normal sequence is a stair case. Half steps designate abnormal modes of foot contact. Timing is indicated by length of step. H = heel, 5 = fifth metatarsal, 1 = first metatarsal, T = great toe.

The signals from each individual sensor have a specific voltage to differentiate its floor contact time from the others. These are processed by the computer to provide 2 types of clinical information: 1) foot support sequence and 2) timing of the individual foot area. The foot support sequence approach includes a diagrammatic

representation of the foot-floor contact sequence through stance. A normal sequence is displayed as a 4-step staircase for easy recognition. Increasing voltage levels (of equal increments) are assigned for the 4 support areas from isolated heel (H) through the 2 stages of foot flat (H-5, H-5-1) to the final area of forefoot only (5-1) or “heel off” (Figure 23-7). Abnormal support patterns of just the fifth (5), the first metatarsal (1), or the heel and first metatarsal (H-1) are assigned half-step voltages. These continue to be a part of the EMG recording system. The same information also is provided in a text format by the stride analyzer printer or as time bars by the EMG analyzer. Computer analysis also can specify the duration of floor contact by the individual sensors (eg, heel, fifth metatarsal) both in milliseconds and the GC interval.

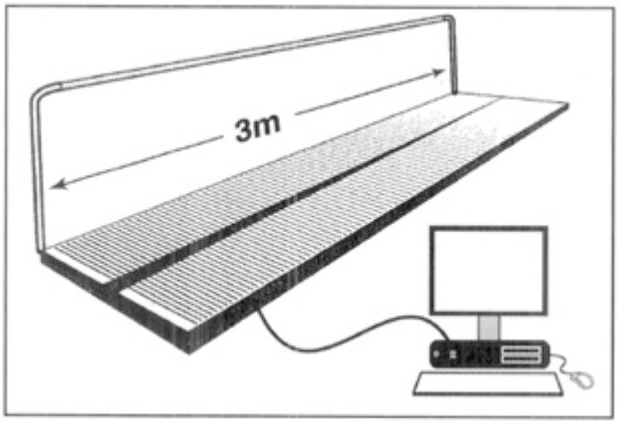

Figure 23-8. Instrumented walkway. Three-meter long mat consisting of narrow instrumented slats divided into right and left halves.

INSTRUMENTED WALKWAYS

Walkways containing on/off sensors have been developed to avoid applying any apparatus to the subject. The early basic designs used a series of electronically instrumented slats to register floor contact time (Figure 23-8). Velocity, stride length, cadence, step length, swing and stance duration, and the foot support pattern were determined by the sequence of slat contact by the 2 feet. Advantages of this type of system are that no special sensors must

be worn and no cables trail behind the subject. The disadvantage is that the walkways often require a substantial amount of floor space when in use.

Currently, 2 instrumented walkway systems are available commercially for measuring spatial (distance) and temporal (timing) parameters: the GaitMat II (manufactured by E.Q., Inc; Chalfont, PA) and the GAITRite (manufactured by CIR Systems, Inc; Havertown, PA). Both are offered in a variety of lengths ranging from approximately 12 to 25 feet and are transportable. The basic GaitMat II system is composed of 4 sections that can be disassembled for storage or transport. If required, additional sections can be purchased to extend the length of the walkway. The GAITRite (Figure 23-9) can purchased in varying lengths and can be rolled up and transported in a convenient plastic golf case.

TESTING PROCEDURES

Testing distance varies among different laboratories. Space availability is a common determinant. The essential component is having sufficient distance that several strides can be recorded. The subject must feel free to walk at his or her usual pace and there must be an interval before and after the data segment to absorb the irregularities of starting and stopping. Rancho found that a 10-meter walkway with the middle 6 meters delineated for data analysis was a convenient compromise between space, patient endurance, and data need (Figure 23-10). The data interval is designated at each end by a photoelectric cell that transmits its signal to the recording instrumentation. In-house studies demonstrated longer approach distances (6 and 12 m) did not alter the data. The probability of a 3-meter data collection area being too short is implied by the significantly slower normal velocity data obtained with a walkway of this distance compared to the data obtained over longer distances in other laboratories.[3,17,27]

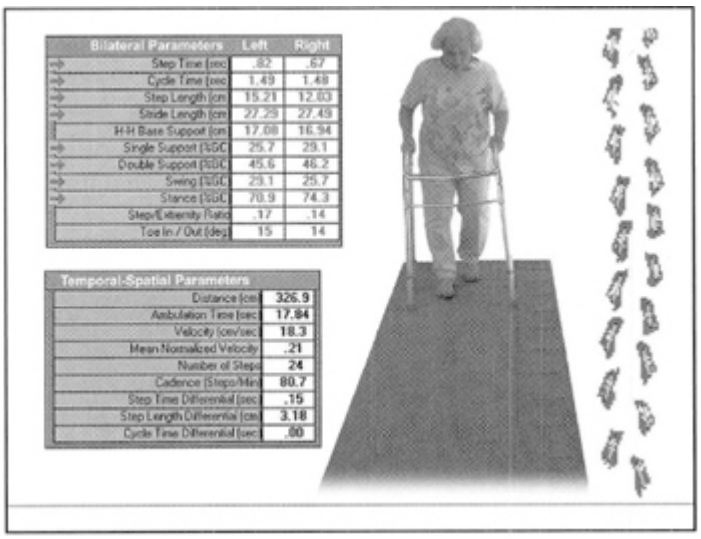

Figure 23-9. Client walking across the GaitRite mat without needing to wear any additional equipment.

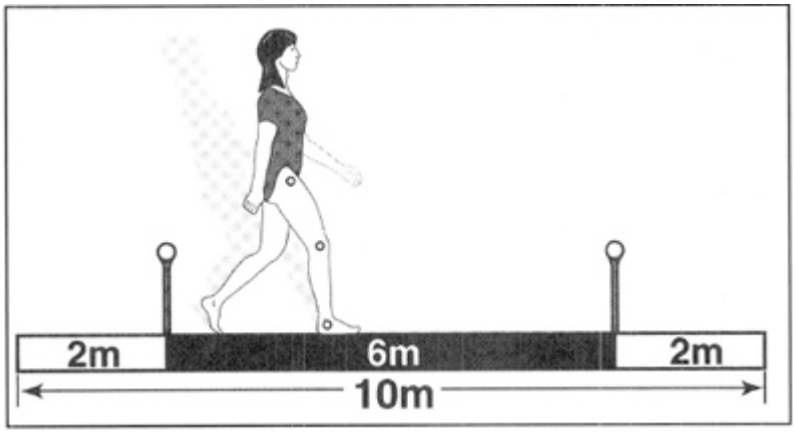

Figure 23-10. Gait recording walkway. Total length is 10 meters. Middle 6 meters used for data collection (black length). Photoelectric sensors (vertical objects) designate ends of data interval.

Gait analysis is an unfamiliar experience for most subjects (ie, persons with and without gait pathology). This was confirmed by the finding of greater variability in data between the first and second walks (7% error) compared to later trials. After the first walk, retest inconsistencies averaged 3%. Usually, the first walk was significantly slower. Hence, an unrecorded trial is indicated before any data are stored.

The signals from the footswitches may be transmitted by telemetry or cable to a strip chart recorder for a printed record or to a computer

for immediate processing. Another option is a portable system. The footswitch and time signals are collected by a microprocessor memory unit fastened to the waist band. After the test, a cable is used to transmit the data to the receiver where the stride characteristics are determined and a printed record provided.[24] The output is based on a testing distance of 6 meters. This latter design allows testing in any environment having a 10-meter walking distance available.

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Chapter 24

Energy Expenditure

Robert Waters, MD

INTRODUCTION

Walking is the most common form of exercise and provides the only means of physical exertion for many sedentary individuals. Limb movement requires energy for muscular contraction. Measurement of the metabolic energy expenditure provides global information on overall gait performance and a means of quantifying the overall physiologic penalty resulting from pathological gait.[40]

A number of investigators have performed physiological energy measurements on normal subjects and patients with gait disabilities while walking using a variety of methodologies and testing equipment. Consequently, it is often difficult to compare the results. For these reasons, the majority of the data presented in this chapter were obtained by the author in the Rancho Los Amigos National Rehabilitation Center (Rancho) Pathokinesiology Laboratory using the same test procedures.

WORK, ENERGY, AND POWER

In physics, work is the product of force times the distance through which the force acts. This definition can cause confusion in biological situations. For example, if a muscle exerts a force under isometric conditions, there is no mechanical work since the length of the muscle remains constant; however, metabolic energy is expended, and the subject experiences physiological effort.

Energy is the capacity to perform work. Energy involved in the production of work is called *kinetic energy*. Energy that is stored is potential energy. There are 6 different forms of energy: chemical, mechanical, heat, light, electrical, and nuclear. Transference of one form of energy to another is according to the law of conservation of energy. The law states that in the process of conversion, energy can neither be gained nor lost. The energy in food is biochemical energy and is converted by contracting muscles during movement to mechanical work and heat.[2]

Power is the term used to express the rate at which work is performed. Power is, therefore, a unit of time. If one man can lift a load over a given distance twice as fast as another, he is twice as powerful.

Efficiency in physiological exercise is defined as the percentage of energy input that is transformed into useful work. Studies performed in optimal situations where work is performed, such as walking on an inclined treadmill or cycling, generally demonstrate that humans can achieve an efficiency of 20% to 30%.[24] Since the law of conservation of energy states that energy cannot be lost, the metabolic energy that is not converted to mechanical work appears as heat energy, causing a rise in the body temperature.

The heat production of a maximally exercising subject may be 50 times the resting rate. Therefore, the physiological temperature-controlling mechanisms that transport heat from the muscles to the skin are extremely important to prevent hyperthermia.[2]

CALORIMETRY

The basic unit of heat energy is the gram-calorie (cal) or the kilogram-calorie (kcal). A gram-calorie is the amount of heat

necessary to raise the temperature of 1 gram of water 1°C.

Because of the law of conservation of energy, the amount of energy that is released by the complete metabolic degradation of food is the same as by its ignition and combustion with oxygen (O_2) in a bomb calorimeter. Approximately 4.82 kcal of heat is liberated when a typical diet of carbohydrate, fat, and protein is burned in 1 L of O_2.[24]

Physiological energy expenditure at rest or during exercise can be measured by determining the body's heat and work production. This method is called *direct calorimetry*. However, measurement of body heat by direct calorimetry is complex and impractical for most exercise laboratory situations.

Indirect calorimetry is a simpler method of determining energy expenditure and is the equivalent of direct calorimetry. Indirect calorimetry depends on the premise that the aerobic metabolic pathways are the principal method for generating adenosine triphosphate (ATP) during prolonged exercise. By measuring O_2 consumption, the energy expenditure is indirectly determined since the anaerobic contribution to energy production is small under this condition.

Energy Units

In most recent literature, O_2 gas volume in milliliters (mL) is reported without converting the data to calories. Since body size affects the amount of O_2 consumed, the volume of O_2 is divided by body weight to enable intersubject comparisons.

The volume of O_2 consumed in exercise studies is generally reported under standard conditions of temperature (0°C), pressure (760 mm Hg), and dryness (no water vapor).

The O_2 rate is the amount of O_2 consumed per minute (mL/kg•min). As will be discussed, the O_2 rate determines the intensity of sustained exercise and is related to the length of time exercise can be performed.

The O_2 cost (mL/kg•m) describes the amount of energy used to perform the task of walking. It indicates the amount of energy needed to walk a standard unit of distance (1 meter). The O_2 cost equals the O_2 rate divided by the speed of walking.

It is often useful to compare the O_2 cost of 2 individuals to determine their relative biological efficiency. One individual is more efficient than another if she has a lower O_2 cost. For this review, the definition of gait efficiency of pathological gait is as follows:

$$\text{Gait Efficiency} = \frac{100 \times O_2 \text{ cost (normal)}}{O_2 \text{ cost (patient)}}$$

Since the O_2 cost for a patient is nearly always greater than normal, the gait efficiency is less than 100%, depending on the degree of disability.

It is extremely important to clearly distinguish the difference between O_2 rate (mL/kg•min) and O_2 cost (mL/kg•m). The O_2 rate (mL/kg•min) indicates the intensity of physical effort during exercise and is a time-dependent parameter. The O_2 cost (mL/kg•m) is not time dependent. An individual may have a high O_2 cost, due to a gait disability, but a low O_2 rate. In this case, walking can be sustained for a prolonged time, since the low O_2 rate indicates the intensity of exercise is low. On the other hand, an individual with a gait disorder resulting in a high O_2 rate may only be capable of walking a short time before the onset of fatigue, regardless of the value of the O_2 cost.

ENERGY METABOLISM

Transfer of energy from the metabolism of foods is by way of biochemical reactions along the chains of the different metabolic pathways through chemical bonds. The final biochemical energy unit is ATP. When ATP is converted to adenosine diphosphate (ADP),

free energy is liberated, which can be transferred to other molecules.[24]

$$ATP \rightarrow ADP + P + Energy$$

This energy shortens the contractile elements in muscle.

The amount of ATP stored in the cells is small and can only sustain muscle contraction for several seconds. In muscle, a limited amount of energy for ATP synthesis can be supplied anaerobically by creatine phosphate (CP).

$$ADP + CP \rightarrow ATP + C$$

Although the quantity of CP in muscle is 3 to 5 times more than the amount of ATP, the majority of energy to reform ATP during sustained muscle activity is actively generated from other sources.

Aerobic Oxidation

During prolonged exercise, aerobic oxidation of carbohydrate and fat are the principal food sources for generating ATP. These substrates are oxidized through a series of enzymatic reactions leading to the production of ATP. The net equation for the aerobic metabolism of glucose ($C_6H_{12}O_6$) is as follows:

$$C_6H_{12}O_6 + 36ADP + 36P + 6O_2 \rightarrow 6CO_2\uparrow + 36ATP + 42H_2O$$

Similar equations summarize the aerobic oxidation of fats.[24]

Anaerobic Oxidation

A second type of oxidative reaction called the *glycolytic cycle* is available that does not require O_2. In this pathway, carbohydrates or

fats are converted to pyruvate and lactate. The net equation for the glycolytic metabolism of glucose is as follows:

$$\text{Glucose} + 2P + 2ADP \rightarrow 2\text{Lactate} + 2ATP$$

Bicarbonate ($NaHCO_3$) buffers lactate in the blood, leading to the formation of CO_2, which is exhaled in the expired air and summarized by the following reactions:

$$\text{Lactate} + NaHCO_3 \rightarrow \text{Na Lactate} + H_2O + CO_2 \uparrow$$

The utilization of either carbohydrate or fat in both the aerobic and anaerobic pathways is dependent upon the type of muscular work (ie, continuous, intermittent, brief, or prolonged, intensity of work in relation to muscle groups involved), the individual's training level, diet, and state of general health.

Aerobic Versus Anaerobic Metabolism

During continuous exercise, there is an interplay between the aerobic and anaerobic metabolic pathways that depends on the work load. The O_2 supply to the cell and the capacity of aerobic energy-producing mechanisms are usually sufficient to satisfy ATP requirements during mild or moderate exercise. During strenuous exercise, both anaerobic and aerobic oxidation processes occur and the serum lactate level rises, reflecting the additional anaerobic activity.

The amount of energy that can be produced anaerobically is limited. As reflected in the previous equations, approximately 18 times more energy is produced by the aerobic oxidation of carbohydrates than by anaerobic oxidation.[24] Anaerobic oxidation is also limited by the individual's tolerance to acidosis resulting from the accumulation of lactate. From a practical standpoint, the anaerobic pathway provides muscle with a method for supplying energy for sudden and short-term strenuous activity.

If exercise is performed at a constant rate at which the aerobic processes can supply the necessary ATP production, an individual can sustain exercise for a prolonged time without an easily definable point of exhaustion.[2] The serum lactate does not rise since the amount of anaerobic metabolism is minimal.

When exercise is performed at more strenuous work rates, the serum lactate rises, reflecting the contribution of anaerobic energy production required to meet the additional ATP demands. The point of onset of anaerobic metabolism is heralded by a rise in the serum lactate, a drop in the blood pH, and a rise in the ratio of expired carbon dioxide (CO_2) to inspired O_2.[24] Since the metabolic contribution of anaerobic pathways, as well as the individual's tolerance for acidosis is limited, the endurance time progressively shortens and fatigue ensues earlier as the intensity of the work load rises.

