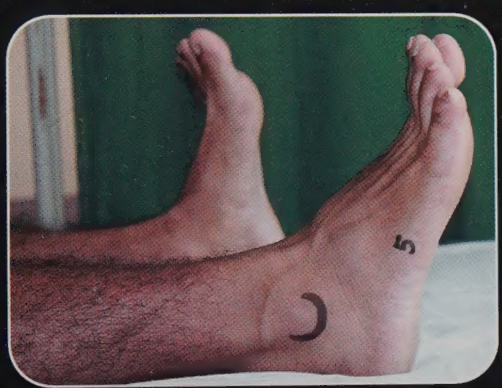
Vascular Rehabilitation



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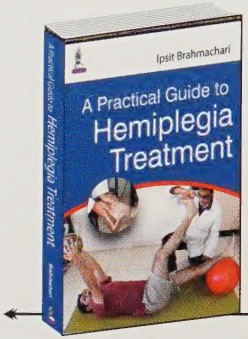

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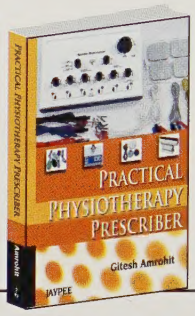

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- Advanced physiotherapy management with dosage, duration of exercise therapy and electrotherapy, Do's/Dont's, home advice, orthotic and prosthetic supports should be kept in mind while writing the book.
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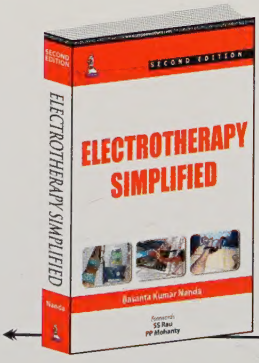

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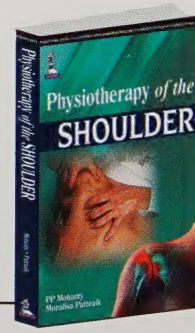
- Electrotherapy simplified thoroughly revised second edition, covers the entire subject, starting from basic electricity and magnetism to theory and practical demonstrations, multiple choice questions and answers in one book.
- Presents the contents of the chapters in simple manner with suitable diagrams which can easily be understood by the students.
- Provides more information about functional electrical stimulation (FES). Includes long-wave diathermy.
- Contains about 50 multiple choice questions with answers.
- Gives adequate information with practical demonstrations.
- The author has utilised his 18 years of teaching experience in the subject to students of Bachelor of Physiotherapy (BPT) for the preparation of this book.

PHYSIOTHERAPY OF THE SHOULDER



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- Enriched with clinical photographs for better understanding.
- Provides guidelines to make appropriate evaluation and assessment of the problem for proper planning of the physiotherapy.
- Useful book for physiotherapy students, clinicians and academicians.

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Vascular Rehabilitation

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Foreword
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Vascular Rehabilitation

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Dedicated to

My parents, SV Solomen and Premila Solomen
My wife, Divya
My children, Angelina and Jerome

Subin Solomen

My students

Pravin Aaron

Foreword

It gives me immense pleasure to write the Foreword for this excellent book *Vascular Rehabilitation*, which, I have no doubt, will be of great help to the students, teachers and practicing physiotherapists. Vascular rehabilitation is an emerging specialized field in physiotherapy, where the students need reference materials on this subject, but sadly there are only a few books and this is such a book. This book provides details on patients having arterial and venous problems with a theoretical basis for clinical practice. It will enhance the physiotherapists' awareness of other aspects of client management and will provide them with a framework to develop evidence-based practice.

Professor Subin Solomen and Professor Pravin Aaron have been associated with Padmashree Institute of Physiotherapy for many years. I must say, as faculties of the institution, they have set standards of excellence in teaching. They are very good therapists who give utmost importance to the patients and their well-being. I congratulate them for this commendable contribution, which has come at a time when universities across the country are deciding to streamline the curriculum.

This book has 15 chapters and is richly illustrated with diagrams, sketches, tables and examples, providing an excellent presentation of the subject. The book, undoubtedly, carries the rich experience of the authors as teachers of the subject and this gets truly reflected in the depth of the information given in this book.

Ashwath Narayan CN
MLA, Malleshwaram Constituency
Managing Trustee
Padmashree Group of Institutions
Bengaluru, Karnataka, India

Foreword

Preface

The World Health Organization (WHO) estimates that between 2000 and 2030, the number of people with diabetes will increase by 114%. Conservative estimates based on population growth and aging, and rate of urbanization in Asia, show that India will have highest number of people with diabetes (79.4 million) by 2030. It is well established that peripheral arterial disease (PAD) is a marker for cardiovascular disease (CVD) and that the risk is greater with more advanced PAD. The prevalence of PAD in diabetics also increases with the duration of diabetes—from 15 to 45% at 10 and 20 years respectively after the diagnosis of diabetes. As per the American Diabetes Association (ADA), 2012, consensus statement on PAD, those with a duration of diabetes more than 10 years have a higher risk for PAD. With an aging population, owing to our longer lifespan, PAD will become more common than its already high prevalence. In this book *Vascular Rehabilitation*, we acquaint physiotherapists with all aspects of peripheral vascular disease. Because of the limitations of medical therapy, there is now a special emphasis on prevention of peripheral vascular disease and a special emphasis is made on the risk factors reduction and their physiotherapeutic treatment.

Chapter 1 discusses the systemic, coronary, pulmonary and lymphatic circulation in brief and anatomy and physiology of artery and vein are also covered. Chapter 2 covers the biomechanics of circulation, i.e. hemodynamics and applied physiology of circulation, is covered. Chapters 3 and 4 cover the epidemiology of peripheral vascular disorders. Epidemiology of a disease includes prevalence, incidence, progression and morbidity and mortality associated with it. We have tried to put maximum literature about it. Chapters 5–7 cover almost all vascular diseases in detail, including the medical and surgical management. Each vascular disorder is described under definition, etiology, types, pathogenesis, clinical features, investigations and management. Chapter 8 covers mainly the assessment, special tests, investigations, outcome measures and findings. In this chapter, investigations and outcome measures are intended for postgraduates. Chapter 9 covers the physiotherapy management, techniques and vascular rehabilitation protocol. Chapter 10 covers arterial, venous and lymphatic surgeries. Chapters 11 and 12 cover physiotherapy after arterial and venous surgeries. Chapters 13 and 14 cover the wound/ulcer assessment and management. Chapter 15 covers about scar assessment and management.

We hope, our hard work will give noteworthy contribution to the teachers and students. Any suggestions or corrections are always welcome.

Subin Solomen
Pravin Aaron

Acknowledgments

First and foremost, I would like to thank Almighty God, without whose blessings *Vascular Rehabilitation* would not have been possible.

I am sincerely obliged to Professor Pravin Aaron, Principal, Department of Physiotherapy, Padmashree Institute of Physiotherapy, Bengaluru, Karnataka, India, who is also the co-author of this book. He helped me in reviewing and correcting the script of this book. It was impossible to deliver this book without him.

I am indebted to my Principal, Jinson K Paul; Dr A Mohammed, Chairman; Sri M Abdunnasir, General Manager of EMS Memorial Co-operative Hospital and Research Centre Ltd, Perinthalmanna, Kerala, India, for providing me support for writing this book. I am indebted to TK Narayanappa, Chairman; Dr Ashwath Narayan CN, Managing Trustee; Dr Vasudeva, Director; and Professor Rajesh Shenoy, Director of Padmashree Group of Institutions, Bengaluru, Karnataka, India, where I did my graduation and worked as a lecturer and as a professor, for their support and cooperation in writing this book.

I am thankful to Shri Jitendar P Vij (Group Chairman) and Mr Ankit Vij (Group President) of M/s Jaypee Brothers Medical Publishers (P) Ltd., New Delhi, India, and the whole team members, for publishing the book in a nice manner. I have taken a lot of information from the books and articles published on vascular diseases, which I have mentioned in the last as 'Bibliography' (articles and books separately mentioned). I acknowledge all the authors who have written on the topics of vascular diseases—both published and unpublished—as I have borrowed information and ideas from those books and articles.

I specially thank my friends, Jimshad and Toms, for their support and cooperation towards the successful completion of the book. I also thank Dr Jewel, Mr Sreejith and Mr Chacko, faculties of the EMS Memorial Co-operative Hospital and Research Centre Ltd, Perinthalmanna, Kerala, India, for helping me to take photographs; and, my relatives, Mrs Sheela Simon and Mrs Laly John; and student, Mr Ajul Siddhi, for providing their photographs.

My thanks also go to my students—Mrs Trupti, Ms Anuradha, Ms Bandana, Ms Anamika, Ms Dhara, Ms Anisha, Ms Barnali, Ms Vishnu, Ms Aaditi, and Ms Rohini, who all helped me in completing the book in the desired format. Last but not least, my thanks go to my wife, Divya, who has continuously encouraged and supported me in writing and completion of the book for the last 10 years.

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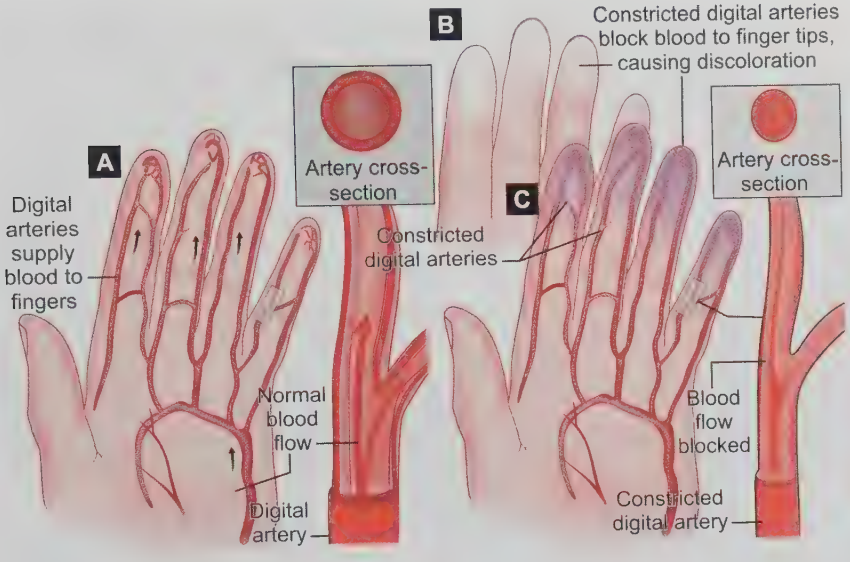

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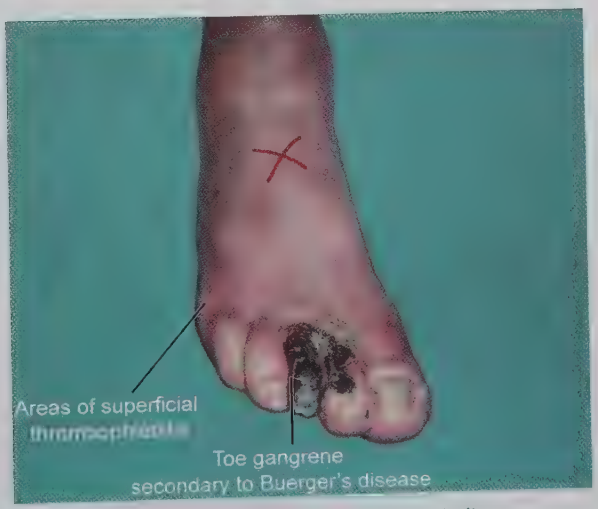
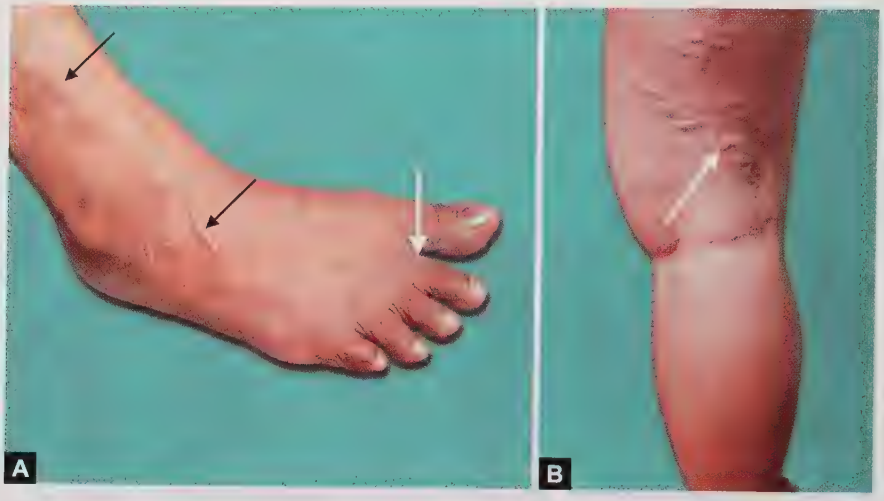


Fig. 5.5.1 A patient's leg with Buerger's disease

PLATE 2



Figs 6.3.3A and B (A) Varicose vein on female's leg, black arrows: dilated vein; white arrow: skin changes; (B) Varicose vein on female's thigh, white arrow: spider vein

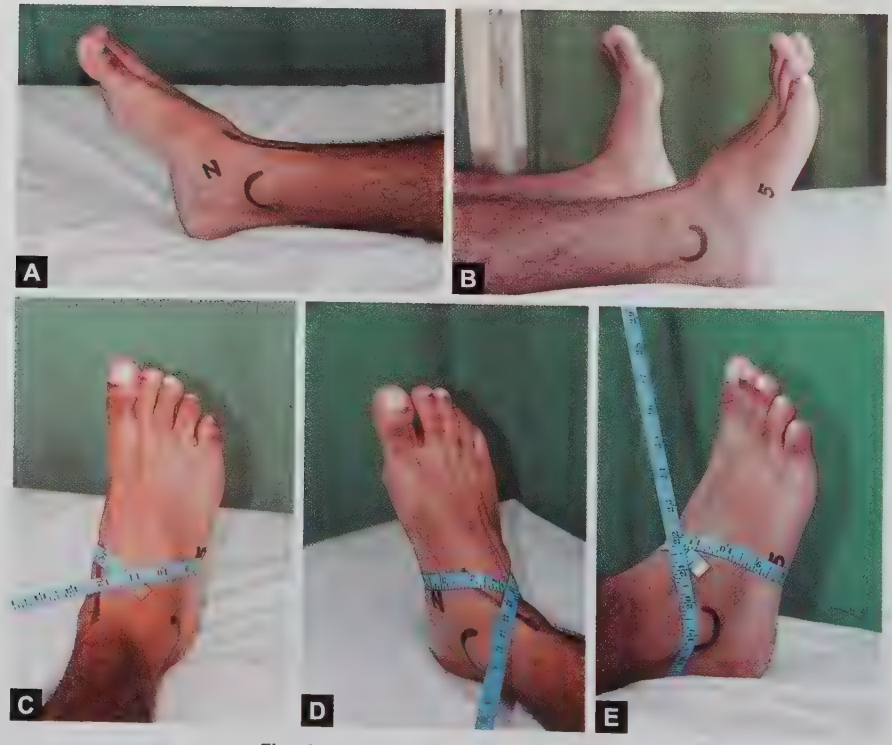


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PLATE 3

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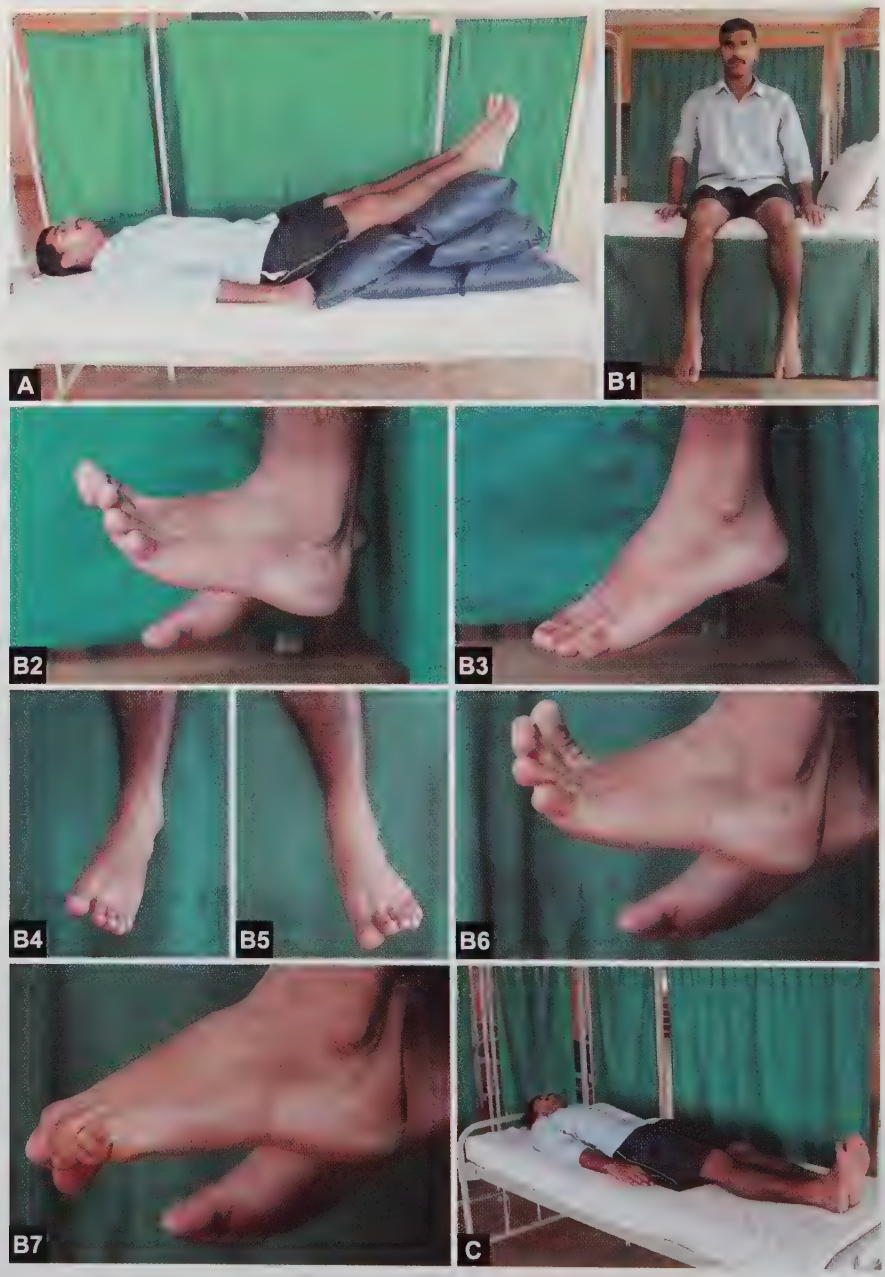