Respiratory Quotient and Respiratory Exchange Ratio

In the analysis of energy expenditure, the respiratory quotient (RQ), which is the ratio of CO_2 production to O_2 consumption at rest, relates to the type of food that is metabolized. Interpretation of the RQ rests on the assumption that analysis of air exchange in the lungs is the same as gas exchange at the cellular level and reflects the oxidation of specific food sources. The RQ for a pure carbohydrate diet is 1.00. The RQ for a pure fat diet is 0.70. A typical mixed diet consisting of 60% metabolized fats and 40% metabolized carbohydrates results in a RQ of 0.82 at rest and the caloric equivalent is 4.8 cal per mL O_2.[24]

The respiratory exchange ratio (RER) is calculated the same as the RQ, and this term is used under exercise conditions. Sustained strenuous exercise resulting in an RER greater than 0.90 indicates anaerobic activity.[2] A ratio greater than 1.00 indicates severe exercise. From a practical standpoint, the RER provides a

convenient, noninvasive method of determining whether significant anaerobic metabolism is occurring.

MAXIMAL AEROBIC CAPACITY

The maximal aerobic capacity (VO_2 max) is the highest O_2 uptake an individual can attain during exercise. It is a time-dependent parameter and is expressed in the same units as the O_2 rate (mL/kg•min). It represents an individual's maximal aerobic energy production capability and is the single best indicator of work capacity and physical fitness.[40] Generally, an individual is able to reach his or her VO_2 max within 2 to 3 minutes of exhausting exercise.

Age influences the VO_2 max. Up to approximately 20 years of age, the maximum O_2 uptake increases. Thereafter, the maximum O_2 uptake declines primarily due to a decrease in both maximum heart rate, stroke volume, and a more sedentary life-style.[1]

Differences in body composition and hemoglobin content are factors that account for a difference in the VO_2 max between the sexes. The ratio of VO_2[2] max to the fat-free body mass is not significantly different between men and women. However, due to generally larger body composition and greater hemoglobin concentration in men, and greater adipose tissue in women, the VO_2 max is 15% to 20% higher in males than females.[2]

Arm Versus Leg Exercise

The maximal aerobic capacity also depends on the type of exercise performed. The O_2 demand directly relates to the muscle mass involved. The VO_2, max during upper limb exercise is lower than with the lower limbs. For any given work load, however, heart rate and intra-arterial blood pressure are higher in upper limb exercise than lower limb exercise.[3] In the trained athlete, the VO_2 max is the same whether maximally running or bicycling.[3]

Deconditioning

A sedentary life-style decreases the VO_2 max.[32] Not only does atrophy of peripheral musculoskeletal structures occur, but there is a central decline in stroke volume and cardiac output and an increase in resting and exercising heart rate as a result of inactivity. Any disease process of the respiratory, cardiovascular, muscular, or metabolic systems that restricts the supply of O_2 to the cell will also decrease the VO_2 max. Bed rest for 3 weeks can result in a 27% decrease in the VO_2 max in normal subjects by decreasing cardiac output, stroke volume, and other factors.[32]

Training

A physical conditioning program can increase the aerobic capacity by several processes: improving cardiac output, increasing the capacity of the cells to extract O_2 from the blood, increasing the level of hemoglobin, and increasing the muscular mass (hypertrophy). All the above lead to increased fat utilization as the primary source of energy.[24] As a result, less lactate is formed during exercise and endurance is increased. Other effects of aerobic training include a decrease in resting and submaximal heart rate and blood pressure, and an increase in stroke volume and, therefore, cardiac output.

Endurance

If exercise is performed at a rate at which the aerobic processes can supply the necessary ATP production, an individual can sustain exercise for a prolonged time without an easily definable point of exhaustion.[2] When exercise is performed at more strenuous rates, anaerobic metabolism assists aerobic metabolism in meeting the demands of sustained strenuous exercise. The contribution of anaerobic metabolic pathways normally begins when the exercise work rate reaches between 55% and 65% of the VO_2 max of healthy

untrained subjects but can occur at over 80% of the VO_2 max in highly trained endurance athletes.[9,24] Anaerobic metabolism is heralded by a rise in the serum lactate and a rise in the RER.

It appears both the VO_2 max and the point at which anaerobic processes begin are determined by different factors. Training, muscle fiber type, capillary density, and alterations in the muscle's oxidative capabilities determine the percentage of the VO_2[2] max that can be sustained in endurance exercise without triggering anaerobic metabolism.[17,24] An experienced endurance athlete tends to compete at an exercise level just above the point of onset of blood lactate accumulation. Since the metabolic contribution of anaerobic pathways is limited, as well as the individual's tolerance for acidosis systemically and in the muscle, endurance progressively shortens and fatigue ensues earlier as the intensity of the work load rises.

Oxygen Pulse

Measurement of the VO_2 max requires the willingness of the subject to work to exhaustion and may be unsafe or undesirable for some subjects, particularly older individuals. When it is not possible or appropriate to directly measure the VO_2 max, considerable information on the level of physical conditioning is obtained by calculating the O_2 pulse.

The O_2 pulse is the ratio of the O_2 rate to the heart rate. In the absence of cardiac disease, there is a linear relation between the O_2 rate and heart rate, although there is considerable variation in the slope between individuals related to differences in physical conditioning, the muscles utilized, and whether exercise is performed with the arms or legs. It is desirable to compare the O_2 pulse when subjects are exercising at approximately the same heart rates. As with the VO_2 max, the O_2 pulse is higher during leg exercises than arm exercises.[3] Because training can increase the cardiac stroke volume and improve the efficiency of the extraction of O_2 from the

blood by muscle, the O_2 pulse increases with training. Conversely, the O_2 pulse decreases with deconditioning.

METABOLIC ENERGY MEASUREMENT

STEADY STATE

After approximately 2 or 3 minutes of exercise at a constant submaximal work load, the rate of O_2 consumption reaches a level sufficient to meet the energy demands of the tissues. The cardiac output, heart rate, respiratory rate, and other parameters of physiology work load reach a plateau and a steady state condition is achieved. Measurement of the rate of O_2 consumption at this time reflects the energy expended during the activity.

SPIROMETRY

O_2 uptake can be measured by either closed or open spirometry techniques. In closed systems, air is rebreathed after absorption of CO_2 in exhaled air in a lime canister. This method requires the subject to be adjacent to a large spirometer. Most closed systems have considerable airway resistance when conducting high volumes of air and therefore are not used in exercise studies, although they are commonly used for pulmonary function studies in hospitals.

Open spirometry is the preferred method for most exercise studies. In open spirometry, the subject does not rebreathe air, as in closed spirometry. Ambient air (O_2 20.93%, CO_2 0.03%, nitrogen 79.04%) is constantly inhaled. Measurement of the volume and the percentage of O_2 in the expired air is used to calculate the amount of O_2 consumption.

TEST PROCEDURES/TREADMILL or TRACK

Measurement of O_2 consumption in normal subjects can be performed on a treadmill with an adjacent O_2 measurement apparatus. This method requires the least amount of laboratory space and also enables continuous, breath-by-breath gas analysis.

Normal subjects easily adapt to walking on a treadmill or on a track. When asked to walk around a circular track at their comfortable speed, normal subjects and minimally disabled patients choose a self-selected velocity that remains relatively constant for a given individual over a sustained walking trial.[37,38,47] Because air resistance is minimal at functional walking velocities, there is no difference in the rate of O_2 consumption measured on a treadmill or on a track at the same speeds.[29]

Unlike normal subjects, patients with moderate or severe gait impairments may have difficulty adapting to a treadmill and as a result may walk more slowly than their CWS. Those requiring walking aids, such as crutches or a walker, may be unable to walk on a treadmill. Thus, treadmill data may not reflect the true energy expenditure under the patient's customary walking conditions. For these reasons and safety, we prefer testing on a stationary track.

Measurement of O_2 consumption can be performed with a portable spirometer carried by the subject. The volume of expired air is measured and a sample of gas collected for later O_2 and CO_2 analysis. Portable spirometers are most useful for situations requiring considerable freedom of body movement such as in skiing, cross-country running, or mountain climbing.

The Douglas Bag technique is the classic method of O_2 analysis. This method enables highly accurate gas analysis. Expired air is collected in a large portable bag over a fixed time interval. At the completion of testing, the volume of air is measured and tested for O_2 and CO_2 content on highly accurate bench apparatus.

To enable continuous gas analysis while subjects walked around a track, Corcoran developed a velocity-controlled, motor-driven, mobile cart carrying the gas measurement apparatus.[8] Since the gas

analysis apparatus is not carried by the subject, weight was not a factor and accurate instrumentation was used that enabled continuous data analysis.

PATHOKINESIOLOGY LABORATORY METHODS

The primary method of gas analysis that has been used in the Rancho Pathokinesiology Laboratory is a modification of the Douglas Bag technique. This selection was based on the simplicity, high reliability, and accuracy of this method. The system is harnessed to the subject's shoulders. A multiported valve enables multiple collected gas samples in nonporous polypropylene bags while the patient walks around a circular, 60.5-m, outdoor track.

The subject breathes through a well-fitted mouthpiece and wears a nose clip to prevent air leakage. The directional flow of inspired and expired air is controlled by 2 large diameter, one-way "J" valves mounted over each shoulder. The large diameter design of the J valve results in a highly competent valve (insignificant retrograde airflow) without adding significant airway resistance even at the high rates of airflow obtained during intense exercise. The 2 valves are mounted over each shoulder and connected by large diameter flexible tubing to a "T" piece attached to the mouthpiece.

It is desirable to minimize the "dead space" in the air collection system to prevent rebreathing since the inhalation of expired CO_2 in sufficient quantity causes hyperventilation. For this reason, a third valve is mounted inside the T piece. This valve does not add significant airflow resistance and is sufficiently competent to prevent CO_2 rebreathing.

A thermistor placed in the T piece just beyond the mouthpiece detects the difference in temperature of inspired and expired air, enabling monitoring of respiratory rate. Electrocardiographic leads are taped to the subject's chest to record the heart rate. A foot switch worn inside the shoe detects step frequency. Heart rate, respiratory rate, and step frequency data are telemetered via a radio transmitter. The total weight of the entire system is less than 1.5 kg.

Currently, the Pathokinesiology Laboratory uses a breath-by-breath analysis system. The lightweight, portable unit records and stores data acquired over extended periods of time. At the completion of a study, the data can be downloaded to a computer for analysis. Alternatively, use of the telemetry feature enables real time analysis of the energy cost data.

RESTING AND STANDING METABOLISM

The basal metabolic rate (BMR) is the minimum level of energy required to sustain the body's vital functions in the waking, resting state.[24] The BMR generally varies according to the diet, body surface area, as well as the percentage of body fat, and this in part accounts for a 5% to 10% difference between females and males. As a function of age, the BMR decreases approximately 2% every decade through adulthood.[24]

In the recumbent position, the basal metabolic rate and resting values of O_2 consumption are approximately the same.[11] O_2 uptake in the sitting position is slightly increased.[26] Quiet standing further elevates the rate of O_2 consumption by approximately 22%, equaling 3.5 mL/kg•min for males and 3.3 mL/kg•min for females.

Electromyographic studies demonstrate minimal muscular activity is required for normal standing.[21] This is consistent with the fact that in the standing posture, the force of gravity acting on the COG of the different body segments passes close to the axes of rotation of the spine, hip, knee, and ankle. Only the calf muscles support the dorsiflexed ankle.[21] This is an example of the principle of energy conservation applied to the design of the musculoskeletal frame and the standing posture.

NORMAL GAIT

Range of Customary Speeds

In a study of adult pedestrians 20 to 60 years of age who were unaware they were observed, the mean walking speed for males (82 m/min) was significantly higher than for the females (74 m/min).[13] Approximately the same mean values, 82 m/min and 78 m/min, were obtained during energy expenditure studies performed in the Pathokinesiology Laboratory around an outdoor, circular track when subjects were instructed to walk at their CWS (Table 24-1).[41] These findings support the conclusion that the gait of normal subjects tested in this manner was not altered by the experimental procedure.

The average slow and fast walking speeds (FWSs) in adults 20 to 59 years of age were 37 m/min and 99 m/min (Table 24-2).[41] It can be concluded that the functional range of walking speeds in adults varies from approximately 40 to 100 m/min.

At speeds above 100 m/min, there is a choice between walking or running. Thorstensson and Roberthson studied adult males and found the transition speed between walking and running averaged 113 m/min with a tendency for longer-legged men to have a higher transition speed.[34] Running becomes more efficient than walking at speeds above approximately 133 m/min.[12]

Table 24-1

Gait Characteristics of Unobserved Adult Pedestrians and Adult Subjects 20 to 60 Years of Age Undergoing Energy Expenditure Testing at Their Customary Walking Speed

	Finley[13]			*Waters*[41]		
	Male	Female	Both	Male	Female	Both
Velocity (m/min)	82	74	78	82	78	80
Cadence (steps/min)	110	116	114	108	118	113
Stride (m)	1.48	1.32	1.38	1.51	1.32	1.42

Table 24-2

Gait Characteristics at Customary Slow, Normal, and Fast Speeds for Adults (20 to 59 Years Old)

	Velocity (m/min)			*Cadence (steps/min)*			*Stride (m)*		
	SWS	*CWS*	*FWS*	*SWS*	*CWS*	*FWS*	*SWS*	*CWS*	*FWS*
Female	37	78	99*	68	118	137	0.89	1.32	1.24
Male	48	82	110*	76	108	125	1.03	1.51	1.67
All	43	80	106	72	113	131	0.97	1.42	1.47

* Indicates significant ($P < .05$) difference between male and female subjects at slow walking speed (SWS), customary walking speed (CWS), and fast walking speed (FWS).

Adapted from Waters RL, Lunsford BR, Perry J, Byrd R. Energy-speed relationship of walking: standard tables. *J Orthop Res.* 1988;6(2):215-222.

Energy Expenditure at the Customary Walking Speed

At the CWS, the O_2 rates for young adults aged 20 to 59 years and senior subjects between 60 and 80 years of age do not significantly differ, averaging 12.1 and 12.0 ml/kg•min.[41] The O_2 rate is higher in teens and children, averaging 12.9 and 15.3 ml/kg•min, respectively (Table 24-3). Expressed as a percentage of the VO_2 max, the O_2 rate at the CWS requires approximately 32% of the VO_2 max of an untrained normal subject 20 to 30 years of age and nearly 48% of the VO_2 max of a senior subject 75 years of age.[1] The RER was below 0.85 for normal subjects of all ages at their CWS, indicating anaerobic metabolism is not required.

Table 24-3

Energy Expenditure at Comfortable and Fast Walking Speeds: The Influence of Age

	Speed m/min		*O_2 Rate mL/kg•min*		*O_2 Cost mg/kg•m*		*Pulse beats/min*		*RER*	
	CWS	FWS	CWS	FWS	CWS	FW	CWS	FWS	CWS	FWS
Children (6 to 12)	70	88	15.3	19.6	0.22	0.22	114	127	0.84	0.87
Teens (13 to 19)	73‡	99	12.9‡	19.2	0.18	0.20	97	117	0.76	0.82
Adults (20 to 59)	80‡	106‡	12.1‡	18.4‡	0.15‡	0.17	99	124‡	0.81	0.92
Seniors (60 to 80)	74‡	90‡	12.0	15.4‡	0.16‡	0.17	103	119‡	0.85	0.92

‡ Indicates significant ($P < .05$) difference between preceding value in younger age group.

Adapted from Waters RL, Lunsford BR, Perry J, Byrd R. Energy-speed relationship of walking: standard tables. *J Orthop Res.* 1988;6(2):215-222.

The results account for the healthy subject's perception that walking requires little effort. It is significant that with advancing years older individuals have progressively smaller aerobic reserves due to a decline in the VO_2 max to accommodate to any added physiologic penalties imposed by gait disorders.

ENERGY EXPENDITURE AT THE FAST WALKING SPEED

When asked to ambulate at a FWS, the average O_2 rate for children, teens, and young adults is approximately the same, averaging 19.6, 19.2, and 18.4 ml/kg•min, respectively.[41] The value for senior subjects at their FWS, 15.0 ml/kg•min, is significantly lower and corresponds to a decline in the average FWS (90 m/min versus 106 m/min). The decline in the average FWS of senior subjects parallels the decrease in the average VO_2 max that occurs with aging. The RER for children, teens, young adults, and seniors at

their fast speed average 0.87, 0.82, 0.92 and 0.92, respectively.[41] These findings indicate that normal adults customarily set their FWS at a level approximating the threshold of anaerobic metabolism.