Figs 9.2.1A to C Buerger's exercise. (A) Patient is lying in supine position, legs 45° elevated; (B1) Patient in high sitting position: Performing; (B2) Ankle dorsiflexion; (B3) Ankle plantar flexion; (B4) Inversion; (B5) Eversion; (B6) Dorsiflexion of ankle and dosiflexion of toes; (B7) Plantar flexion of ankle with plantar flexion of toes; (C) Patient is lying in supine position

1 Anatomy of Blood Vessels

INTRODUCTION

Blood and lymph flows through circulatory system and lymphatic system respectively. The circulatory system consists of the heart and blood vessels through which the blood circulates.

The lymphatic system consists of lymph nodes, lymph organs and lymph vessels, through which colorless lymph flows. It consists of central and peripheral lymphoid system. Central lymphoid system includes thymus and bone marrow and peripheral includes lymph nodes, spleen, lymph vessels, etc.

COMPONENTS OF CIRCULATORY SYSTEM (FLOW CHART 1.1)

- *Systemic circulation* ***(Fig. 1.1)***: The blood pumped out from the left ventricle is carried by the branches of the aorta around the body and is returned to the right atrium of the heart by the superior and inferior vena cava.
- *Pulmonary circulation*: This consists of the circulation of blood from the right ventricle of the heart to the lungs and back to the left atrium. In the lungs, carbon dioxide is excreted and oxygen is absorbed.
- *Coronary circulation*: The blood is circulated through coronary arteries, which is a branch of ascending arteries and supplies the heart itself and returned to right atrium. Compare to other circulation heart receives its blood supply during diastole.

BLOOD VESSELS

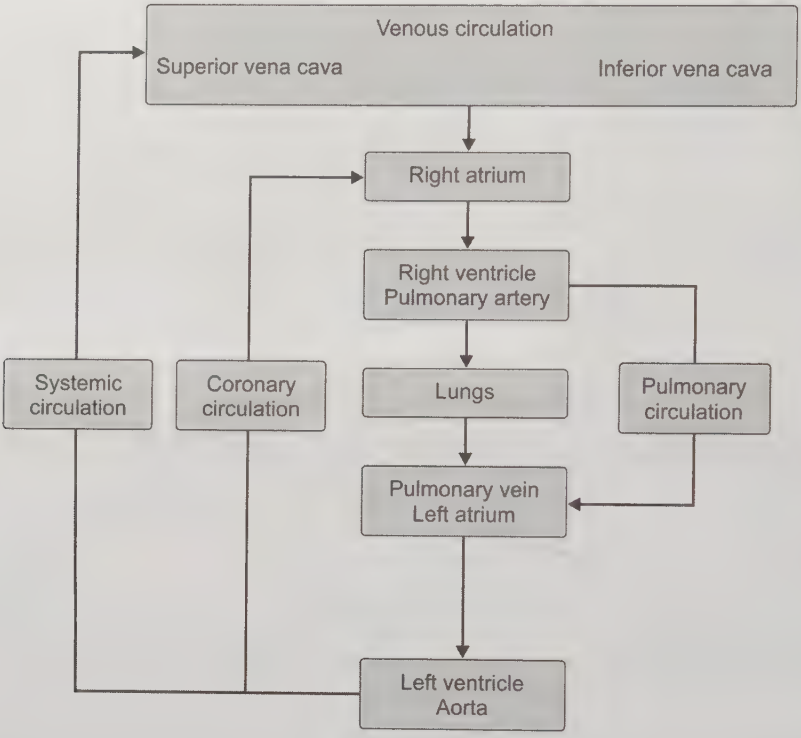
The heart pumps blood into vessels that vary in structure, size and function, and there are several types of blood vessels—arteries, arterioles, capillaries, venules and veins **(Table 1.1)**.

Arteries and Arterioles

Arteries and arterioles are the blood vessels that transport blood away from the heart. They vary considerably in size and their walls consist of three layers of tissue:

- *Tunica adventitia* or outer layer of fibrous tissue

Flow chart 1.1 Circulatory system



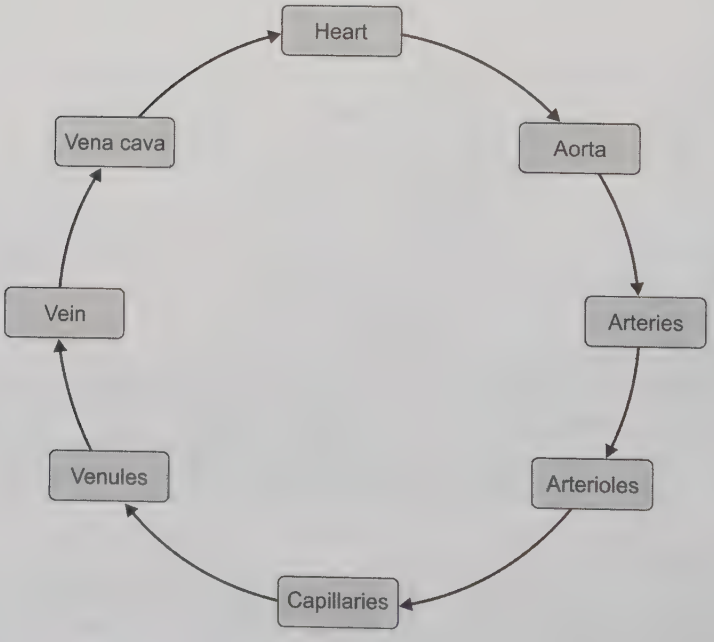


Fig. 1.1 Systemic circulation

Table 1.1 Pathway of blood from periphery to the heart

Blood vessel	*Function*
Capillary	Exchange of substance between blood and tissue fluid
Venule	Connects capillary bed to venules
Superficial vein	Primary collecting veins in extremity
Perforating vein	Connect superficial veins to the deep veins
Deep veins	Carry blood from perforating and superficial veins to vena cava
Vena cava	Carries blood from periphery to right atrium of heart

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- *Tunica media* or middle layer of smooth muscle and elastic tissue
- *Tunica intima* or inner lining of squamous epithelium called *endothelium*

Arterial walls are thicker because of more smooth muscles compared to veins. In the large arteries, elastic arteries, the tunica media consists of more elastic tissue and less smooth muscle. In the arteries, the tunica media consists almost entirely of smooth muscle **(Fig. 1.2)**.

Anastomoses and End-arteries

Anastomoses are arteries that form a link between main arteries supplying an area, e.g. the arterial supply to the palms of the hand and soles of the feet, the brain, joints and to a limited extent, the heart muscle. When there is a block in the arteries, these arteries ensures the circulation is bypassed through a collateral circulation. End-arteries are the arteries with no anastomoses or those beyond the most distal anastomoses, e.g. the branches from the circulus arteriosus (circle of Willis) in the brain or the central artery to the retina of the eye. When an end-artery is occluded the tissues it supplies die because there is no alternative blood supply.

Capillaries

The smallest arterioles break up into a number of minute vessels called capillaries. Blood cells and large-molecule substances such as plasma proteins do not normally pass through capillary walls. The capillaries form a vast network of tiny vessels, which link the smallest arterioles to the smallest venules **(Fig. 1.3)**. The capillary bed is the site of exchange of substances between the blood and the tissue fluid, which bathes the body cells.