Males Versus Females

Investigators have reported higher rates of O_2 consumption in males while walking. Others have reported higher values in female subjects or no significant difference.[5] In a review of 225 normal subjects, we found no significant differences due to sex were observed at the customary slow, normal, or fast speeds.[41] The heart rate was higher in females than males in all age groups, consistent with other types of exercise in which higher heart rates are observed in females.[41]

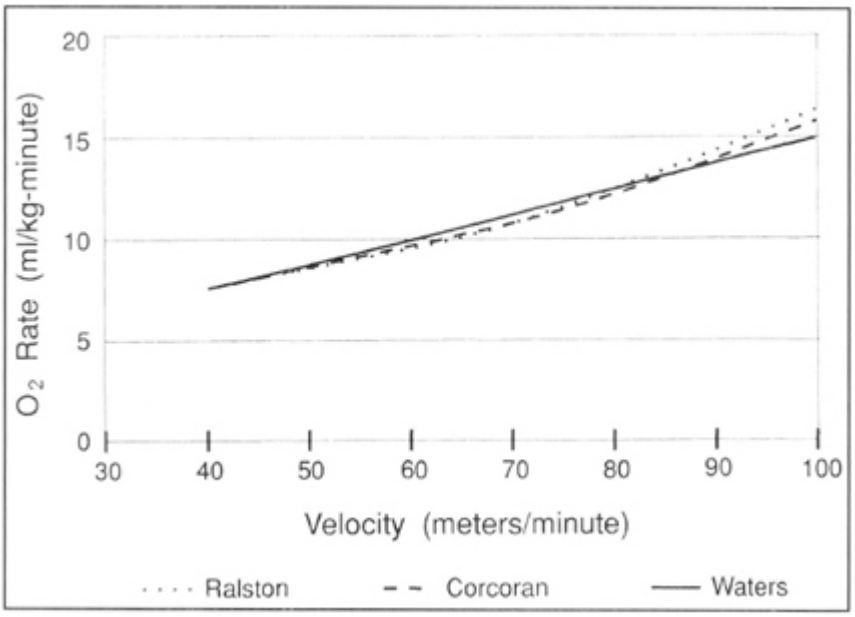

Figure 24-1. Rate of O_2 consumption-speed relationship in 3 different studies (references 8 and 30 and unpublished author data). This relationship is approximately linear in the range of functional walking speeds between 40 and 100 m/min.

Energy-Speed Relationship

Different investigators have derived second-order equations to describe the energy-speed relationship, which are typified by the following:

$$O_2 \text{ Rate} = 0.00110\ V2 + 5.9 \text{ (Ralston)}^{30} \textbf{ Eq (1)}$$
$$O_2 \text{ Rate} = 0.00100\ V2 + 6.2 \text{ (Corcoran and Gelmann)}^{8} \textbf{ Eq (2)}$$

where the O_2 rate is the mL/kg•min of O_2 and V equals the velocity.

Inspection of these equations indicates they are approximately linear within the customary range of walking speeds from 40 to 100 m/min. We found second- or higher-order regressions did not significantly improve the data fit in comparison to linear regression at velocities below 100 m/min. Since the functional range of walking speeds is below 100 m/min, we prefer the following linear regression to describe the energy-speed relationship[41]:

$$O_2 \text{ Rate} = 0.129\ V + 2.60 \textbf{ Eq (3)}$$

Comparison of Eq (1) through Eq (3) indicates all generate similar values in the customary range of walking speeds from 40 to 100 m/min (Figure 24-1). Ralston, Eq (1), performed testing on a treadmill at controlled velocities. Corcoran and Gelmann, Eq (2), performed testing on a track while subjects walked along a velocity-controlled mobile cart carrying the gas analysis instrumentation. Our results, Eq (3), were obtained on an outdoor track at the subjects' self-selected, comfortable, slow, and FWSs. The equivalency of the equations for the energy-speed relation by 3 different methods indicates the adaptability of normal gait to different laboratory conditions without the introduction of experimental artifact.

The above equations were not determined at extremely slow velocities. Measurement of the O_2 rate at extremely slow speeds in adults resulted in an average of 5.7 mL/kg•min.[20] This value represents the effort required to maintain the body in motion at a barely perceptible speed.

The regression equations for the energy-speed relationship in adults 20 to 59 years of age and seniors 60 to 79 years of age are

the same. However, the regression equations for children and teens significantly differ from adult values as follows:

$$\text{Children: } O_2 \text{ Rate} = 0.188\ V + 2.61 \quad \textbf{Eq (4)}$$

$$\text{Teens: } O_2 \text{ Rate} = 0.147\ V + 2.68 \quad \textbf{Eq (5)}$$

$$\text{Adults: } O_2 \text{ Rate} = 0.129\ V + 2.60 \quad \textbf{Eq (6)}$$

Interestingly, the Y-intercept of the regression equations are essentially the same in all 3 groups and approximate the value for quiet standing (zero velocity).

Inherent differences in body composition and size account for the higher rate of O_2 consumption in children. The lean muscle mass in children comprises substantially greater percentage of total body weight than in adults. With increasing age, a larger percentage of total body weight becomes fat and skeleton, which are relatively metabolically inert.

Oxygen Cost-Speed Relation

The O_2 cost per meter walked is obtained by dividing the rate of O_2 uptake by the velocity. The equation for O_2 cost at different speeds can be derived by dividing Eq (4 to 6) by the velocity, yielding the following equations:

$$\text{Children: } O_2 \text{ Cost} = 0.188 + 2.61\ V^{-1} \quad \textbf{Eq (7)}$$

$$\text{Teens: } O_2 \text{ Cost} = 0.147 + 1.68\ V^{-1} \quad \textbf{Eq (8)}$$

$$\text{Adults: } O_2 \text{ Cost} = 0.129 + 2.60\ V^{-1} \quad \textbf{Eq (9)}$$

It is evident that children are the least efficient walkers.

Walking Surface and Footwear

The type of walking surface has little effect on energy expenditure unless the surface is extremely rough. A 10% decrease in energy

cost was found between treadmill and asphalt or cinder path walking for a young normal adult population when wearing leather combat boots.[26]

LOADING

Loading the body with weights increases the rate of energy expenditure depending on the location of the loads. Loads placed peripherally on the foot have a much greater effect than loads placed over the trunk.[20] Placement of a 20-kg load on the trunk of a male subject did not result in a measurable increase in rate of energy expenditure. On the other hand, a 2-kg load placed on each foot increased the rate of O_2 uptake 30%. This finding is predictable since forward foot acceleration is much greater than trunk acceleration and, therefore, greater effort is required. These findings are of clinical significance for patients requiring lower extremity orthoses or prostheses and indicate the importance of minimizing weight.[43]

GRADE WALKING

A number of investigators have studied walking up an incline. Bobbert combined his own data with those obtained from the literature and determined the logarithm of the O_2 rate increased linearly with the slope of the grade.[4]

Table 24-4

Energy Expenditure Following Hip Fusion, Ankle Fusion, and Knee Immobilization

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Gait Efficiency Percent*
Ankle fusion	67	12.0	0.17	92
Knee immobilization	64	12.7	0.20	76
Hip fusion	67	14.7	0.22	68

PATHOLOGICAL GAIT

It is evident that energy conservation was a major factor in the evolutionary design of the lower limbs and bipedal gait mechanism. Interruption of the normal GC due to significant lower extremity impairment results in increased energy expenditure. Nevertheless, a patient will adapt if there is sufficient neurological control. Compensatory gait substitutions represent one mechanism to minimize the additional energy expenditure.[20] Persons with greater deficits will require arm assistance (swing-through or reciprocal gait) or orthoses to substitute for inadequate muscle control. Two forms of arm-assisted gait can increase the energy cost. Swing-through crutch-assisted gait requires intense physical effort in comparison to normal walking. In the swing phase, the arms and shoulder girdle musculature must lift and swing the entire body forward for each step. Reciprocal gait is less demanding on the shoulder and preserves partial weight-bearing support through the legs, but the velocity is slower. Orthoses provide individual joint stabilization when muscles are unable to meet the challenge. The mechanism of compensating varies with the diagnosis and severity of disability.

JOINT IMMOBILIZATION

Measurement of the energy expenditure after joint immobilization by surgical joint fusion or plaster casts identifies the energetic importance of specific lower limb joint movements to the GC. The joints most commonly studied include the ankle, hip, and knee.

ANKLE FUSION

Following ankle fusion, the average O_2 rate, 12.0 ml/kg•min, is approximately the same as for normal subjects, 12.1 mL/kg•min, but the CWS, 67 m/min, is 16% slower or 84% of normal (80 m/min) (Table 24-4).[37] Using Eq (3) to control for velocity, the average ankle fusion patient requires an O_2 rate 3% greater than a normal subject walking at the same speed.

Due to the slow speed, the average O_2 cost for ankle fusion patients is 0.166 mL/kg•m in comparison to 0.151 mL/kg•m for normal adults.[37] Therefore, gait efficiency is 92% of normal. The fact that there is only an 8% decrease in gait efficiency following ankle fusion is consistent with the observation that ankle fusion does not require major compensatory changes in the overall gait pattern.

HIP FUSION

Following unilateral hip fusion, the average CWS is below normal, 67 m/min. This is the same as the mean following ankle fusion (see Table 24-4).[37] Although there is no notable difference in the CWS, the average O_2 rate for hip fusion patients, 14.7 mL/kg•min, is significantly higher than normal or following ankle fusion. Using Eq (3) to control for velocity, this increase represents a 32% higher rate than for normal subjects.

Because of both the reduced CWS and higher O_2 rate, the average O_2 cost for hip fusion patients, 0.223 mL/kg•m, is

significantly greater than following ankle fusion, 0.170 mL/kg•m. In comparison to normal walking, hip fusion patients achieve 68% gait efficiency. The elevated O_2 rate and O_2 cost indicate the importance of hip motion in the GC.

Knee Immobilization

The importance of knee motion to the GC can be determined by applying a cylinder cast to the leg that prevents knee motion without interfering with ankle or hip motion.[39] Interestingly, the values for the O_2 rate, 12.7 mL/kg•min, and the O_2 cost per meter, 0.200 mL/kg•m following cylinder cast immobilization are intermediary between the values for ankle fusion and hip fusion patients.[37] Gait efficiency is 76% of normal values. In summary, the loss of motion at the ankle, hip, or knee increases the O_2 rate and O_2 cost with a greater change occurring at each more proximal joint.

FRACTURE

A high O_2 rate (averaging 15.7 mL/kg•min) and heart rate (153 bpm) result from swing-through gait in fracture patients at their CWS. The elevated RER (1.08) signifies that the energy demand of swing-through crutch ambulation cannot be sustained by aerobic energy supply alone and requires exercise under anaerobic conditions (Table 24-5).

The high O_2 rate and heart rate observed in fracture patients account for the common clinical experience that older patients who are unable to weight bear on an injured leg are commonly severely restricted ambulators. Because of the decreased strength and VO_2 max associated with age, many are unable to meet the intense energy demand and prefer a wheelchair. Conditioning programs to improve the exercise capacity are indicated when a swing-through crutch-assisted gait pattern is required of a sedentary or older patient. Weight bearing should be allowed on the injured limb at the

earliest possible time consistent with adequate healing to lessen energy expenditure.

Table 24-5

Energy Expenditure of Swing-Through Crutch Walking

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*	*RER*
Fracture[38]	50	15.7	0.32	153	1.08
Paraplegia[42]	29	16.3	0.88	140	--

SPINAL CORD INJURY

Individuals with paraplegia using a swing-through gait typically require bilateral KAFOs in the absence of sufficient quadriceps strength to stabilize the knees. Not only must the arms lift and swing the body forward in the swing phase, but they must also provide antigravity support during the stance phase if the hip and trunk extensors are paralyzed. People with paraplegia may also lack motor control of lower trunk and hip flexors, further concentrating the demand on the shoulder and arm musculature to swing the body forward.

For the above reasons, it is not surprising that people with paraplegia with intact trunk musculature who require a swing-through gait are extremely slow ambulators. As a group, their CWS, 29 m/min, is approximately half the value of the fracture patients, 50 m/min. Despite the slow CWS, people with paraplegia have a high O_2 rate, 16.3 mL/kg•min, and O_2 cost, 0.88 mL/kg•m. The high O_2 rate is particularly significant as the VO_2 max for those with paraplegia is lower than normal.[16] These findings account for the fact that few individuals with paraplegia who are dependent on a swing-through gait pattern continue to walk after gait training in a rehabilitation center.

Approximately half of the patients with spinal cord injuries have incomplete lesions. Patients with incomplete paraplegia have the potential to reinforce their residual muscle function with orthoses and to weight bear through their legs. This reduces the demand on their arms, making a reciprocal gait pattern a possibility.

Reciprocal Gait

Hussey and Stauffer concluded that there is a direct relationship between motor power, the reciprocal gait, and walking ability.[19] Those patients who were able to walk in the community had a reciprocal gait pattern and at least fair (grade 3) hip flexor strength and fair extensor strength in one knee so that no more that one KAFO was required. With respect to motor strength, the principal indication for the use of a KAFO is knee instability due to quadriceps paresis. The most common indication for the use of an AFO is ankle instability due to weakness in the plantar flexors or dorsiflexors.[46]

Ambulatory Motor Index

The Ambulatory Motor Index (AMI) was developed to define the relationship between motor paralysis and walking ability. The AMI quantitates the extent of paralysis and its relationship to the physiological indices of energy expenditure and gait performance.[45] It is based on a 4-grade scale derived from the standard 6-grade manual motor scale. The strength of key lower extremity muscles is first determined using the standard 6-grade scale (absent = 0; trace, visible, or palpable contraction = 1; poor, active movement through ROM with gravity eliminated = 2; fair, active movement through ROM against gravity = 3; good, active movement through ROM against gravity and resistance = 4; and normal = 5). In terms of the amount of strength needed to walk, there is no significant difference between a grade of trace or poor, since there is little difference in the amount of force generated between these muscle grades. Similarly, from a functional stand-point, a grade of good is sufficient to meet the

demands of level walking. Therefore, the grades trace and poor are combined into a single group as were the grades good and normal, yielding a 4-point scale (0 = absent; 1 = trace or poor, 2 = fair; 3 = good or normal).

The AMI is calculated by adding the bilateral motor scores for hip flexion, hip abduction, hip extension, knee extension, and knee flexion using the abridged scale discussed above. The sum of these scores was expressed as a percentage of the maximum possible score (30 points). The AMI is intended for use in patients with intact trunk and pelvic strength sufficient to stabilize the trunk.

Table 24-6

Ambulatory Motor Index

	AMI ≤40	*AMI >40, <60*	*AMI ≥60*
O_2 rate (mL/kg•min)	17.4	14.2	14.4
RER	0.80	0.89	0.85
Heart rate (bpm)	132	123	106
O_2 rate increase (% norm)	216	112	49
O_2 cost (mL/kg•m)	0.98	0.50	0.26
Velocity (m/min)	26.8	34.0	56.3
PAL (%BW)	43.1	28.3	6.3

Adapted from Waters RL, Yakura JS, Adkins R, Barnes G. Determinants of gait performance following spinal cord injury. *Arch Phys Med Rehabil.* 1989;70:811-818.

Ambulatory Motor Index and Gait Performance

The AMI in patients meeting Hussey and Stauffer's criteria for community ambulation is greater or equal to 60% of normal strength.[47] The mean walking CWS is 56 m/min, mean heart rate 106 bpm, average O_2 rate 14.4 mL/kg•min, and average O_2 cost 0.26 ml/kg•m (Table 24-6). In contrast, the majority of patients with an AMI of less than 40% required 2 KAFOs to ambulate. Their mean CWS is only 27 m/min, mean heart rate 132 bpm, O_2 rate 17.4 ml/kg•min, and average O_2 cost 0.98 ml/kg•m.