Venules

Venules connect the capillaries (arterial system) to the venous system. Venules are very small in diameter and oval-shaped at rest. When pressures increase, they become more circular. While blood vessels typically have three distinct layers, the end venules have only an endothelial layer with a thin layer of collagen fibers (tunica intima).

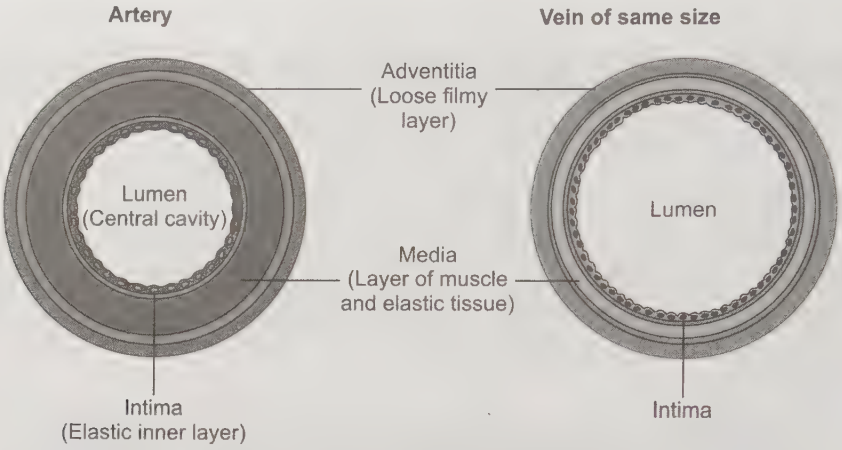


Fig. 1.2 Histology of artery and vein

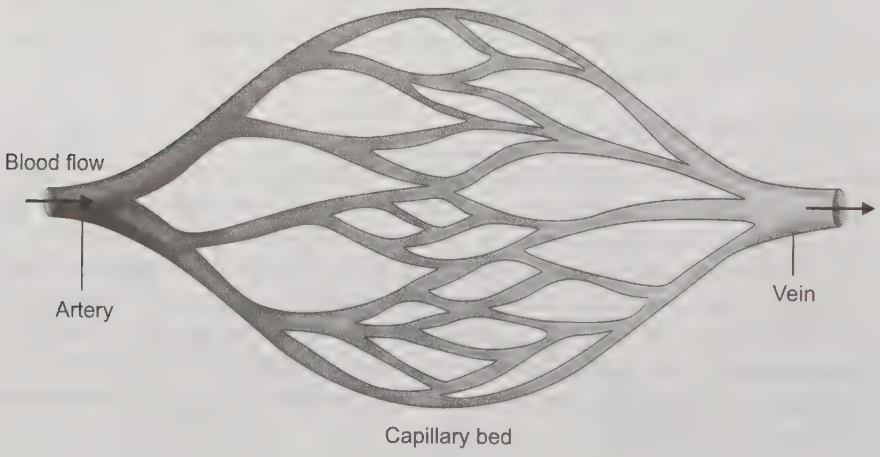


Fig. 1.3 The artery-capillary-vein system

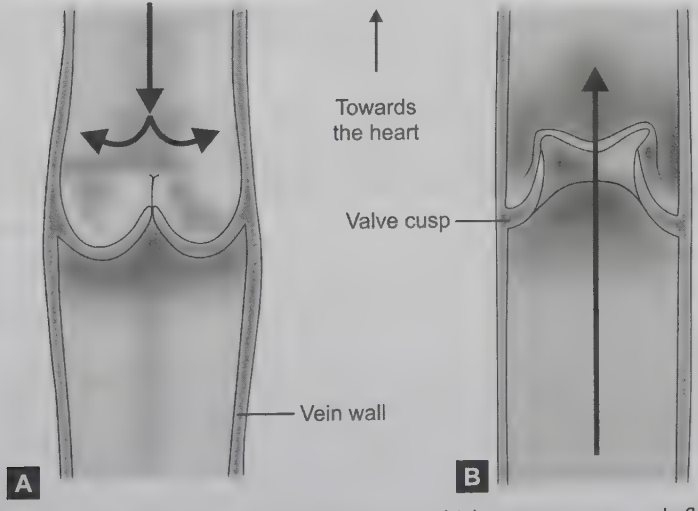
Veins

As venules increase in diameter and become visible veins, they have the typical three layers mentioned earlier. Veins in the upper extremities and lower extremities are superficial and deep. The two superficial vein extremities are of upper extremities are cephalic and basilic vein. The deep veins are named corresponding to the accompanying arteries or arterial system at the similar level of the vascular system. The *basilic vein* originates from the dorsal venous network of the hand. It ascends the medial aspect of the upper limb. At the border of the *teres major*, the vein moves deep into the arm. Here, it combines with the *brachial veins* to form the axillary vein. The cephalic vein arises from the *dorsal venous network* of the hand. It ascends the anterolateral aspect of the upper limb, passing anteriorly at the elbow. At the shoulder, the cephalic vein

travels in the deltopectoral groove and enters the axilla. Within the axilla, the cephalic vein terminates by joining the *axillary vein*. In cubital fossa, they are connected by median cubital vein, which is common site of intravenous (IV) injection. Another site for IV is dorsal venous arch of hand. Median cubital vein is commonly affected with superficial thrombophlebitis and dorsal venous arch is a common site for cellulitis.

There are three different types of lower extremity veins, which are superficial, perforating, and deep veins. The superficial veins conduct blood from the skin and subcutaneous tissue. The perforating veins connect the superficial veins to the deep veins that convey blood from the periphery to the heart. One anatomical structure the perforating, superficial and deep veins have in common is valves. The superficial veins have fewer valves than the deeper veins. Vein valves are bicuspid and avascular. They consist of thin sheets of collagen and smooth muscle with an endothelial covering. Valves appear to become less flexible as people age. The valves prevent retrograde blood flow, thus allowing veins to overcome gravity effects **(Figs 1.4A and B)**.

The main, superficial leg veins include the greater saphenous, lesser saphenous, and the lateral (subdermis) venous system. Descriptions of saphenous veins are listed in **Table 1.2** and their difference is given in **Table 1.3**. These veins are very thin walled and distensible. The superficial veins lie above the main fascia plane and are the primary blood collection system for the lower leg. They lack the extensive fascial restriction experienced by the deep veins. Consequently, superficial veins may undergo dramatic volume changes or distention. The lateral venous system (lateral subdermic) above/below the knee is a common area for varicosities during pregnancy and occasionally puberty. The superficial veins deliver blood to the deep veins, such as the femoral and



Figs 1.4A and B (A) Valve in a closed position which prevents retrograde flow; (B) Valve in open position which allows proximal flow

Table 1.2 Main superficial veins of lower extremity

Veins	*Anatomical locations*	*Concerns*
Great saphenous vein	Starts at medial foot, above malleolus; goes to anterior, medial calf, knee, medial thigh. Ends at common femoral vein	Often site of varicose veins in lower thigh and upper calf
Lesser or short saphenous vein	Starts at lateral foot and posterior/ lateral malleolus, goes up posterior/ lateral *or Short*) leg below knee, between gastrocnemius heads into popliteal fossa. Connects (lateral calf, lower 1/3 of leg behind the lateral malleolus)	Reflux usually where this vein and tributaries are superficial to fascia
Lateral venous (sub dermic system)	On lateral aspect of leg; above and below the knee	Common area for isolated spider veins in young women

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Table 1.3 Description of great saphenous and small saphenous vein

Features	*Great saphenous vein*	*Small saphenous vein*
Synonym	Long saphenous vein	Short saphenous vein
Origin	By joining medial end of dorsal venous arch and dorsal vein of great toe	By joining lateral end of dorsal venous arch and dorsal vein of little toe
Course: At ankle Leg Knee Thigh	 Anterior to medial malleolus Anterior (tibial border) Behind knee Medial side of the thigh	 Posterior to lateral malleolus Posterior (along the border of tendo-achilles)
Termination	To femoral vein in femoral triangle	To popliteal vein in popliteal fossa

Table 1.4 Perforating veins of lower extremity

Vein	*Location*
Hunterian	Mid to upper medial thigh
Dodd	Proximal, medial knee, distal medial thigh
Boyd's	Lower medial calf
Cockett's	Posterior medial ankle; posterior arch of foot

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popliteal veins. However, the superficial system also connects to perforating veins.

The perforating veins penetrate fascia and connect the superficial venous system to the deep veins (**Table 1.4**). Muscle contraction produce pressure that assists blood movement from perforators into the deep veins (muscle pump). Perforating veins have valves, which prevent retrograde blood flow, i.e. back to superficial. The lower leg and foot have more perforating veins than the upper leg.

In the legs, deep veins often run parallel to superficial veins. The perforating veins connect superficial veins to deep veins like slanted rungs of a ladder. The deep veins are named corresponding to the accompanying arteries or arterial system at the similar level of the vascular system.

Functions of Vein

- To carry blood to the heart/lungs for gas, nutrient and waste exchange.
- Storage of large blood volume.
- Veins are barriers between intravascular and extravascular tissues. The proximal venules allow movement of interstitial fluids, large molecules and white blood cells through the venule wall.
- Vein walls also have cellular functions. White blood cells attach to vein endothelium in order to be available should there be an injury or disease process.
- Veins also are a factor in cardiovascular pressures. In the capillary bed, veins influence arterial output resistance. Vein walls produce nitric oxide that causes vasodilation via decreased vascular tone.
- Veins also have a role in heart filling pressure. The skeletal muscle pump, venous myogenic response and venous smooth muscle tone prevent orthostatic hypotension.
- The venules also appear to be a site of angiogenesis.
- Venous endothelium has a role in lessening platelet aggregation to prevent clot development via the formation of prostacyclin.
- *Other unique or regional functions:*
  - Facial veins allow for blushing and temperature regulation of the head.
  - Cutaneous veins help regulate body/skin temperature.
  - The internal jugular vein facilitates cranial pressure regulation.
  - Proximal portion of the vena cava and pulmonary veins may play a role in cardiac pacing. Their tunica media layers have cardiac myocytes that might provide cardiac pacing during some pathological conditions.

Mechanism, Which Helps in Back Flow of Blood from the Legs to the Heart

- *Pressure from behind*: The slight pressure pushing blood onwards from the capillaries into the venules. Although this is sufficient when we lie down, it is totally inadequate when we are standing or walking.
- *Suction effect of the lungs*: When we take a deep breath a negative pressure, rather similar to a vacuum, is created in the chest that helps to draw blood upwards to the heart.
- *Pumping action of the leg muscles*: These muscles are enclosed in a dense sheath of fibrous tissue (called the deep fascia). When you walk about, the muscles contract and the veins contained in the fibrous sheath are squeezed.
- *Valves in the deep vein*: At intervals along the insides of the veins are beautifully constructed valves, consisting of two flaps that meet each other exactly. Though very simple they are quite sufficient nevertheless to

make sure that the blood can be squeezed in one direction only back to the heart.

- *Valves in the perforating vein*: The veins that drain these superficial tissues are linked up with the deep veins through a number of perforations in the fibrous sheath. Each linking or communication vein is guarded by a one-way valve. As we walk, the muscle pump squeezes the deep veins in our legs; blood is pushed upwards towards the heart; and the negative pressure within the deep veins sucks in blood through the communicating veins from the skin and the fat.
- Controlled venous diameter, high pressure on the arterial side, and the low right atrium pressure that draws blood from the great veins.

LYMPHATIC CIRCULATION (FIG. 1.5)

In the interstitial space, majority of the tissue fluid drains to their venous end whereas the remainder diffuses through permeable walls of the lymph capillaries and becomes lymph. Lymph is a clear watery fluid, similar in composition to blood plasma, with the important exception of plasma proteins and identical in composition to interstitial fluid.

The lymphatic system consists of:

- Lymph
- Lymph vessels
- Lymph nodes
- Lymph organs, e.g. spleen and thymus
- Diffuse lymphoid tissue, e.g. tonsils
- Bone marrow.

The lymphatic system function are to drain tissue fluid, plasma proteins and other cellular debris back into the bloodstream, and is also involved in immune defense. Fat and fat-soluble materials, e.g. the fat-soluble vitamins, are absorbed into the central lacteals (lymphatic vessels) of the villi.

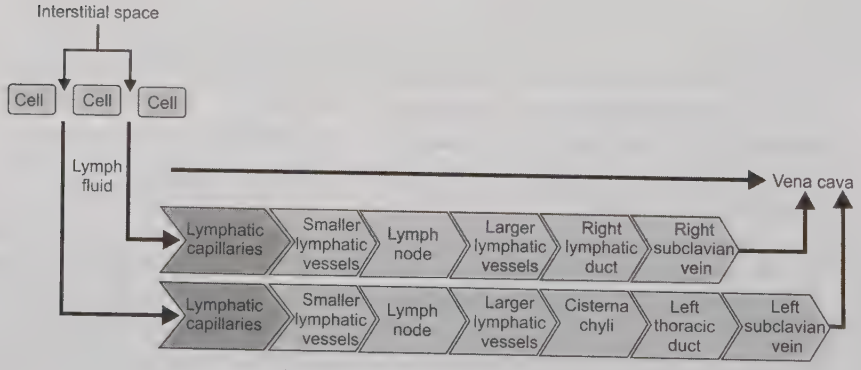


Fig. 1.5 Lymphatic circulation

Lymph Capillaries

Lymph capillaries originate as blind-end tubes in the interstitial spaces. They have the same structure as blood capillaries, i.e. a single layer of endothelial cells, but their walls are more permeable to all interstitial fluid constituents, including proteins and cell debris. The tiny capillaries join up to form larger lymph vessels.

Larger Lymph Vessels

The walls of lymph vessels are about the same thickness as those of small veins and have the same layers of tissue, i.e. a fibrous covering, a middle layer of smooth muscle and elastic tissue and an inner lining of endothelium. Lymph vessels have numerous cup-shaped valves, which ensure that lymph flows in one way only, i.e. towards the thorax. There is no 'pump', such as the heart, involved in the onward movement of lymph, but the muscle tissue in the walls of the large lymph vessels has an intrinsic ability to contract rhythmically (the lymphatic pump). In addition, any structure that periodically compresses the lymphatic vessels can assist in the movement of lymph along the vessels, commonly including the contraction of adjacent muscles and the pulsation of large arteries. Lymph vessels are divided into superficial and deep lymphatic vessels. Lymph vessels become larger as they join together, eventually forming two large ducts, the thoracic duct and right lymphatic duct, that empty lymph into the subclavian veins.

Thoracic Duct

Thoracic duct begins at the cisterna chyli, which is a dilated lymph channel situated in front of the bodies of the first two lumbar vertebrae. The duct is about 40 cm long and opens into the left subclavian vein in the root of the neck. It drains lymph from legs, the pelvic and abdominal cavities, the left half of the thorax, head and neck, and the left arm.

Right Lymphatic Duct

Right lymphatic duct is a dilated lymph vessel about 1 cm long. It lies in the root of the neck and opens into the right subclavian vein. It drains lymph from the right half of the thorax, head and neck and the right arm.

Lymph Nodes

Lymph nodes are oval or bean-shaped organs that lie, often in groups, along the length of lymph vessels. The lymph drains through a number of nodes, usually 8–10 nodes, before returning to the venous circulation. Function of lymph nodes includes filtering, phagocytosis and proliferation of lymphocytes.

2 Biomechanics of Circulation

CLASSIFICATION OF CIRCULATION

Circulation is divided into major circulation and minor circulation.

Characteristics of Major Circulation

Systemic circulation begins from the left ventricle and derives from the aorta. It contains renal, coronary and mesenteric (splanchnic) circulation. The blood flow is autoregulated in many regions. The blood pressure of arterial circulation is eight times higher than that of pulmonary circulation. This is because of two reasons. The total length of arteries in systemic circulation is much bigger than pulmonary circulation. Also the diameter of pulmonary vessels is bigger than that of arterial system. Blood flow autoregulates in many regions.

Characteristics of Minor Circulation

Pulmonary circulation begins from right ventricle and derives from pulmonary artery. Compared to systemic circulation, blood pressure of pulmonary circulation is low because of above mentioned reasons. Autoregulation does not play a major role in this circulation.

Role of Cardiovascular System

- Transportation of blood
- Distribution of nutrients
- Chemical signaling
- Heat dissipation
- Host defence.

ANATOMIC AND FUNCTIONAL CLASSIFICATION

Anatomic names for the vessels according to dimension and position is already described in Table 1.1. Here the functional classifications of vessels are described **(Table 2.1)**.

Table 2.1 Anatomic and functional classification

Anatomic classification	*Functional classification*
Heart	Pump
Large elastic arteries, muscular arteries	Distribution vessels
Arterioles, precapillary sphincters	Resistance vessels
Capillary, postcapillary venules	Exchange vessels
Venules, veins	Capacitance vessels
Vena cava	Variable high volume, low pressure reservoir

Pump: The heart act as a pump, which provides the force to circulate the blood through it.

Distribution vessels: Elastic and muscular arteries form the distribution vessels. Elastic tissue present in the layer of arteries serves to change the intermittent contractions of the heart into a smoother flow of blood. The smooth muscle present in the arteries allows controlled contraction or relaxation of these muscular walls permits the blood flow to other organs in accordance with their need.

Resistance vessels: Muscles present in their wall of arteries contract, which is the primary source of peripheral resistance to blood flow. This resistance together with cardiac output determine the arterial blood pressure.