Since ambulation for routine community activities such as shopping may require an individual to walk distances more than 250 meters,[22] the average patient in this group would need to walk at least 10 minutes. At this intense exercise rate, he or she would arrive at his or her destination in a state of tachycardia (rapid heart rate), hyperhidrosis (sweating), and tachypnea (rapid respiratory rate). On the other hand, wheelchair propulsion on a level surface requires a much lower rate of energy expenditure, which is comparable to normal walking.

In a previous study, the average velocity of wheeling for spinal cord injury (SCI) patients was 72 m/min, the heart rate 123 beats/min, and the O_2 rate 14.5 ml/kg•m.[42] Clearly, the high rate of physiological energy expenditure of walking compared to wheeling is the reason why many patients with severe paralysis discontinue walking as a primary means of mobility. The difference between long-term wheelchair ambulatory patients is due to the differences in physiological demands of walking, and this physiological difference is due to the degree of lower extremity paralysis.

Ambulatory Motor Index and Peak Axial Load

In SCI patients who can reciprocally ambulate, the relationships between O_2 rate, O_2 cost, and speed are complex since the amount of force applied by the upper extremities to crutches can widely vary depending on the amount of paralysis and need for upper extremity gait assistance. To determine these relationships, the peak axial load (PAL) applied to the patient's upper extremity assistive devices was measured.[47]

As might be anticipated, there is a strong relationship between the PAL exerted by the arms on crutches, canes, and/or a walker and the AMI, which is defined as follows:

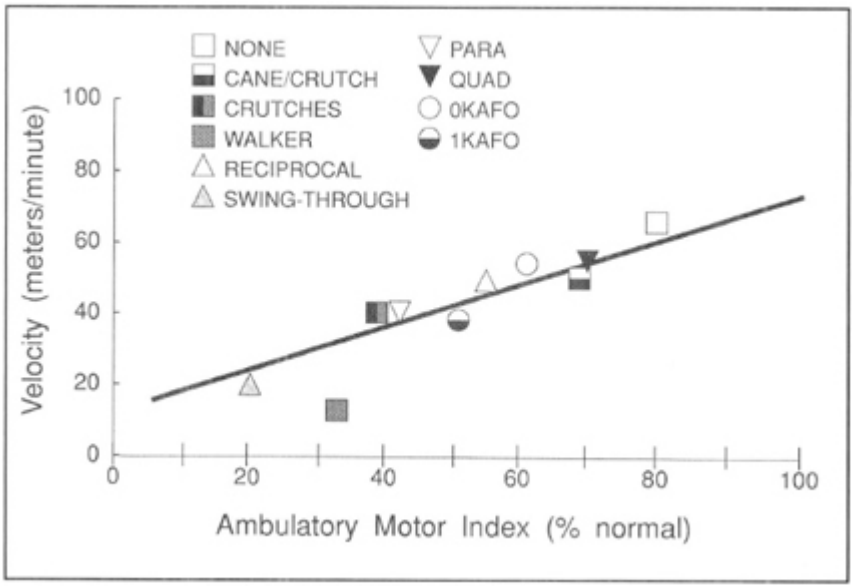

Figure 24-2. The PAL exerted on the different assistive devices was linearly related to the AMI. PAL = 82.75 – (1.72 x AMI) + (0.009 x AMI^2), [R = 0.73, p < 0.0001].

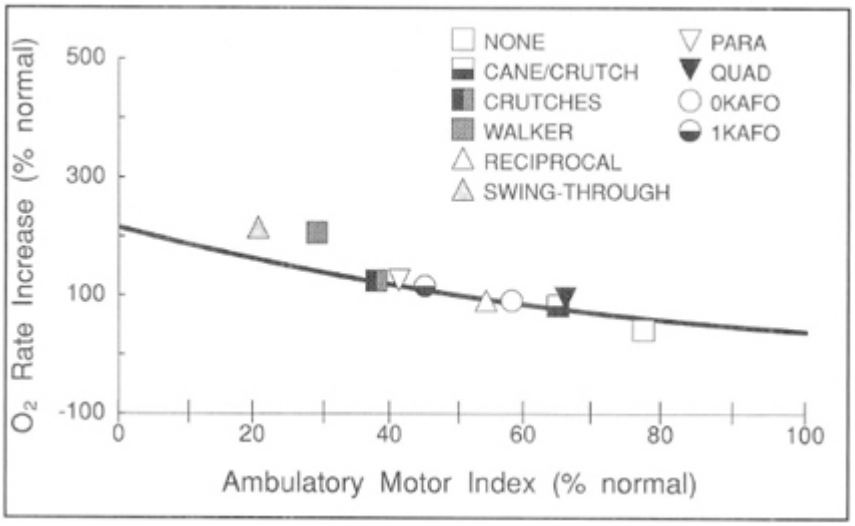

Figure 24-3. The PAL was directly related to the O_2 rate increase. O_2 rate increase + 27.1 + (3.63 x PAL), [R = 0.91, p < 0.0001].

$$PAL = 82.75 – (1.72 \times AMI) + (0.009 \times AMI^2)$$

where the PAL is expressed as a percent of total body weight (Figure 24-2).

In addition, the PAL is very strongly related to the O_2 rate increase.

$$O_2 \text{ rate increase} + 27.1 + (3.63 \times PAL)$$

It is evident that with diminished lower limb strength, increased upper arm exertion is required and is responsible for the added rate of physiologic energy expenditure (Figures 24-2 and 24-3).

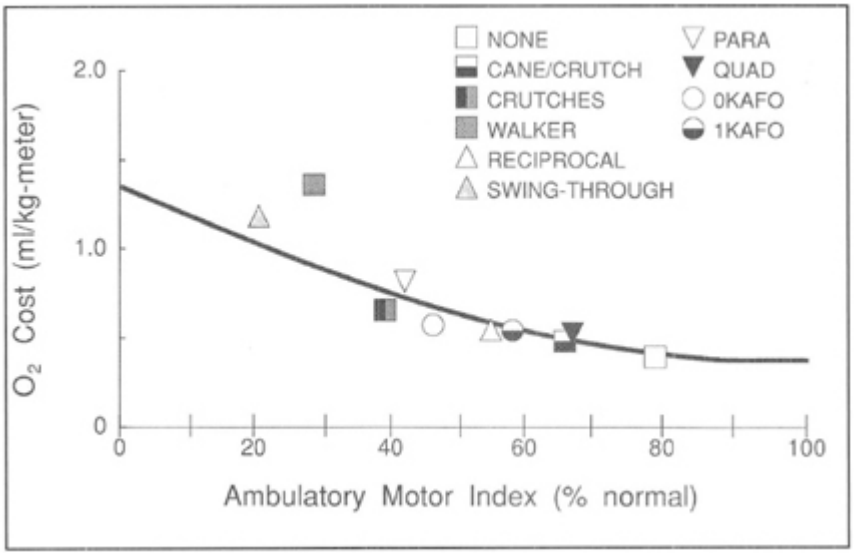

Figure 24-4. Velocity was strongly linearly related to the AMI. Velocity = 8.6 + (0.62 x AMI), [R = 0.73, $p < 0.001$].

Ambulatory Motor Index and Energy Expenditure

A comparison of the AMI to the gait velocity reveals a close linear relationship described by the following equation:

$$\text{Velocity} = 8.6 + (0.62 \times AMI)$$

where velocity is in m/min (Figure 24-4).

There are large differences in walking speed among patients, and these occur because of differences in the amount of paralysis. To control for velocity, the O_2 rate for normal walking can be calculated using Eq (3) and subtracted from the patient's value. There is a close relationship between the AMI and the O_2 rate increase characterized by the following equation:

$$O_2 \text{ rate increase} = 257.5 - (2.82 \times AMI)$$

where the O_2 rate increase is the percent increase in the rate of O_2 consumption per minute in the SCI patient, in comparison to the value for a normal subject walking at the same speed (Figure 24-5).

The O_2 cost per meter is also strongly related to the AMI (Figure 24-6). This relationship is defined by the following second-order regression equation:

$$O_2 \text{ cost} = 1.39 - (0.027 \times \text{AMI}) + (0.00015 \times \text{AMI}^2)$$

Orthotic Requirement

A decrease in AMI is related to a need for more extensive KAFO support. This orthotic management can be classified as no KAFO, one KAFO, or 2 KAFOs. The corresponding AMIs at each level of orthotic management are 58%, 47%, and 31%, respectively. An increased need for orthotic support is also associated with reduced velocity. With no orthoses, the average walking velocity was 48 m/min; with one orthosis, 37 m/min; and with 2 orthoses, 19 m/min. Greater orthotic requirement was also associated with an increased O_2 rate (81%, 107%, and 226%, respectively), O_2 cost (0.37 ml/kg•m, 0.46 ml/kg•m, and 1.15 ml/kg•m, respectively), and PAL (13.9%, 20.4%, and 79.0%, respectively). These data are summarized in Table 24-7.

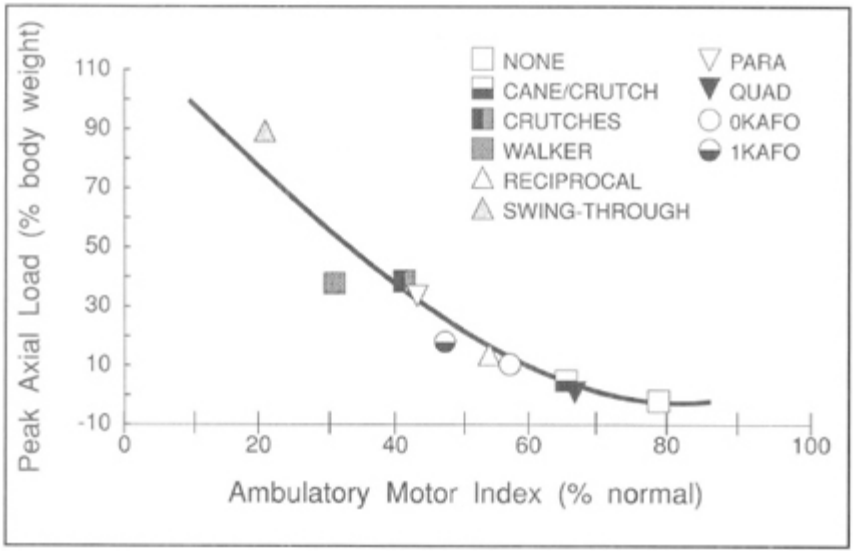

Figure 24-5. The O_2 rate increase was linearly related to the AMI. O_2 rate increase = 257.5 – (2.82 x AMI), [R = 0.68, p < 0.001].

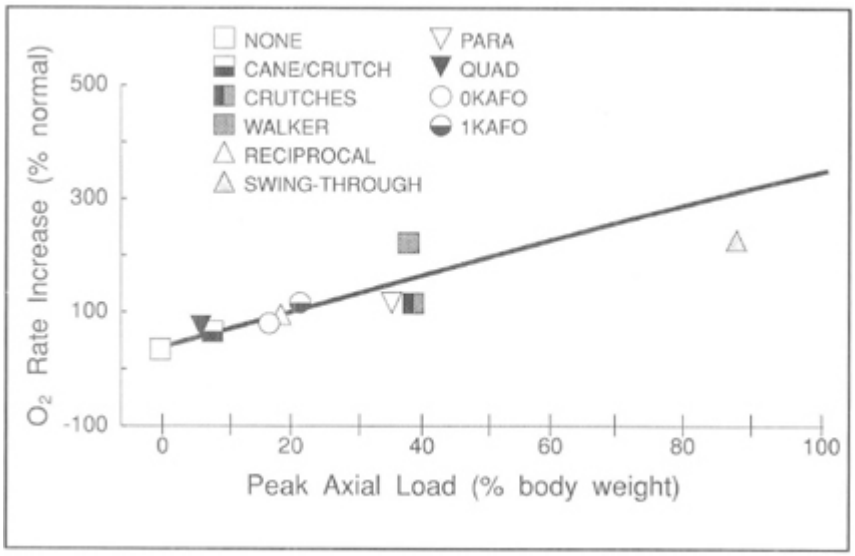

Figure 24-6. The O_2 cost was linearly related to the AMI. O_2 cost = 1.39 – (0.027 x AMI) + (0.00015 x AMI^2), [R = 0.77, p < 0.0001].

Assistive Devices

Subdividing SCI patients according to the need for upper extremity assistive devices also relates to the parameters of gait performance and energy expenditure. Between 4 groups (none, one cane or crutch, 2 crutches, and walker), there are significant differences in the O_2 rate increase, O_2 cost, walking speed, PAL, and AMI (Table 24-8). In general, these measures were consistent with the need for upper extremity assistance. In the 4 groups, the O_2 rate increase averaged 29%, 64%, 130%, and 210%; the O_2 cost averaged 0.22 ml/kg•m, 0.29 ml/kg•m, 0.56 ml/kg•m, and 1.20 ml/kg•m; the velocity 66 m/min, 48 m/min, 38 m/min, and 12 m/min; the PAL 0%, 7.0%, 30.8%, and 39.2%; and the AMI 79%, 68%, 44%, and 34%. These findings emphasize the high demand imposed on the upper extremities when walking requires bilateral crutches or a walker.

Table 24-7

Spinal Cord Injury Orthotic Requirement

	0 KAFO	*1 KAFO*	*2 KAFO*
O_2 rate (mL/kg•min)	15.1	14.7	14.9
RER	0.86	0.88	0.82
Heart rate (bpm)	115	125	122
O_2 rate increase (% norm)	81	107	226
O_2 cost (mL/kg•m)	0.37	0.46	1.15
Velocity (m/min)	48.1	37.1	18.9
PAL (% BW)	13.9	20.4	79.0
AMI (% norm)	58	47	31

Table 24-8

Upper Extremity Assistive Devices

	None	*Cane/Crutch*	*Crutches*	*Walker*
O_2 rate (mL/kg•min)	14.2	14.2	15.7	12.7
RER	0.82	0.87	0.87	0.76
Heart rate (bpm)	106	103	126	120
O_2 rate increase (% norm)	29	64	130	210
O_2 cost (mL/kg•m)	0.22	0.29	0.56	1.20
Velocity (m/min)	66.5	17.9	37.8	11.8
PAL (% BW)	-	7.0	30.8	39.2
AMI (% norm)	79	68	44	34

LEVEL OF SPINAL CORD INJURY

Individuals with tetraplegia who are able to walk have incomplete neurological lesions. Their average AMI is higher than for individuals with paraplegia (69% versus 44%). This is because persons with tetraplegia have varying degrees of upper extremity paralysis and are less capable of utilizing upper extremity assistive devices than individuals with paraplegia having normal upper extremities. As a consequence, the people with tetraplegia as a group require relatively greater preservation of lower extremity musculature to walk

than the typical person with paraplegia. Thus, they demonstrate a significantly lower mean PAL (5% versus 29% of body weight) (Table 24-9).

Table 24-9

Influence of Level of Spinal Cord Injury on Select Measures During Walking

	Level of SCI	
	Paraplegia	Tetraplegia
O_2 rate (mL/kg•min)	15.3	14.4
RER	0.87	0.83
Heart rate (bpm)	123	109
O_2 rate increase (% normal)	133	66
O_2 cost (mL/kg•m)	0.16	0.32
Velocity (m/min)	35.6	52.0
PAL (%BW)	28.6	4.8
AMI (% normal)	44	69

LONG-TERM OUTCOME

The energy expenditure was compared at discharge from rehabilitation and at 1 year follow-up using the AMI to assess motor strength.[49] At follow-up, patients walked faster, at a lower O_2 cost, had slower heart rates, and required decreased axial load on upper extremity assistive devices. Improvement was attributable to increased neurological recovery and/or physical conditioning. Patients with relatively weaker lower extremities at initial and follow-up testing demonstrated a larger conditioning effect and increased O_2 pulse. Those patients with relatively weaker lower extremities will have larger conditioning effects if ambulation is continued due to increased stress on the cardiovascular system by the demands of gait assistive devices.

MYELODYSPLASIA

The child with myelodysplasia has a pattern of motor paralysis that parallels traumatic SCI. There is a correlation between the orthotic requirement and the measures of energy expenditure if there is no associated neurologic impairment above the level of the spinal lesion due to hydrocephalus, Arnold-Chiari malformation, or instability of the spine or hip. In general, the orthotic requirement depends on the strength deficits in the quadriceps, ankle dorsiflexors, and plantar flexors. To provide adequate knee and ankle stability, there are 4 levels of orthotic management: no orthosis, AFO(s), one KAFO, and 2 KAFOs.

SWING-THROUGH

Swing-through ambulation was associated with elevated heart rates greater than 140 bpm in all patient groups requiring orthotic support. Speed was slowest in patients requiring 2 KAFOs, 22 m/min. The O_2 cost progressively rose as the need for orthotic support increased (no orthoses, 0.29 ml/kg•m; AFOs, 0.41 mL/kg•m; one KAFO, 0.41 mL/kg•m; and 2 KAFOs, 0.77 mL/kg•m; Table 24-10).

Table 24-10

Myelodysplasia Swing-Through Gait

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
0-Orthosis	47	13.8	0.29	120
AFO(s)	41	15.6	0.41	147
1-KAFO	46	18.7	0.41	143
2-KAFO	22	14.9	0.77	149

Adapted from Waters RL, Yakura JS. The energy expenditure of normal and pathological gait. *Critical Reviews in Physical and Rehabilitation Medicine.* 1989;1:187-206.

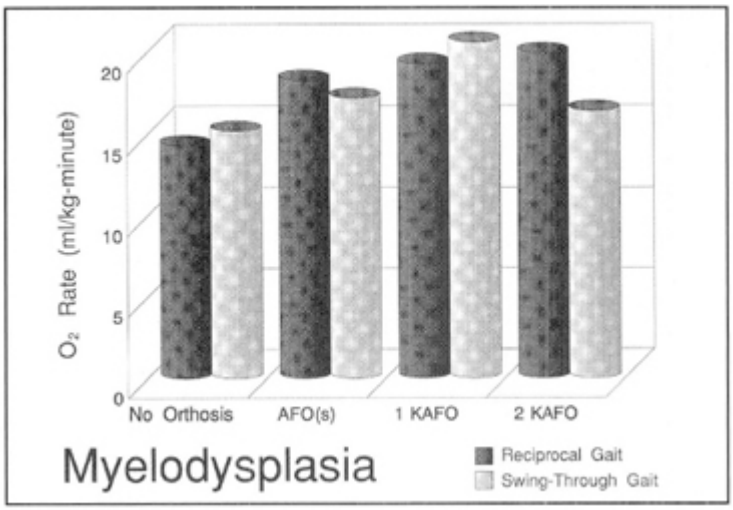

Figure 24-7. Speed in children with myelodysplasia walking with swing-through and reciprocal gait patterns requiring different types of lower extremity orthotic support.

Reciprocal Walking

Patients requiring bilateral KAFOs had the greatest O_2 rate (18.1 mL/kg•min; Figure 24-7), highest O_2 cost (1.35 mL/kg•m; Figure 24-8), and slowest CWS (22 m/min; Figure 24-9). As gait velocity progressively improved, the O_2 cost and O_2 rate progressively diminished in the patient groups with lesser paralysis and needing less orthotic support.

Swing-Through Gait Versus Reciprocal Gait

A direct comparison of energy expenditure during both swing-through and reciprocal walking was obtained in 10 children.[48] The indices reflecting physiological effort (rate of O_2 uptake and heart rate) were slightly higher during swing-through crutch-assisted gait, but gait velocity was faster (Table 24-11). As a consequence, swing-through walking proved the more efficient gait (0.68 ml/kg•m versus 0.40 mL/kg•m). Seven of 10 children preferred the swing-through

mode of crutch use for most activities. It may be concluded that attempts to train children who prefer a swing-through gait to a reciprocal gait are probably unwarranted.

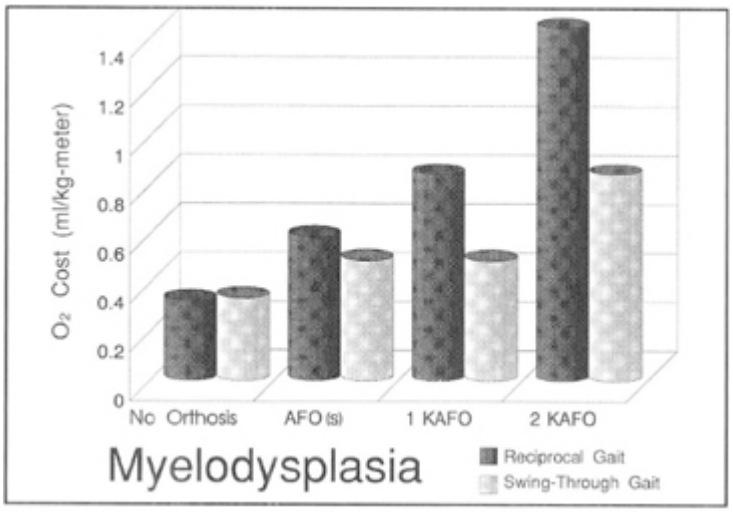

Figure 24-8. O_2 rate in children with myelodysplasia with swing-through and reciprocal gait patterns requiring different types of lower extremity orthotic support.

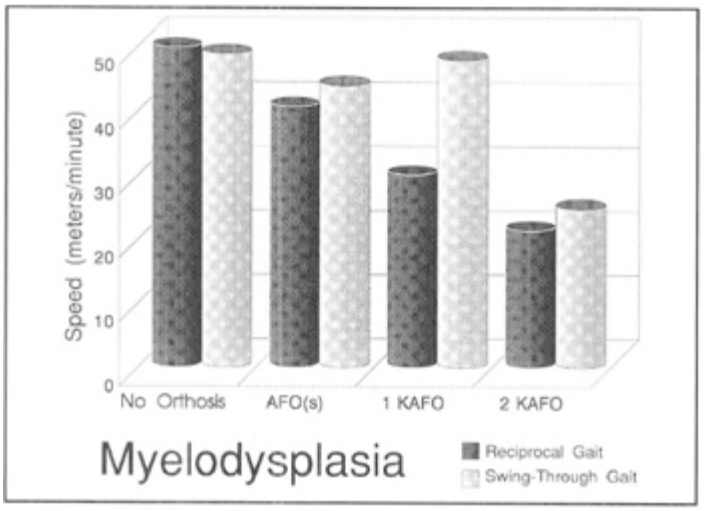

Figure 24-9. O_2 cost in children with myelodysplasia walking with swing-through and reciprocal gait patterns requiring different types of lower extremity orthotic support.

Table 24-11

Myelodysplasia Reciprocal and Swing-Through Gait Versus Wheelchair

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
Reciprocal	30	15.8	0.68	138
Swing-through	42	16.3	0.40	146
Wheelchair	65	11.6	0.17	124

Adapted from Waters RL, Yakura JS. The energy expenditure of normal and pathological gait. *Critical Reviews in Physical and Rehabilitation Medicine.* 1989;1:187-206.

In contrast to the experience in adults with SCI, it is a common clinical experience that a swing-through gait pattern in children is functional for community ambulation outside the home. The difference in the response is due to the higher ratio of arm strength to body weight in children, their higher exercise capacity, and VO_2 max. Also, myelodysplastic children have proportionately lighter legs due to the effects of paralysis during the growing years.

Nevertheless, as children with myelodysplasia approach maturity and gain weight, sustaining walking activity with a swing-through gait becomes more difficult and increasing reliance is placed on the wheelchair. These factors coupled with the normal decline in the maximal exercise capacity with aging account for the reason many patients choose wheeling rather than walking in later years.

AMPUTATION

Following lower extremity amputation, the patient has a choice of walking with crutches in place of a prosthesis or wearing a prosthesis. A special problem confronting many older patients with dysvascular limbs is a limited exercise ability due to associated medical disease.

Prosthesis Versus Crutches

The direct comparison of walking ability by unilateral traumatic and dysvascular amputees at the Syme's, transtibial (TT), and transfemoral (TF) levels with a prosthesis utilizing a swing-through crutch-assisted gait without a prosthesis reveals that all, with the single exception of individuals with vascular TF amputations, have a lower rate of energy expenditure, heart rate, and O_2 cost when using a prosthesis.[44] This difference is significant in persons with dysvascular TF amputations and is related to the fact that even with a prosthesis, most of these patients require crutches for some support, thereby increasing the O_2 rate and heart rate.[35,44]

It can be concluded that a well-fitted prosthesis that results in a satisfactory gait not requiring crutches significantly reduces the physiologic energy demand. Since crutch walking requires more exertion than walking with a prosthesis, crutch walking without a prosthesis should not be considered an absolute requirement for prosthetic prescription and training.

Amputation Level

Two studies in which patients were healthy, young adults and tested under similar laboratory conditions illustrate the importance of the amputation level (Table 24-12). The first group consists of unilateral amputees at the TT, knee disarticulation (KD), and TF levels secondary to trauma.[44] The second group consists of hip disarticulation (HD) and hemipelvectomy (HP) patients following surgical amputation.[25]

The combined results of these 2 studies indicate that the average O_2 rate at all amputation levels when walking with a prosthesis without crutches approximates the value for normal subjects (Figure 24-10). However, as the amputation level ascends, the average CWS progressively decreases, resulting in an increasing O_2 cost (Figures 24-11 and 24-12).

In summary, TT, KD, TF, HD, and HP amputees progressively adapt to the increasingly inefficient gait pattern (higher O_2 cost) resulting from higher-level amputations by reducing their gait velocity so that the O_2 rate does not exceed normal limits.

DYSVASCULAR AMPUTATIONS

It is important to distinguish older dysvascular amputees from their younger, usually traumatic counterparts. A comparison of the 2 etiologies of amputation at the TT and TF levels reveals the CWS is slower and the O_2 cost higher for the dysvascular TT amputee than for the traumatic TT amputees. At the TT level, the CWS and O_2 cost for traumatic amputees (45 m/min and 0.20 ml/kg•m) are significantly different than for traumatic amputees (71 m/min and 0.16 mL/kg•m).

Table 24-12

Energy Expenditure of Persons With Unilateral Amputations

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
Vascular[44]				
TF	36	10.8	0.28	126
TT	45	9.4	0.20	105
Syme	54	9.2	0.17	108
Surgical[25]				
HP	40	11.5	0.29	97
HD	47	11.1	0.24	99
Traumatic[44]				
TF	52	10.3	0.20	111
KD	61	13.4	0.20	109
TT	71	12.4	0.16	106

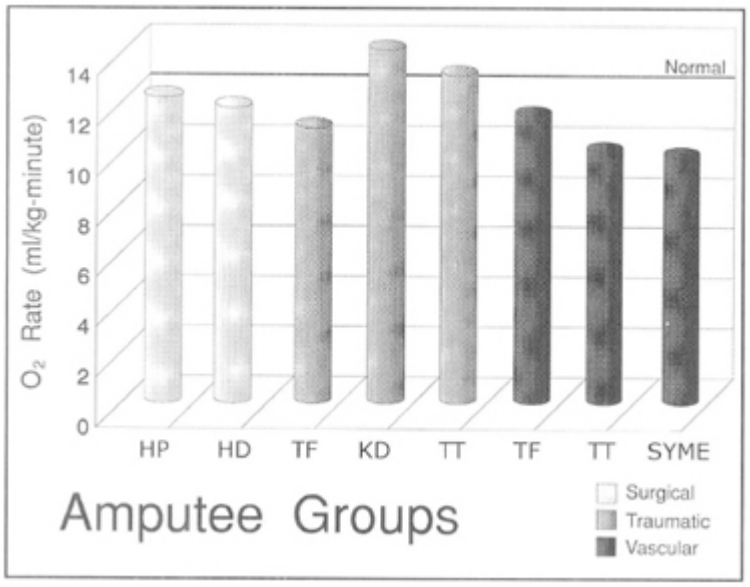

Figure 24-10. Influence of amputation level and underlying cause of amputation on O_2 rate.

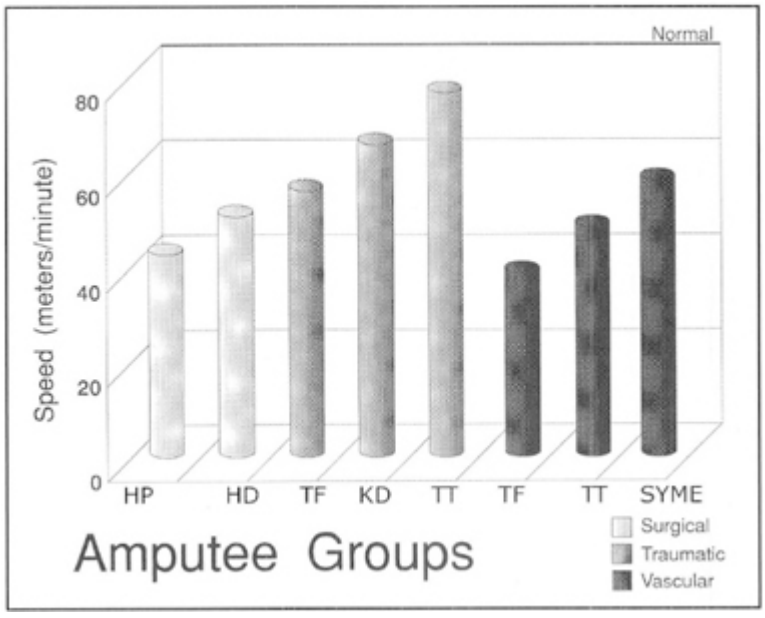

Figure 24-11. Influence of amputation level and underlying cause of amputation on walking speed.

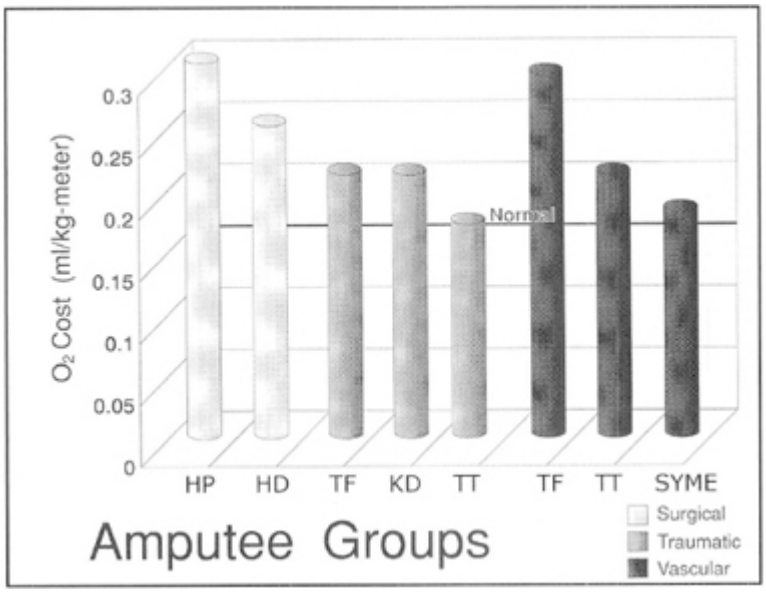

Figure 24-12. Influence of amputation level and underlying cause of amputation on O_2 cost.

The same differences were observed at the TF level between dysvascular and traumatic amputees (36 m/min and 0.28 mL/kg•m versus 52 m/min and 0.20 mL/kg•m). Among dysvascular amputees, Syme patients had a faster CWS and lower O_2 cost (54 m/min and 0.17 mL/kg•m) than TT patients (45 m/min and 0.20 mL/kg•m), demonstrating the same importance of the level of amputation observed in younger traumatic and surgical amputees (see Table 24-12).[44]

Most older patients who have TF or higher amputations for vascular disease are not successful prosthetic ambulators. Very few are able to walk with a prosthesis without crutch assistance.[33,44] If able to walk, they generally have a very slow CWS and an elevated heart rate if crutch assistance is required.[44] It can be concluded that every effort must be made to protect dysvascular limbs early so that TF amputation does not become necessary. If amputation is mandatory, every effort should be made to amputate below the knee.

Table 24-13

Energy Expenditure of Persons With Bilateral Amputations

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
Traumatic				
TT/TT[44]	67	13.6	0.20	112
TF/TF[44]	54	17.6	0.33	104
Vascular				
Syme/Syme[44]	62	12.8	0.21	99
TT/TT[44]	40	11.6	0.31	113
TT/TT[10]	40	7.8	0.23	116
Stubbies[36]	46	9.9	0.22	86

Residual Limb Length

Two studies examined the relationship of residual limb length to gait performance in TT patients.[14,18] In the first, residual limbs ranged from 14 to 19 cm in length and in the second, residual limbs ranged from 9 to 24 cm. Nearly all sockets were patellar tendon bearing (PTB). No significant correlations were noted between CWS and energy expenditures in either study. Of particular clinical importance, a residual limb as short as 9 cm results in TT performance (lower O_2 cost and higher CWS) that is superior than at the KD and TF levels.