Exchange vessels: Capillaries and postcapillary venules are called so because exchange of gases, nutrients, metabolic products occurs across the walls of the capillaries.

Capacitance vessels: Larger venules and veins are called so because they can hold large amount of blood volume.

Reservoir: Vena cava are called reservoir because they store the blood and send back to right atrium.

CLOSED CIRCUIT

Some animals have open circulatory system, compared to that humans have closed circulatory system. In open circulatory system, blood is not contained within an enclosed circuit of vessels and they are more free—flowing and passive. The blood of a closed system always flows inside vessels. They are more structured and controlled.

HEMODYNAMICS

Hemodynamic is also called rheology of blood; rheology is the science, which deals with flow of blood. It refers to principles that govern blood flow in the cardiovascular system therefore concepts of flow pressure resistance and capacitances apply to blood flow to and from the heart within blood vessels.

Hemodynamic Principles

Velocity of Blood Flow

It is important to distinguish between velocity, which is displacement per unit time (cm/s), and flow, which is volume per unit time (cm^3/s):

$$V = Q/A$$

where,

V = velocity of blood flow, which is linear velocity and refers to the rate of displacement of blood per unit time (cm).

Q = flow that is volume flow per unit of time (mL). In cardiovascular system, this is equal to cardiac output.

A = area that is the correct as cross-sectional area of a blood vessel or group of blood vessels (cm^2).

If flow is kept constant as 10 mL/sec, the velocity reduces as the diameter increases **(Table 2.2)**.

Relationship between Velocity and Diameter

Velocity is inversely proportional to the diameter of the blood vessels. So, velocity is more in aorta, but less in capillaries.

Interrelationship between Pressure and Flow

Inter relationship of pressure and flow can be explained by Hagen-Poiseuille's law, which is as follows:

$$\text{Flow, } Q = (P_1\text{-}P_2)\pi r^4/8\eta l$$

where,

P_1 and P_2 are pressure at both ends of a tube.

r is the radius.

η is the viscosity.

l is the length of the tube.

- A slight dilation of blood vessels (r increases) results in tremendous increase in the flow (increases by four folds). In case of muscular exercise blood pressure (BP) increases, P1 increases the blood flow is also increased.

Interrelationship of Pressure and Velocity

In order to understand this relationship, the lateral pressure and total pressure has to be differentiated. The lateral pressure is exerted laterally against the walls

Table 2.2 Effect of the diameter of the blood vessel on the velocity and blood flow

Diameter			
Area	1 cm^2	10 cm^2	100 cm^2
Flow (constant)	10 mL/sec	10 mL/sec	10 mL/sec
Velocity	10 cm/sec	1 cm/sec	0.1 cm/sec

of the vessel and the total pressure is the pressure that determines the volume of blood flowing to an organ. Bernoulli principle interrelates between total pressure, lateral pressure, viscosity and velocity of the blood flowing through the tube:

$$E = P + 0.5\ \rho v^2$$

where,

E = total pressure
P = lateral pressure
ρ = density
v = velocity of the blood.

In case of large diameter vessels, the velocity is high so the lateral pressure is almost equal to total pressure. In case of small diameter vessels the velocity is less so, total pressure is more than lateral pressure.

Interrelationship of Pressure and Tension

Interrelationship of pressure and tension in the ventricles can be explained by Laplace law. This law states that tension in the wall of a cylinder (T) is equal to the product of the transmural pressure (P) and the radius (r) divided by the wall thickness (w):

$$T = Pr/h$$

where,

T is the wall tension in the ventricles.
P is intraventricular pressure.
r is radius.
h is the chamber thickness.

The law of Laplace also makes clear a disadvantage faced by dilated hearts. When the radius of a cardiac chamber is increased, a greater tension must be developed in the myocardium to produce any given pressure; consequently, a dilated heart must do more work than a nondilated heart.

Even interrelationship of pressure and tension in the blood vessels can be explained by Laplace law. The smaller the radius of a blood vessel, the lower the tension in the wall is necessary to balance the distending pressure. The principal reason for their relative invulnerability for not prone to rupture even though they are thin walled and delicate is their small diameter.

APPLICATION OF OHM'S LAW TO CIRCULATION

Blood always flows, of course, from areas of high pressure to areas of low pressure, except in certain situations when momentum transiently sustains flow. The relationship between mean flow, mean pressure and resistance in the blood vessels is analogous in a general way to the relationship between the current, electromotive force, and resistance in an electrical circuit expressed in Ohm's law ($I = \Delta V/R$):

$$\text{Ohm's law } (Q) = \Delta P/R$$

where,

Q = flow (mL/min).
ΔP = pressure difference (mm Hg).
R = resistance (mm Hg/mL/min).

- Blood flow is directly proportional to the pressure gradient
- Blood flow is inversely proportional to the resistance:
  - Resistance is directly proportional to the vessel length.
  - Resistance is inversely proportional to the radius.

It is often useful to use electrical analogies and symbols for fluid variables. A table of such corresponding variables is presented in **Table 2.3**.

Resistance to Blood Flow

Blood vessels and blood itself constitute resistance to blood flow. The relationship between resistance, blood vessel diameter (or radius) and blood viscosity is described by the Poiseuille's equation:

$$R = \frac{8\,\eta l}{\pi r^4}$$

where,

R = resistance.
η = viscosity of blood (directly proportional to resistance).
l = length of blood vessel (directly proportional to resistance).
r^4 = radius of blood vessel raised to the fourth power (inversely proportional to resistance).

Resistance in Series (Flow chart 2.1)

Total resistance of system arranged in series is equal to sum of individual resistance.

$$R_T = R_1 + R_2 + R_3 + R_4$$

In human circulation:
Total systemic circulation resistance (R_T) = R_1/artery + R_2/arterioles + R_3/capillary + R_4/vein + R_5/venule.

In heart:
R_1/right atrium + R_2/tricuspid valve + R_3/right ventricle + R_4/pulmonary valve + R_5/pulmonary vascular resistance + R_6/left atrium + R_7/mitral value + R_8/left ventricle.

The right and left heart are said to be arranged in series or in line, one after the other, right to left.

Table 2.3 Ohm's law

Fluid variable	Electrical variable
Pressure, P	Voltage, e
Flow, Q	Current, i
Volume, V	Charge, q
Resistance, $R = \Delta P/Q$	Resistance, $R = \Delta e/i$
Capacitance, $C = \Delta V/\Delta P$	Capacitance, $C = \Delta q/\Delta e$

If they are not in series, following consequences can happen:

- If output of left exceed than right by as little as 2%, the whole pulmonary circulation would have been drained in less than 10 minutes.
- If right heart output exceed than left by a similar amount, the pulmonary circulation would overflow and a person drowns with his/her own body fluids.

Normally, the above mechanisms never happen in a healthy person.

The series arrangement of the right and left heart also implies that malfunctions in the left heart will be transmitted back into the pulmonary circulation and the right heart, potentially causing the respiratory system to malfunction. Indeed, one of the first clinical signs of left heart failure is respiratory distress. Conversely, problems originating on the right side of the circulation, affect the output of the left heart and risk the blood supply to all systemic organs. Large blood clots can form in the major veins of the leg and abdomen following surgery. These clots can break away and slip through the tricuspid valve into the right ventricle and then into the pulmonary artery, where they eventually lodge, to form what is called pulmonary embolus. Pulmonary emboli compromising about 75% of the pulmonary circulation can block enough flow into the left heart to kill a person.

Resistance in Parallel (Refer Flow chart 2.1)

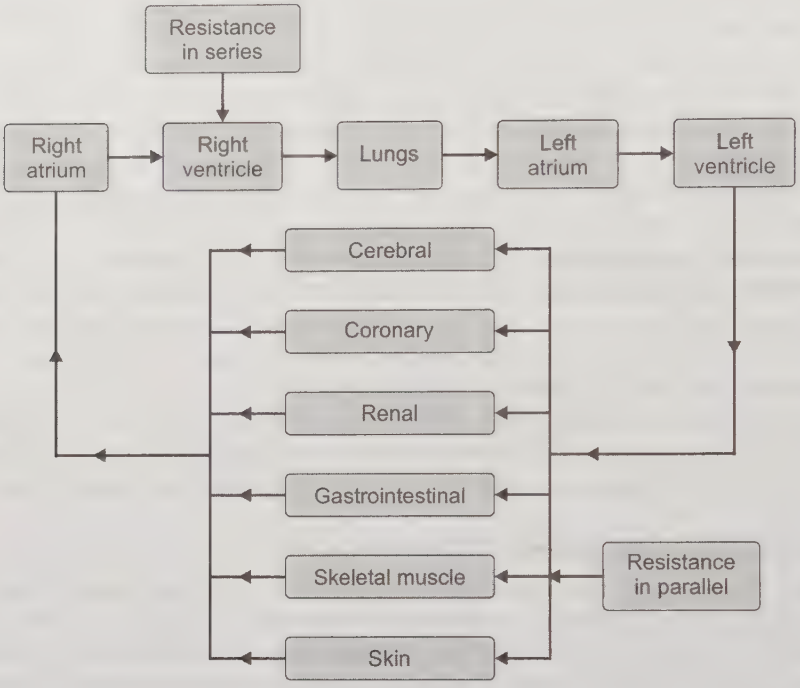
Blood vessels branch extensively to form parallel circuits that supply blood to the many organs and tissues of the body. This parallel arrangement permits each tissue to regulate its own blood flow, to a great extent, independently of flow to other tissues. For blood vessels arranged in parallel, the total resistance to blood flow is expressed as:

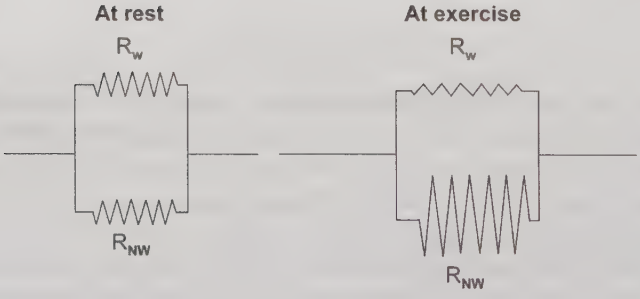
$$\frac{1}{R_{total}} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3}$$

It is obvious that for a given pressure gradient, far greater amounts of blood will flow through this parallel system than through any of the individual blood vessels. Therefore, the total resistance is far less than the resistance of any single blood vessel. Flow through each of the parallel vessels is determined by the pressure gradient and its own resistance, not the resistance of the other parallel blood vessels. However, increasing the resistance of any of the blood vessels increases the total vascular resistance. It may seem paradoxical that adding more blood vessels to a circuit reduces the total vascular resistance. Many parallel blood vessels, however, make it easier for blood to flow through the circuit because, each parallel vessel provides another pathway or conductance, for blood flow. The total *conductance* (C total) for blood flow is the sum of the conductance of each parallel pathway.

For example, brain, kidney, muscle, gastrointestinal, skin and coronary circulations are arranged in parallel, and each tissue contributes to the overall conductance of the systemic circulation. Blood flow through each tissue is a fraction of the total blood flow (cardiac output) and is determined by the resistance (the reciprocal of conductance) for blood flow in the tissue, as well as the pressure gradient. Therefore, amputation of a limb or surgical removal of a kidney also removes a parallel circuit and reduces the total vascular conductance

Flow chart 2.1 Resistance in series and parallel in human body





R_{NW} = Resistance of nonworking muscle

R_w = Resistance of working muscle

Fig. 2.1 Resistance in parallel at rest and during exercise

as well as total blood flow (i.e. cardiac output), while increasing total peripheral vascular resistance. Total resistance is less than of the individual resistance.

Resistance are arranged in parallel between exercising muscles and non-exercising muscles **(Fig. 2.1)**. At rest both of the resistance are same, but during exercise resistance of exercising muscle are reduced and resistance of non-exercising muscles is increased. That means when lower limb exercises are performed there is reduced resistance of blood vessels and increased circulation to the lower limb and in upper limb there is increased resistance of blood vessels

and reduced circulation to upper limbs. During exercise sympathetic system increases all the resistances whereas the muscle activity and potassium dilates blood vessels of lower limb muscles thereby reducing resistance. Total resistance is always less than of the individual resistances, when it is arranged in parallel. That is why the total peripheral resistance is reduced even though all resistances are increased except resistance of exercising muscles.

Blood flow to individual organs can be controlled primarily independently because circulations to individual organs are arranged in parallel. The arterial system delivers blood to organ systems that are arranged in a parallel, or side-by-side, network. Therefore, in most cases, blood flow into one organ system is not dependent on blood flow through another organ upstream. The metabolic demands of our muscles, digestive system, brain, etc. may be different relative to one another and relative to their own resting values depending on the activity in the organ at a given time. The parallel arterial distribution system of organ blood supply allows adjustment of blood flow to an individual organ to meet its own needs without creating major disturbances in the blood supply to other organs. A notable exception to this arrangement, however, is seen in the portal circulation. Venous outflow from the intestines and other splanchnic organs drains into the liver through the portal vein before being emptied into the inferior vena cava (IVC). The liver obtains blood from the portal vein as well as its own arterial supply and can be considered to be arranged in series with much of the splanchnic circulation.

TYPES OF FLOW (LAMINAR VS TURBULENT FLOW)

Laminar Flow (Fig. 2.2)

The flow of blood in straight blood vessels, such as the flow of liquids in narrow rigid tubes, is normally laminar. Within the blood vessels, an infinitely thin layer of blood in contact with the wall of the vessel does not move. The next layer within the vessel has a low velocity, the next a higher velocity and so forth,

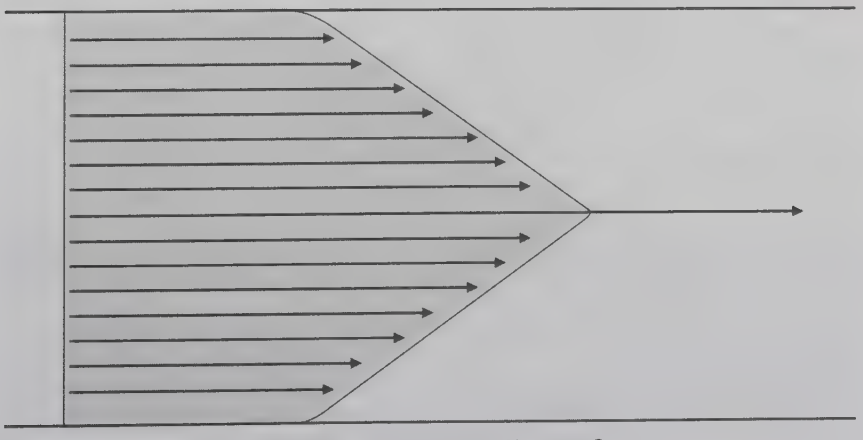


Fig. 2.2 Longitudinal view of laminar flow

velocity being greatest in the center of the stream. In summary in laminar flow, blood moves with different velocity and innermost layer have highest velocity. The velocity profile of lamina takes a paraboloid shape. When a fluid takes a laminar flow, it is called Newtonian fluid. According to Newton, center most lamina moves forward and drags its adjacent lamina. Thus there is a shearing force which causes the lamina to move forward. Viscosity is the lack of slipperiness in between the adjacent lamina. Higher the viscosity, greater is the resistance and lesser will be the flow. The two factors which influence the viscosity are hematocrit value and velocity of blood flow. When, red blood cell (RBC) count increases the viscosity increases and the BP increases. In muscular exercise the velocity increases, viscosity reduces and the blood flow increases.

Turbulent Flow (Fig. 2.3)

In turbulent flow, the fluid molecules move in haphazard directions and these molecules collide with one another. The turbulent flow results in noisy flow, bruit, murmur, damage to endothelial lining and thrombus formation.The probability of turbulence is also related to the diameter of the vessel and the viscosity of the blood. This probability can be expressed by the ratio of inertial to viscous forces as follows:

$$Re = \rho DV/\eta$$

where,

Re is the Reynolds number.
ρ is the density of the fluid.
D is the diameter of the tube under consideration.
V is the velocity of the flow.
η is the viscosity of the fluid.

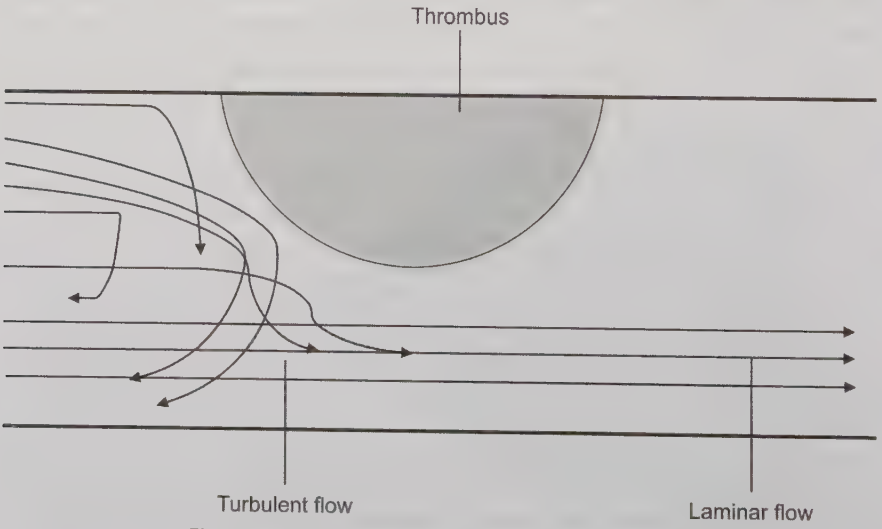


Fig. 2.3 Creation of turbulent flow due to thrombus

The higher the value of Re, the greater will be the probability of turbulence. When Re is more than 3,000, turbulence is almost always present.