Weight of Prosthesis

Most unilateral amputees prefer a prosthesis that is lighter in weight than the missing limb segment. Mattes et al investigated the hypothesis that increasing the mass and moment of inertia of a transtibial prosthesis to match the intact limb would improve gait symmetry without increasing energy cost.[23] However, the loading configuration required to produce a match in the moments of inertia

of the prosthetic and intact legs resulted in greater gait asymmetry and higher energy cost.

Bilateral Amputees

Not surprising, bilateral amputees expend greater effort than unilateral amputees (Table 24-13).[10,44] Dysvascular patients with the Syme/Syme combination walk faster and have a lower O_2 cost than dysvascular patients with the TT/TT combination. Traumatic TT/TT amputees walk faster and at a lower energy cost than their dysvascular TT/TT counterparts.

Considering that approximately one-third of diabetic amputees lose the remaining leg within 3 years, it is important to preserve the knee joint even if the residual limb is short since, should a unilateral TT amputee undergo another TT amputation, energy expenditure would be less than a patient with a unilateral TF amputation.[14] Bilateral vascular amputees rarely achieve a functional ambulation status if one amputation is at the transfemoral level.

Table 24-14

Energy Expenditure—Arthritis of the Hip

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
Preoperative THA[6]	41	10.3	0.28	106
Postoperative THA[6]	55	11.1	0.20	108
Girdlestone[45]	46	12.2	0.39	118
Hip Fusion[37]	67	14.7	0.22	112

Evaluation of a 21-year-old bilateral KD/KD patient with stubby prostheses revealed that the patient attained a CWS of 46 m/min at an O_2 rate of 9.9 mL/kg•min, achieving an O_2 cost of 0.22 mL/kg•m.[36] While walking on stubbies is cosmetically unacceptable for most patients (except for gait training or limited walking in the

home), the data from this single patient illustrate it can be functionally useful.

ARTHRITIS

HIP

Arthritic hip pain causes an antalgic gait pattern. Testing performed on patients with unilateral osteoarthritis of the hip prior to total hip arthroplasty (THA) reveals the average CWS, 41 m/min, is approximately half the normal speed and the O_2 rate, 10.3 mL/kg•min, is also below normal.[6] THA results in an improved walking speed at a reduced O_2 cost. The CWS 1 year following surgery increased from 41 m/min to 55 m/min without any further increase in the O_2 rate (Table 24-14). Since the CWS improves without a corresponding increase in the O_2 rate, there is a lower O_2 cost from 0.28 mL/kg•m to 0.20 mL/kg•m, indicating improved gait efficiency.

It is of clinical importance to compare the results of the THA to the previously discussed results following hip fusion since both procedures can be performed for unilateral hip arthritis. Hip fusion results in a faster gait, but requires a 32% greater O_2 rate than THA (see Tables 24-4 and 24-14). A decreased O_2 rate is an advantage of THA over hip fusion in a patient with unilateral disease.

Girdlestone hip resection arthroplasty (ie, excision of the hip joint and removal of the prosthesis) is commonly performed because of persistent infection after THA. This is one of the most severe types of complication after joint replacement surgery. The CWS following Girdlestone arthroplasty averages 46 m/min, O_2 rate 12.2 mL/kg•min, and heart rate 118 bpm.[45] The elevated heart rate reflects the reliance of the majority of these patients on crutches for partial weight support.

KNEE

Evaluation of patients with severe osteoarthritis of the knee tested prior to total knee arthroplasty reveals that the average reduction in the CWS and increase in the O_2 cost are approximately the same as in patients with osteoarthritis of the hip.[45] The similarity of these results in the 2 different joint is not surprising. In an effort to relieve pain, patients with arthritis at either the hip or the knee adopt many of the same strategies: decreasing the duration of the single stance phase on the involved leg to minimize the duration of loading and using upper extremity gait assist devices, which reduces the magnitude of the load.

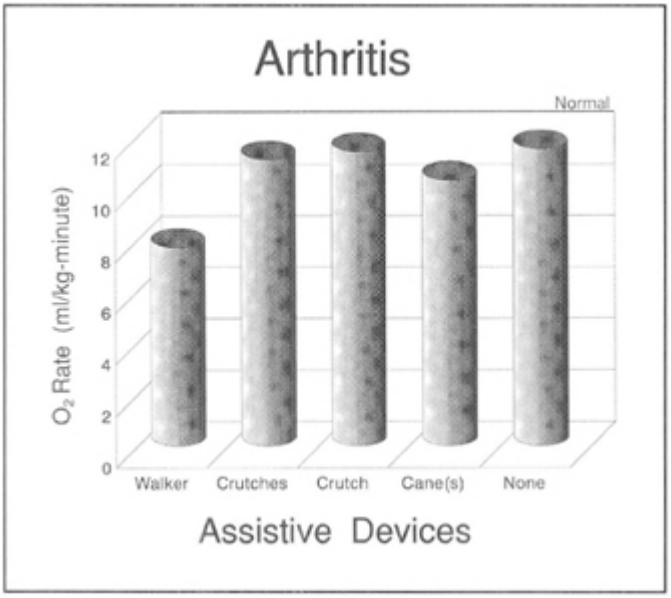

Figure 24-13. Rate of O_2 consumption in arthritis patients requiring different types of upper extremity assistive devices.

RHEUMATOID ARTHRITIS

Evaluation of patients with rheumatoid arthritis indicates a functional benefit of total knee joint replacement surgery even in the presence of systemic illness.[45] Evaluation of rheumatoid patients with severe degeneration of the knee joint as the primary problem

indicates that a significant improvement in gait velocity occurs after surgery (58 m/min versus 33 m/min) with only a slight increase in the O_2 rate (11.4 mL/kg•min versus 10.3 mL/kg•min). Also, there is a marked improvement in the O_2 cost (0.41 mL/kg•m versus 0.71 mL/kg•m).

Influence of Upper Extremity Assistive Devices

The use of crutches, canes, or walkers in arthritis usually depends on the severity of pain. Forces across an inflamed joint are progressively unloaded by provision of a cane, one crutch, 2 crutches, or a walker.

In a group of patients with severe knee rheumatoid arthritis tested prior to surgery, the O_2 rate was not elevated above the value for normal subjects at their CWS (Figure 24-13). Patients using a walker had the slowest CWS (Figure 24-14) and highest O_2 cost (Figure 24-15).[45] Speed progressively increased in the groups of patients requiring 2 crutches, one crutch, cane(s), or no assistive devices (Table 24-15). Conversely, O_2 cost was highest in patients requiring a walker and least in those requiring no upper extremity assistive devices.

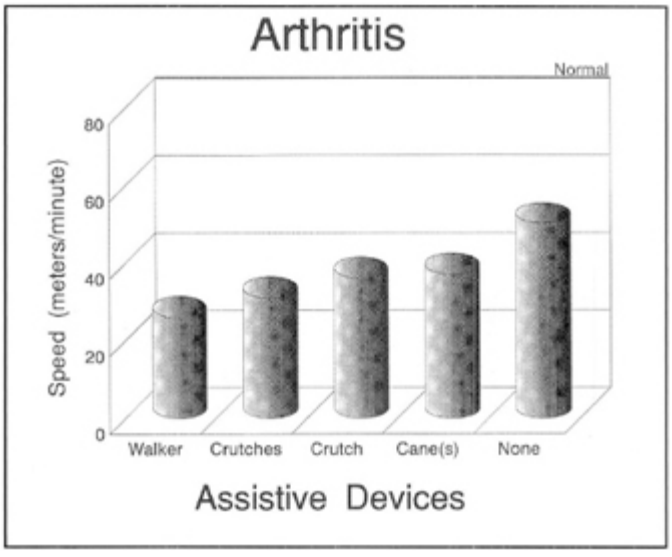

Figure 24-14. Speed in arthritis patients requiring different types of upper extremity assistive devices.

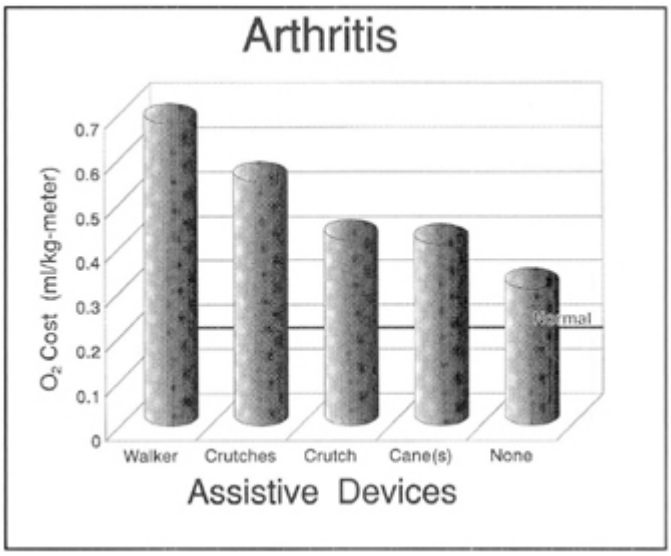

Figure 24-15. O_2 cost in arthritis patients requiring different types of upper extremity assistive devices.

DECONDITIONING

The O_2 pulse is lower than normal in both osteoarthritis and rheumatoid arthritis patients tested prior to surgery, even when upper extremity assistive devices are not employed.[45] These findings signify that pain leading to a more sedentary lifestyle and/or the systemic effects of rheumatoid disease result in deconditioning.

HEMIPLEGIA (STROKE)

Spasticity and primitive patterns of motion characterize hemiplegic gait, and the degree of impairment depends on the magnitude of the neurological deficit.[15] Because of a marked reduction in the speed of the typical patient, the O_2 rate is less than normal despite the inefficiency of the hemiplegic gait pattern and high O_2 cost.[15] The mean O_2 rate among ambulatory hemiplegic patients was 11.5

mL/kg•min, slightly below the mean for normal walking. The average speed was very slow, 30 m/min, accounting for a high O_2 cost, 0.54 ml/kg•m (Table 24-16). It can be concluded that hemiplegic gait is not physiologically stressful for the typical patient unless there is severe cardiovascular disease.

Table 24-15

Rheumatoid Arthritis of the Knee Preoperative Evaluation of Influence of Upper Extremity Assistive Devices

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
Walker	21	7.2	0.63	124
Crutches	26	10.6	0.50	124
Crutch	31	10.9	0.37	102
Cane(s)	32	9.8	0.36	97
No assistive device	45	11.0	0.26	115

Adapted from Waters RL, Perry J, Conaty P, Lunsford B, O'Meara P. The energy cost of walking with arthritis of the hip and knee. *Clin Orthop.* 1987;214:278-284.

Table 24-16

Hemiplegia

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
Wheelchair	32	10.0	0.27	107
Walking	30	11.5	0.54	109

Adapted from Hash D. Energetics of wheelchair propulsion and walking in stroke patients. In: Energetics: Application to the study and management of locomotor disabilities. *Orthop Clin North Am.* 1978;9:351-377.

ENERGY EXPENDITURE OF FLEXED-KNEE GAIT

Neurological disorders such as spastic diplegia due to cerebral palsy cause the patient to walk with a flexed lower limb posture. The biomechanical requirements of flexed-knee stance are greater than normal and are associated with increased quadriceps, tibio-femoral, and patello-femoral forces.[28] The most significant increases occur at angles of knee flexion beyond 15°.

Table 24-17

Energy Expenditure of Flexed-Knee Gait

	Speed m/min	*O_2 Rate kg•min*	*O_2 Cost mL/kg•m*
0°	80	11.8	0.16
15°	77	12.8	0.17
30°	75	14.3	0.19
45°	67	14.5	0.22

Adapted from Rueter K, Pierre M. Energy cost and gait characteristics of flexed knee ambulation. In: Waters RL, Lunsford BR, eds. *Energy Expenditure of Normal and Pathologic Gait: Application to Orthotic Prescription. Atlas of Orthotics.* St. Louis, MO: CV Mosby Co; 1985.

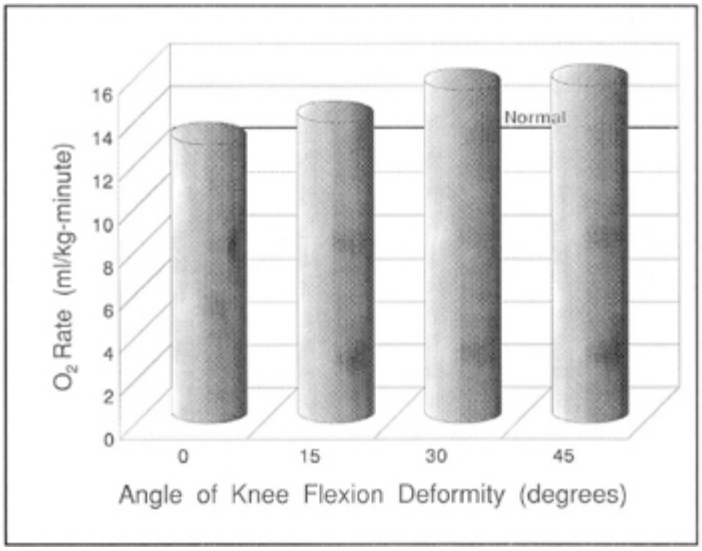

Figure 24-16. O_2 rate in normal subjects walking with progressive knee deformity.

The importance of full knee extension during the stance phase of gait and the significance of knee flexion deformity is illustrated in normal subjects wearing a specially designed hinged knee orthosis

that restricted knee extension but allowed full flexion.[31,43] As the amount of simulated knee flexion contracture is increased, the O_2 rate and O_2 cost progressively rise and the CWS decreases (Table 24-17).

Restricting knee extension to 30° of flexion leads to a 15% rise in O_2 rate and a 19% increase in O_2 cost (Figures 24-16, 24-17, and 24-18). Restricting knee extension to 15° causes a 7% rise in the rate of O_2 consumption and a 13% increase in O_2 cost in comparison to normal walking. Limiting knee extension to 45° resulted in a 21% increase in O_2 rate and 38% increase in O_2 cost.

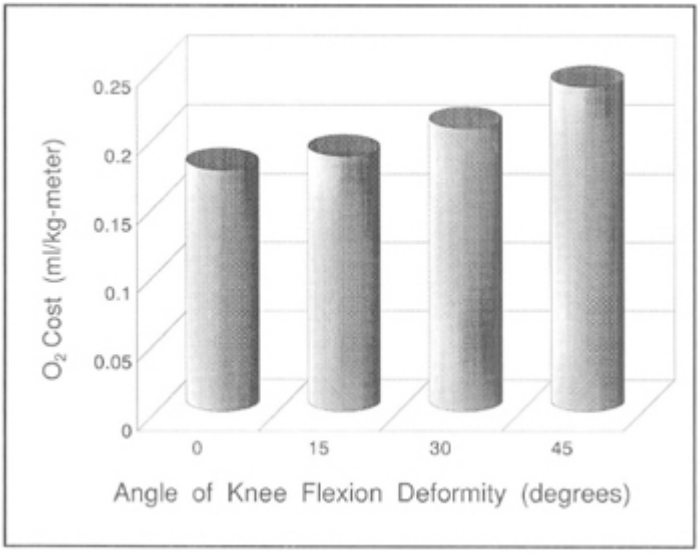

Figure 24-17. O_2 cost in normal subjects walking with progressive knee deformity.

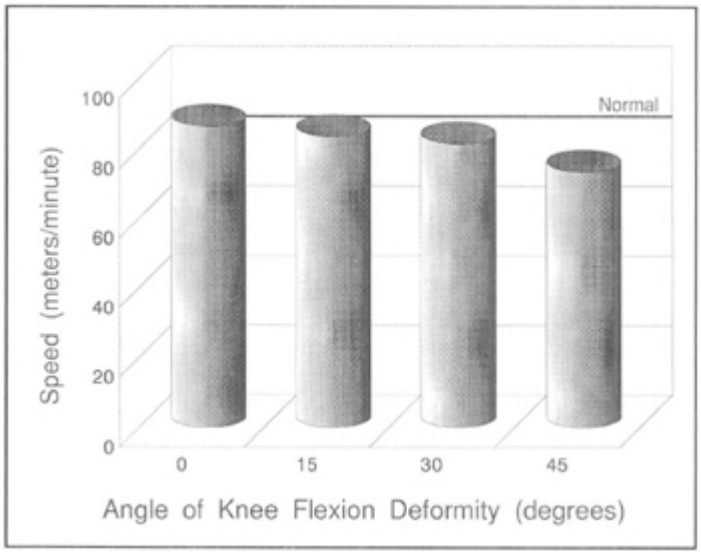

Figure 24-18. Speed in normal subjects walking with progressive knee deformity.

SPASTIC DIPLEGIA (CEREBRAL PALSY)

The child with spastic diplegia involving both lower limbs typically has spasticity and a pervasive loss of motor control, which depends on the degree of involvement. When moderate or severe disability is present, the child typically fails to fully extend the hips and knees and has a flexed hip and knee posture. Walking speed in a group of spastic diplegic children between the ages of 5 and 17 years averaged 40 m/min.[7] The mean heart and O_2 rates were significantly higher than normal, averaging 145 bpm and 18.6 mL/kg•min. As a group, these children had a very inefficient gait, averaging 0.72 mL/kg•m (Table 24-18).

Table 24-18

Cerebral Palsy

	Speed m/min	*O_2 Rate mL/kg•min*	*O_2 Cost mL/kg•m*	*Pulse beats/min*
Spastic diplegia	40	18.6	0.72	145

Adapted from Campbell J, Ball J. Energetics of walking in cerebral palsy. In: Energetics: Application to the study and management of locomotor disabilities. *Orthop Clin North Am.* 1978;9:351-377.

Most individuals with gait disabilities that do not require the use of upper extremity assistive devices purposefully slow their speed and are able to keep the rate of O_2 consumption from rising above normal limits. Nevertheless, elevated rates of O_2 consumption and high heart rates were consistently recorded in spastic diplegic children even when upper extremity assistive devices were not required.

The flexed hip and knee posture of the child with spastic diplegia requires considerable muscular effort by the antigravity muscles to prevent collapse even at a slow speed. Another reason for the elevated O_2 rate is the limited motor control to perform the necessary compensatory gait substitutions since both legs are involved. In contrast, the patient with hemiplegia can perform substitutions with the uninvolved limb and spends approximately 80% of the GC on the uninvolved leg. Also, during the period of SLS on the affected side, the patient is able to maintain the hip and knee in a relatively extended position.[27]

The O_2 rate decreases in individuals without pathology as they grow older. The opposite trend was observed in children with spasticity. Their O_2 rate increased with age. This observation has clinical significance for the spastic child who complains of fatigue or the need for rest. Clinically, this is consistent with the increased body weight and size in older children and the greater difficulty of the child with impaired motor control and spasticity carrying the added weight. As a result, the older child may prefer to walk less and increasingly rely on a wheelchair.

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Abbreviations and Acronyms

ABBREVIATION TERM

ADD LONG	adductor longus
ADD MAG	adductor magnus
ADP	adenosine diphosphate
AFO	ankle-foot orthosis
AK	above knee
AMI	Ambulatory Motor Index
ANK	ankle
ANT TIB	anterior tibialis
AP	anterior-posterior
ASIS	anterior superior iliac spine
ATIB	anterior tibialis
ATP	adenosine triphosphate
BFLH	biceps femoris long head
BFSH	biceps femoris short head
BK	below knee
BMR	basal metabolic rate
bpm	beats per minute
BW	body weight
BW/LL	body weight per leg length
cal	calorie (gram)
CC	calcaneocuboid
C/G	center of gravity
cm	centimeter
CO_2	carbon dioxide
COG	center of gravity
COM	center of mass
COP	center of pressure
CP	creatine phosphate
C/P	center of pressure
C7	seventh cervical vertebra
CTO	contralateral toe-off

CVA	cerebral vascular accident
CWS	customary/comfortable walking speed
db	decibel
DER	dynamic elastic response
DF	dorsiflexion/dorsiflexes/dorsiflexor
du	digitized units
EDL	extensor digitorum longus
EHL	extensor hallucis longus
EMG	electromyograph(y)
F	force
FCT	fibrous connective tissue
FDL	flexor digitorum longus
FHL	flexor hallucis longus
FMS	Functional Mobility Scale
FTSW	foot switch
FWS	fast walking speed
g	gram
GAST	gastrocnemius
GC	gait cycle
GMax	gluteus maximus
GMax L	gluteus maximus, lower
GLUT MAX L	gluteus maximus, lower
GLUT MAX U	gluteus maximus, upper
GLUT MED	gluteus medius
GMFCS	Gross Motor Functional Classification Scale
GRAC	gracilis
GRF	ground reaction force
GRFV	ground reaction force vector
H	heel
HAT	head, arms, and trunk
HD	hip disarticulation
Hg	mercury
H-1	heel, first metatarsal
H-1-5	heel, first and fifth metatarsal
H-5	heel, fifth metatarsal
HP	hemipelvectomy
HST	heel strike transient
Hz	hertz
IC	initial contact

IEMG	integrated electromyography
ILIAC	iliacus
ISw	initial swing
IT	iliotibial
ITB	iliotibial band
ITO	ipsilateral toe-off
KAFO	knee-ankle-foot orthoses
kcal	kilogram-calorie
kg	kilogram
kPa	kilopascal
LA	lever arm
LED	light emitting diode
LGMax	lower gluteus maximus
LL	leg length
LR	loading response
L3	third lumbar vertebra
m	meter
MG	medial head of gastrocnemius
MHam	medial hamstring
MHR	maximum heel raise
min	minute
mL	milliliter
ML	medial-lateral
mm	millimeter
m/min	meters per minute
MMT	manual muscle test
M OHM	meg ohm (10 ohms)
MP	metatarsophalangeal
ms	millisecond
MSt	mid stance
MSw	mid swing
MT	midtarsal
MT5	fifth metatarsal
MTP	metatarsophalangeal
MU	motor unit
mv	millivolt
MVC	maximum voluntary contraction
N	number
Nm	Newton-meter

N•m/kg	Newton-meter/kilogram
N•m/kg•m	Newton-meter/kilogram-meter
O_2	oxygen
OPP FS	opposite foot support
PAL	peak axial load
PB	peroneus brevis
PCSA	physiological cross sectional area
PF	plantar flexion/plantar flexes
pH	hydrogen ion concentration
PL	peroneus longus
psi	pounds per square inch
PSIS	posterior superior iliac spine
PSw	pre-swing
PT	posterior tibialis
PTB	patellar tendon bearing
PTIB	posterior tibialis
% N	percent normal
r	correlation coefficient
REF FS	reference foot support/switch
RER	respiratory exchange ratio
RF	rectus femoris
RF SURF	rectus femoris surface
RLANRC	Rancho Los Amigos National Rehabilitation Center
ROM	range of motion
RQ	respiratory quotient
SACH	solid ankle, cushioned heel
SART	sartorius
SCI	spinal cord injury
SD	standard deviation
sec	second
SI	sacroiliac
SLA	swing limb advancement
SLS	single limb support
SMEMB	semimembranosus
SOL	soleus
ST	subtalar
STEND	semitendinosus
S_2	second sacral vertebra
SWS	slow walking speed

T	torque
TA	tibialis anterior
TAMP	time adjusted mean profile
TF	transfemoral
TFL	tensor fascia lata
THA	total hip arthroplasty
3D	three-dimensional
TK	through knee
TL	thoracolumbar
TN	talonavicular
TO	toe-off
TP	tibialis posterior
TPE	triaxial parallelogram electrogoniometer
TSt	terminal stance
TSw	terminal swing
T_{10}	tenth thoracic vertebra
TT	transtibial
µ	micron
uv	microvolt
V	velocity
VI	vastus intermedius
VL	vastus lateralis
VML	vastus medialis longus
VMO	vastus medialis oblique
VO_2	volume of oxygen, maximal aerobic capacity
WA	weight acceptance
W/kg	watts per kilogram

Glossary

abduction: Movement away from the midline of the body.
accelerometer: An instrument that measures the rate of change of speed (ie, acceleration).
active: Muscle contraction provides the effective force.
adduction: Movement toward the midline of the body.
assistive devices: Walking aids such as crutches, canes, walkers.
aerobic: Muscle-energizing process that uses oxygen.
Ambulatory Motor Index: Sum of the bilateral muscle strength grades of the hip (flexion, abduction, extension) and knee (extension and flexion).
amplifier: An electronic device used to enlarge the myoelectric signals.
amputation: Loss of the distal portion of the limb.
anaerobic: Muscle-energizing process without the use of oxygen.
ankle rocker: Dorsiflexion at the ankle that contributes to limb progression.
arthrodesis: Surgical ablation of joint motion.
automated motion analysis: A system that senses and quantifies limb motion without operator intervention.
automated video systems: Operator-free motion recording systems that use video as the medium.
bilateral: Involving both the right and left limbs (sides of the body).
body weight vector: Force line that indicates the mean alignment and magnitude of body weight relative to the joint of interest.
burn: Tissue injury from excessive heat.
cadence: Step rate per minute.
calcaneus gait: Walking primarily on the heels.
calcaneograde: Walking on the heel.
calorimetry: The measurement of body heat to determine physiological energy expenditure.
cauda equina: The group of spinal roots within the vertebral canal that extend below the spinal cord.

center of pressure: The location of the mean weight-bearing forces at the foot-floor interface (base of the vector).
cerebral palsy: Nonprogressive paralysis resulting from a brain injury near the time of birth.
clubfoot: Congenital deformation resulting in twisting of the newborn's foot in all 3 planes (adduction, inversion, and equinus).
concentric contraction: Shortening of the muscle as it creates its force.
contracture: Fibrous connective tissue shortening that causes a loss of normal joint range.
contralateral: The opposite side of the body; the other limb.
coronal plane: The plane that divides the body into anterior and posterior halves; also known as the frontal plane.
customary walking speed: The rate of walking that is voluntarily assumed.
deceleration: Slowing or inhibition of the prior rate of motion.
deformity: A fixed (static) malalignment of the bone or joint.
degenerative arthritis: An older synonym for osteoarthritis. Deterioration of the joint cartilage and bone of nonspecified etiology, often attributed to wear and tear from overuse or maluse.
digigrade: Walking on one's toes.
diplegia: Paralysis involving both lower limbs; a cerebral palsy classification.
dorsiflexion: Movement of the top of the foot toward the anterior surface of the tibia with motion occurring at the ankle.
double stance: Stance with both feet in contact with the floor (eg, initial contact, loading response and pre-swing).
drop foot: Passive equinus, excessive ankle plantar flexion during swing.
dynamic: Active muscular control.
dynamic electromyography: Recording of the muscle activation signals during functional activities.
dysvascular amputee: An individual with the loss of a limb secondary to circulatory impairment.

eccentric contraction: Lengthening of the contracting muscle due to a higher external load.
efficiency: Percentage of energy input that is transformed into useful work.
effort: The exertion of a force to either inhibit or create motion.
elastic contracture: Fibrous tissue restraint of motion that partially stretches with body weight or a very vigorous manual force.
electrode: The devices used to capture the myoelectric signals (eg, wire, needle, disc).
electrogoniometer: A device attached to the limb to record joint motion.
electromyography: A system for recording the myoelectric signals generated during active muscle contraction.
energy: The capacity to perform work.
energy conservation: Functional measures used to reduce the energy cost of activity.
equinus: A toe-down position of the foot in which the forefoot is lower than the heel.
eversion: Lateral tilting of the subtalar joint.
extension: A straightening of the limb in which the bones become more parallel; opposite of flexion.
extensor thrust: Rapid backward motion at the knee that does not create overt hyperextension.
flexed-knee gait: Gait in which the knee remains bent throughout stance.
flexion: Bending the joint (ie, the distal segment rotates toward the proximal segment).
filtration (electronic): Electronic exclusion of waveform with a designated frequency.
foot flat: Floor contact by both the heel and the forefoot.
foot support patterns: The different combinations of floor contact by the heel, medial and lateral metatarsal heads, and great toe.
footswitch: A device that measures the time of floor contact by the designated areas of the foot.
force: Any influence that causes a change in position or alters the direction of speed of motion.

force plate: A platform set on or into the floor that is instrumented to measure the forces imposed on it.

forefoot contact: Impact of the forefoot with the floor.

forefoot rocker: Progression of the limb (and body) while the forefoot is the pivotal area of support.

fracture: Broken bone.

free gait: Walking at one's own spontaneous (customary) rate of travel.

frequency (electronic): A quality of an electronic signal relative to its sine wave content.

frontal plane: The plane that divides the body into anterior and posterior halves; also known as the coronal plane.

gait analysis: A method for diagnosing the way people walk.

gait cycle: A single sequence of events between 2 sequential initial contacts by the same limb.

gait phases: The divisions in the walking cycle that represent specific functional patterns.

ground reaction forces: The forces recorded by a force plate generated by falling body weight or muscle action as the person walks across that area of the floor.

ground reaction force vector: The mean directional and magnitude sum of the force imposed on the ground (floor) for that sample.

hamstrings: The posterior thigh muscles extending from the pelvis to the shank (semimembranosus, semitendinosus, and long head of the biceps femoris).

HAT: Head, arm, neck, and trunk segments that comprise the passenger unit that rides atop the locomotor system.

heel rocker: Progression of the limb (and body) while the heel is the pivotal area of support.

heel strike: Floor contact with the heel; the normal mode for initiating stance.

hemiplegia: Paralysis of the arm, leg, and trunk on the same side of the body (right or left).

hyperextension: Posterior angulation of the joint beyond neutral (ie, excessive extension).

iliotibial band: A length of dense fascia on the lateral side of the thigh that extends from the pelvis (crest of the ilium) to the anterior, proximal margin of the tibia.
inertia: Resistance to initiation or change of motion.
initial contact: First impact with the floor; the event that begins stance.
initial double stance: The beginning of stance when both feet are on the floor, equivalent to initial contact and loading response.
initial swing: The first phase of limb advancement of the foot is lifted from the floor.
instrumented walkway: A length of flooring that contains sensors to record the floor contact events.
inversion: Inward tilting of the subtalar joint.
ipsilateral: On the same side of the body or limb.
isokinetic contraction: Muscle action occurring while there is a consistent rate of joint motion.
isometric contraction: Muscle action that occurs while all joint motion is inhibited.
lean: Tilt of the trunk away from vertical position (eg, forward, backward, or to the side).
ligamentous skeleton: The ligament retained to preserve the natural connections between the bones of the body (or a segment).
limb advancement: Forward movement of the unweighted limb; a function of swing.
loading response: The second phase of the gait cycle; a phase of initial double limb support.
locomotor unit: The 2 lower limbs and the pelvis that provide the mechanics of walking.
lordosis: Posterior angulation of the spine in the sagittal plane.
low heel contact: Floor impact by the heel with the forefoot very close to the floor thus providing a limited heel rocker.
markers: Balls or disks applied to the skin over designated anatomical landmarks used to designate the segments for remote motion analysis.
mid stance: The first portion of the single limb support interval.

moment: The rotational potential of the forces acting on a joint; also called *torque*.

momentum: The tendency to remain in motion unless an opposing force is applied.

motion analysis: A system to define the movement of the different body segments during walking and other functional activities.

motor unit: The functional neuromuscular unit consisting of the peripheral neuron (cell body, axon, end plate), myoneural junction, and the muscle fibers controlled by the branches of that axon.

muscle grade: The strength of the muscle designated by the manual muscle test on a scale of 0 (no force produced) to 5 (normal).

muscular dystrophy: An inherited, progressive disease of the muscle that causes increasing weakness and contracture formation.

myelodysplasia: A congenital form of paralysis resulting from a spinal cord malformation characterized by failure of neural tube closure.

neuron: A single nerve fiber consisting of a cell body, axon, and terminal junction.

normalization (EMG): Relationship of the raw EMG of an activity to a basic reference EMG (eg, a manual muscle test or the maximum activity recorded during gait).

observational gait analysis: Visual definition of an individual's limb and trunk motions during walking.

optoelectrical recording: Automated motion analysis using electronic signals (lights) as the landmarks.

orthosis: An external device to provide support that can limit or assist motion.

osteoarthritis: Progressive deterioration of the joint cartilage and bone of nonspecific etiology.

oxygen cost: The amount of oxygen used per meter walked (millimeters/kilogram of body weight/meter walked).

oxygen pulse: The ratio between the amount of oxygen used per minute and heart rate.

oxygen rate: The amount of oxygen used per minute (millimeters/kilogram/minute).

pantalar fusion: Surgical arthrodesis of the ankle, subtalar, and midtarsal joints.

parallelogram electrogoniometer: A device to measure joint motion that has rectangular shaped arms consisting of 4 linked segments for free-shape changes to accommodate the change in joint axis location.

paraplegia: Paralysis of the 2 lower limbs; also referred to as *diplegia.*

passenger unit: The composite body mass composed of the head, arm, neck, and trunk segments that rides atop the locomotor system.

passive: The structure has no means of generating a force. The motion results from an outside source.

pass-retract: Excessive hip flexion (pass) followed by rapid extension (retract) to provide terminal swing knee extension in the absence of adequate quadriceps.

pathokinesiology: The science of defining the function of persons with physical impairments.

pathological gait: An abnormal walking pattern.

patterned movement: Mass extension or flexion of the limb by primitive locomotor control.

peak axial load: The maximum vertical force registered during walking.

pelvic drop: Descent of one side of the pelvis below the neutral axis (zero line).

pelvic hike: Elevation of one side of the pelvis above the neutral axis (zero line).

pelvic tilt: Angulation (either anterior or posterior) of the pelvis from neutral alignment in the sagittal plane.

percent gait cycle: The one-hundredth part within one sequence of walking mechanics.

perimalleolar muscles: The posterior muscles that pass close to the medial and lateral malleoli of the ankle as they extend from their origins on the tibia and fibula to their insertions within the

foot (tibialis posterior, flexor hallucis longus, flexor digitorum longus, peroneus longus, peroneus brevis).

plantar flexion: Movement of the foot away from the anterior surface of the tibia (ie, straightening the ankle joint).

plantigrade: Simultaneous floor contact by the forefoot and heel.

poliomyelitis: Paralysis caused by a viral invasion of the motor nerve cells in the anterior horn of the spinal cord.

power: The rate at which work is performed.

premature heel rise: Elevation of the heel from the floor prior to the onset of terminal stance.

pre-swing: The last phase of stance that also is the second period of double limb support.

primitive locomotor control: A simple voluntary source of motion that uses mass extension and mass flexion of the limb joints. It is an extrapyramidal control system.

progression: Advancement along the sagittal plane when walking.

quadriplegia: Paralysis of both upper and both lower extremities. Also referred to as tetraplegia.

reciprocal gait: Alternate function of the right and left limbs.

relative effort: Percent of the baseline maximum muscular effort displayed by normalized EMG.

repetition rate: The number of action potentials occurring per second.

respiratory quotient: The ratio of carbon dioxide production to oxygen consumption.

rheumatoid arthritis: A systemic inflammatory disease that attacks the joints.

rigid contracture: Fibrous tissue restraint of joint motion that does not yield under body weight or forceful manual stretching.

rotation: Motion about a center point (axis) in which the distal end of the segment travels further than the proximal end.

sagittal plane: The plane of the body that extends from front to back, parallel to the sagittal suture of the skull.

sarcomere: The intrinsic contracting (force) unit within a muscle fiber.

scoliosis: Lateral curvature of the spine.

selective control: Voluntary control that allows individual muscle activation for the appropriate duration and intensity that is functionally required.

shock absorption: Muscle action that lessens the impact of limb loading by allowing controlled joint motion.

shear: Sliding displacement parallel with the surface of the joint.

single axis: Joint movement in one plane.

single limb support: Full weight-bearing on one lower extremity, corresponding with the phases of mid stance and terminal stance.

spasticity: An over-reactive response to quick stretch.

spirometry: Measurement of the amount of air the respiratory system can move (inhale and exhale).

stability: Relationship between the body's center of mass and the base of support.

stance: The period in walking when the foot is in contact with the floor. This includes the phases of initial contact, loading response, mid stance, terminal stance, and pre-swing.

static: Stationary, nonmoving.

steady state: Each cycle of function is the same, being free of accelerations or decelerations.

step: The interval in the gait cycle between initial contact with one foot and then the other foot (ie, right to left).

step length: The distance between the sequential points of initial contact by the 2 feet.

stiff knee gait: Significantly limited swing phase knee flexion.

stride: The interval in the gait cycle between 2 sequential initial contacts with the same foot (ie, right to right).

stride length: The distance between the sequential points of initial contact by the same foot.

stride characteristics: Measurement of the time and distance qualities of the person's walk (eg, stride length, cadence, single limb support).

surface electrode: Devices applied to the skin surface to sense the underlying electromyography.

swing through gait: A form of crutch walking that alternates support by both crutches and then by both feet. Progression

occurs as the 2 limbs swing through during the crutch support period.

symphysis down: Anterior tilt of the pelvis that places the symphysis below the neutral resting position.

symphysis up: Posterior tilt of the pelvis that raises the symphysis above the neutral resting position.

swing: The period in the gait cycle when the foot is not in contact with the floor. This is made up of initial swing, mid swing, and terminal swing.

terminal double stance: The last phase of stance when both feet are in contact with the floor (ie, pre-swing).

terminal stance: The last half of the single limb support period.

terminal swing: The last third of the limb advancement interval.

tetraplegia: Paralysis of both upper and both lower extremities. Also referred to as quadriplegia.

time-adjusted EMG quantification: The mean electromyographic (muscle activity) profile is located within the mean onset and cessation time of a series of strides.

toe drag: Advancement of the limb during swing is accompanied by continuing floor contact by the toe.

toe rocker: Rotation of body weight over the terminal segment of the foot during the final phase of stance.

torque: The rotational potential of the forces acting on a joint (also called moment).

transverse plane: The plane that divides the body into upper and lower halves; also called the horizontal plane.

trauma: Tissue injury by a force.

treadmill: A device with a belt on rollers that allows locomotion (walking or running) in place.

Trendelenburg limp: Trunk lean to the same side as the hip pathology (ie, an ipsilateral lean).

triaxial: Movement by a joint in three planes.

triple arthrodesis: Surgical fusion of the hind foot (subtalar, calcaneocuboid and talonavicular joints).

unguligrade: Tip-toe walking.

valgus: Lateral angulation of the distal segment of a joint, turning the foot out (a clinical synonym for eversion).

varus: Medial angulation of the distal segment of a joint, turning the foot in (a clinical synonym for inversion).

vector: The mean weight-bearing line of the body with both magnitude and directional qualities.

velocity: The speed of walking in a designated direction.

video recording: A record of a subject's function that allows observational analysis.

weight acceptance: The initial period in the gait cycle when body weight is dropped onto the limb. The phases of initial contact and loading response are involved.

wire electrode: A pair of fine, nylon-coated wires (50 μ) with 2-mm barbed tips that are inserted within the designated muscle to record the intensity and timing of function.

Wolf's law: The basic bony structure will be modified by the weight-bearing and muscular forces it experiences.

work: The product of a force times the distance the force acts.

Appendix A Normative Joint Motion

Normative sagittal plane joint motion of the ankle (n=54), knee (n=54), thigh (expressed relative to vertical; n=55) and hip (n=55) recorded at a self-selected walking speed.

% Gait Cycle	Ankle		Knee		Thigh (Versus Vertical)		Hip	
	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)
0	-2.0	3.0	4.7	4.8	21.6	3.3	31.2	6.2
1	-3.0	3.0	6.4	4.7	21.7	3.3	31.2	6.1
2	-4.1	3.0	8.0	4.6	21.7	3.4	31.2	6.1
3	-5.1	2.9	9.6	4.5	21.7	3.4	31.2	6.1
4	-5.7	3.0	11.2	4.6	21.6	3.5	31.0	6.1
5	-5.7	3.1	12.8	4.8	21.6	3.6	30.9	6.1
6	-5.2	3.2	14.6	5.1	21.5	3.8	30.7	6.2
7	-4.3	3.1	16.0	5.2	21.2	3.9	30.3	6.1
8	-3.4	3.0	16.9	5.2	20.7	3.9	29.7	6.1
9	-2.6	3.0	17.5	5.1	20.0	3.9	28.9	6.0
10	-1.8	3.0	17.8	5.0	19.2	3.8	28.0	6.0
11	-0.9	3.0	17.9	5.0	18.2	3.8	27.0	6.0
12	-0.1	2.9	17.8	5.1	17.2	3.8	26.0	6.0
13	0.7	2.8	17.6	5.2	16.1	3.8	25.0	6.0
14	1.4	2.8	17.3	5.3	15.0	3.8	23.9	6.0
15	2.0	2.9	16.8	5.4	13.8	3.8	22.7	6.0
16	2.6	2.9	16.1	5.5	12.6	3.7	21.6	5.9
17	3.1	3.0	15.5	5.5	11.4	3.6	20.5	5.9
18	3.5	3.0	14.7	5.4	10.1	3.5	19.2	5.8
19	3.9	3.0	13.9	5.4	8.8	3.5	18.0	5.8
20	4.4	3.0	13.0	5.4	7.5	3.4	16.7	5.8
21	4.8	3.0	12.2	5.4	6.2	3.3	15.5	5.8
22	5.1	3.0	11.4	5.3	4.9	3.2	14.3	5.7
23	5.4	3.0	10.6	5.2	3.6	3.2	13.1	5.8
24	5.8	3.0	9.8	5.2	2.4	3.1	11.9	5.7
25	6.1	3.0	9.1	5.2	1.2	3.1	10.7	5.7
26	6.4	3.0	8.3	5.2	-0.1	3.0	9.5	5.8
27	6.7	3.0	7.6	5.1	-1.3	3.0	8.3	5.8
28	7.0	3.0	6.9	5.1	-2.6	3.0	7.1	5.8
29	7.3	3.0	6.3	5.2	-3.8	3.0	6.0	5.8
30	7.5	3.0	5.6	5.2	-4.9	3.0	4.9	5.8
31	7.7	3.1	5.1	5.3	-6.1	3.1	3.8	5.8
32	8.0	3.1	4.6	5.4	-7.2	3.2	2.7	5.9
33	8.2	3.2	4.1	5.5	-8.3	3.2	1.7	5.9
34	8.5	3.3	3.8	5.6	-9.3	3.3	0.7	5.9
35	8.9	3.3	3.5	5.7	-10.3	3.4	-0.4	5.9

% Gait Cycle	Ankle		Knee		Thigh (Versus Vertical)		Hip	
	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)
36	9.2	3.4	3.3	5.8	-11.3	3.4	-1.4	6.0
37	9.4	3.5	3.2	5.9	-12.2	3.5	-2.3	6.1
38	9.7	3.5	3.2	6.0	-13.2	3.6	-3.2	6.1
39	9.9	3.6	3.3	6.0	-14.0	3.6	-4.0	6.2
40	10.2	3.6	3.5	6.1	-14.8	3.7	-4.9	6.3
41	10.4	3.7	3.8	6.1	-15.6	3.8	-5.6	6.4
42	10.6	3.7	4.2	6.2	-16.3	3.8	-6.4	6.5
43	10.7	3.8	4.8	6.1	-16.9	3.9	-7.0	6.6
44	10.7	3.8	5.5	6.1	-17.4	3.9	-7.6	6.6
45	10.7	3.8	6.3	6.1	-17.9	3.9	-8.1	6.7
46	10.6	3.8	7.3	6.1	-18.3	4.0	-8.6	6.8
47	10.3	3.8	8.4	6.1	-18.6	4.0	-8.9	6.8
48	9.9	3.8	9.5	6.1	-18.9	4.0	-9.2	6.9
49	9.2	3.8	10.8	6.1	-19.1	4.1	-9.5	6.9
50	8.2	3.9	12.2	6.0	-19.2	4.1	-9.6	6.9
51	7.1	4.2	13.8	6.1	-19.2	4.1	-9.6	6.9
52	5.6	4.4	15.6	6.1	-19.0	4.1	-9.5	7.0
53	3.6	4.6	17.6	6.3	-18.6	4.2	-9.1	7.0
54	1.3	4.8	19.7	6.4	-18.1	4.2	-8.7	7.1
55	-1.5	5.0	22.2	6.6	-17.3	4.4	-7.9	7.2
56	-4.3	5.2	24.9	6.7	-16.2	4.5	-7.0	7.3
57	-7.3	5.3	27.9	6.9	-15.0	4.7	-5.9	7.4
58	-10.2	5.4	31.1	7.1	-13.6	4.8	-4.7	7.5
59	-12.9	5.4	34.3	7.2	-12.0	4.9	-3.2	7.5
60	-15.0	5.4	37.6	7.2	-10.4	5.1	-1.6	7.6
61	-16.6	5.4	40.9	7.2	-8.6	5.2	0.1	7.7
62	-17.4	5.5	44.3	7.0	-6.6	5.2	2.0	7.8
63	-17.5	5.6	47.4	6.7	-4.7	5.1	4.0	7.8
64	-17.0	5.7	50.2	6.3	-2.7	5.0	6.0	7.7
65	-16.0	5.6	52.6	6.0	-0.8	4.9	8.1	7.7
66	-14.8	5.5	54.6	5.6	1.3	4.8	10.2	7.6
67	-13.4	5.3	56.2	5.3	3.3	4.7	12.2	7.5
68	-12.0	5.1	57.4	5.1	5.2	4.6	14.2	7.4
69	-10.5	4.9	58.3	5.0	7.1	4.4	16.2	7.3
70	-9.0	4.7	58.6	4.9	8.9	4.3	18.0	7.2
71	-7.5	4.6	58.7	4.9	10.6	4.2	19.9	7.1
72	-6.1	4.5	58.4	4.8	12.3	4.1	21.7	7.0

% Gait Cycle	*Ankle*		*Knee*		*Thigh (Versus Vertical)*		*Hip*	
	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)	Mean (deg)	SD (deg)
73	-4.8	4.5	57.9	4.8	13.9	3.9	23.4	7.0
74	-3.6	4.5	57.0	4.9	15.5	3.9	25.0	6.9
75	-2.4	4.4	56.0	5.1	16.9	3.8	26.5	6.8
76	-1.4	4.3	54.5	5.2	18.2	3.7	27.8	6.8
77	-0.5	4.2	52.8	5.4	19.4	3.7	29.1	6.7
78	0.3	4.1	50.7	5.7	20.4	3.6	30.2	6.7
79	1.0	4.0	48.3	6.0	21.4	3.6	31.2	6.6
80	1.5	3.9	45.6	6.3	22.2	3.6	32.0	6.5
81	1.9	3.8	42.5	6.7	22.8	3.6	32.7	6.5
82	2.1	3.7	39.3	7.1	23.3	3.6	33.2	6.5
83	2.3	3.6	35.7	7.6	23.7	3.6	33.5	6.5
84	2.2	3.5	31.9	8.0	23.9	3.7	33.7	6.4
85	2.1	3.5	27.9	8.3	23.9	3.7	33.7	6.4
86	1.9	3.5	23.8	8.6	23.8	3.7	33.6	6.4
87	1.7	3.5	19.7	8.8	23.6	3.7	33.3	6.5
88	1.5	3.5	15.7	8.8	23.3	3.7	33.0	6.5
89	1.2	3.5	11.9	8.6	22.9	3.7	32.5	6.5
90	0.8	3.5	8.3	8.2	22.4	3.7	32.0	6.5
91	0.5	3.5	5.1	7.6	22.0	3.6	31.5	6.5
92	0.2	3.5	2.6	7.0	21.6	3.6	31.1	6.5
93	0.0	3.5	0.7	6.4	21.3	3.5	30.8	6.4
94	-0.1	3.5	-0.4	6.0	21.1	3.5	30.5	6.4
95	-0.3	3.5	-0.9	5.5	21.0	3.4	30.3	6.4
96	-0.5	3.4	-0.7	5.3	21.0	3.3	30.3	6.4
97	-0.8	3.3	0.2	5.1	21.0	3.3	30.3	6.3
98	-1.0	3.2	1.5	4.9	21.1	3.3	30.3	6.3
99	-1.5	3.1	3.0	4.9	21.2	3.3	30.4	6.3