Laminar flow can be disturbed at the branching points of arteries and the resulting turbulence may increase the likelihood that atherosclerotic plaques will be deposited. Constriction of an artery likewise increases the velocity of blood flow through the constriction, producing turbulence and sound beyond the constriction.

- In anemia, low hematocrit results in low viscosity in turn results in high Reynolds number and results in turbulence.
- In thrombi, there is a reduced diameter result in increased diameter, which results in increased Reynolds number results in turbulence.
- In case of valvular disorders of heart, flowing fluid hit against the damaged valves results in murmur.

COMPLIANCE OF THE BLOOD VESSELS

In the human body, the veins are an important blood reservoir. Normally, they are partially collapsed and oval in cross-section. A large amount of blood can be added to the venous system before the veins become distended to the point where further increments in volume produce a large rise in venous pressure. The veins are therefore called capacitance vessels. At rest, at least 50% of the circulating blood volume is in the systemic veins. Compliance can be calculated as:

$$C = V/P$$

WINDKESSEL VESSELS AND EFFECT

Windkessel vessels are the aorta and its major branches. The main pulmonary arterial trunk is also a Windkessel vessel. These blood vessels contain elastic fibers. If they are stretched, it elongates, but recoils and regains the original length as soon as the stretch is withdrawn. As heart pumps an additional amount of blood thrust into aorta. So aorta suddenly distends. Aorta stores energy during ventricular systole, which is released by heart during the diastole. Now the aorta recoils and surge forward with reserved blood. Applications are as follows:

- During ventricular diastole although ventricular pressure falls very low, aorta is able to maintain diastole BP of 80 mm Hg.
- During systole it prevents too much rise of pressure.
- In old age arteries stiffen, the Windkessel effect deteriorates, the systolic BP becomes high, but the diastole becomes low.

STARLING'S LAW

The rate of filtration at any point along a capillary depends on a balance of forces sometimes called Starling forces. One of these forces is the hydrostatic pressure gradient (the hydrostatic pressure in the capillary minus the hydrostatic pressure of the interstitial fluid) at that point. The other force is the osmotic pressure gradient across the capillary wall (colloid osmotic pressure

of plasma minus colloid osmotic pressure of interstitial fluid). This component is directed inward. Thus

$$\text{Net fluid movement} = k\,[(P_c - P_i) - (\pi_c - \pi_i)]$$

where,

k = capillary filtration coefficient.

P_c = capillary hydrostatic pressure.

P_i = interstitial hydrostatic pressure.

π_c = capillary colloid osmotic pressure.

π_i = interstitial colloid osmotic pressure.

The osmotic pressure gradient (π_c–π_i) usually equals the oncotic pressure. The capillary filtration coefficient takes into account and is proportional to, the permeability of the capillary wall and the area available for filtration.

FAHRAEUS-LINDQVIST EFFECT

In capillaries where diameter is small the velocity can be less this result in high viscosity. This can cease blood flow in the capillaries. So, because of Fahraeus-Lindqvist effect blood flow is still possible even though the diameter is small. One of the major causes of this effect is axial streaming. In a narrow vessel, the RBCs of the blood occupy the central or axial line and the plasma occupies the periphery. This phenomenon is called axial streaming. Small branches divide from these large branches therefore consists of plasma and few RBCs the hematocrit value is reduced.

3 Epidemiology

INTRODUCTION

Peripheral vascular disorders consist of both arterial and venous disorders. Venous and arterial diseases have been traditionally considered to be two distinct pathophysiological entities, but now it is observed that there are some common risk factors for these two disorders. There is biological plausibility to the concept that atherosclerosis and venous diseases may have a similar pathogenesis. In both arterial and venous thrombosis, activation of endothelium, platelets and leukocytes can be demonstrated, setting the prothrombotic stage for thrombus formation in the veins as well as arteries. Other factors associated with arterial and venous thrombosis are high levels of clotting components, older age, obesity, smoking, dyslipidemia and hormonal agents. The arterial cause for peripheral vascular disease (PVD) predominated over venous causes as the risk factors for peripheral artery disease (PAD) such as diabetes, smoking, obesity; hypertension is more prevalent in Indian patients.

Cardiovascular disease may be grouped into a triad consisting of cerebral, coronary and lower limb circulation. This triad is interwoven. Therefore, clinically evident coronary artery disease (CAD) coexists with PAD in 40% of cases. The CAD is also the major cause of death in patients with PAD. Globally speaking, 202 million people have PAD.

EPIDEMIOLOGY

Epidemiology of a disease includes prevalence, incidence, progression and morbidity and mortality associated with it. The PVD is a major cause of morbidity and mortality in the elderly population. The PVD of the lower extremity affects 20 million people in India. The 5-year rate of nonfatal cardiovascular events (including myocardial infarction and stroke) among patients with symptomatic PAD is approximately 20%; mortality ranges from 15% to 30%. Of those who develop critical limb ischemia (CLI) (1–2%), as many as 25% will ultimately require amputations and annual mortality among these patients is as high as 25%. Approximately 10–35% of patients with PAD present with classic claudication

and 20–40% have atypical leg pain. Nearly 50% of all patients with PAD are asymptomatic.

Although coronary and peripheral arterial diseases are macrovascular complications of diabetes, the clinical manifestations of PVD occur almost a decade later than CAD. Majority of patients with PVD have associated CAD, however the opposite is not true. Although atherosclerosis in patients with diabetes is similar to that seen in non-diabetic patients it is generalized, occurs prematurely and progresses at an accelerated pace.

Recent estimates by the World Health Organization (WHO) show that India already has the largest number of diabetic patients in any given country and this trend will continue in the future. Several studies have shown that the prevalence of CAD is very high among Asian Indians. The WHO estimates that between 2000 and 2030, the number of people with diabetes will increase by 114%. Conservative estimates based on population growth and aging and rate of urbanization in Asia show that India will have highest numbers of people with diabetes (79.4 million) by 2030. It is well-established that PAD is a marker for CVD and that the risk is greater with more advanced PAD.

It is a common condition with variable morbidity affecting men and women over the age of 45 years. It is going to be a major health problem in our country as the Indian population is aging.

Atherosclerosis is a generalized disorder and involves medium and large sized arteries. It is estimated that 74% patients of atherosclerotic CAD have involvement of some other vascular bed also. 40% patients of CAD have associated PVD, 14% have carotid artery stenosis and 17% have associated renal artery stenosis.

The incidence of PAD in the majority of European and North American studies, which range between 5 and 44 per 1,000 individual-years. Alzamora MT (2016) found the incidence of PAD in Spain (ARTPER cohort) is 4.3% at 5 years and 8.6 cases per 1,000 individual-years, being higher in men than in women, particularly in those under the age of 65 years. Smoking, age and physical activity limitations are the factors that are the most frequently associated with a decrease in ankle-branchial index (ABI) and the appearance of PAD.

Literature shows that the prevalence rate of PAD in different countries as follows; Greece 44%, USA 33%, UK 23.5%, Germany 16%, Srilanka 5.6%, Thailand 5.2%, Saudi Arabia 11.7%, China 19.8%, Brazil 20.4%, Spain 10.5%, Sweden 18% and South Africa 29.3%. In India, the prevalence rate in different regions of India is as follows; south India 3.9% and central India 3.79%.

Ashok Khurana (2013) analyzed the prevalence of PAD in North India (Punjab) in subjects type 2 diabetes mellitus (T2DM) as measured by ABI was 33% (66 out of total 200 patients). Among these 66 patients having PVD 51.6% (34) patients belonged to the age group 60 years and above, 30 patients had duration of diabetes >10 years, 62.1% overall patients had PAD had correlation with hemoglobin A1C (HbA1C) levels as compared to patients without PAD. The Chennai Urban Population Study, an epidemiological study conducted in south India showed the overall prevalence of 3.2% and known diabetic subjects had a higher (7.5%) prevalence of PVD. Hospital based studies from north India

by Agrawal et al (2004) showed that the prevalence of PVD is 735 (18.0%) out of 4,400 sample size. An unpublished clinic based data from north India (Guru Teg Bahadur Hospital (GTBH), Delhi, done by Madhu and Kant (2006), (n = 364 diabetic subjects) found the prevalence of PAD in diabetics to be between 18.1% and 13.73%, respectively. In the study done by Bhavana Sosale et al of 600 T2DM patients, 17.8%, one in six patients from south Indians with T2DM had asymptomatic PAD. A higher percentage of rural patients, i.e. 20% had PAD as opposed to 16.8% in the urban group.

RISK FACTORS OF PERIPHERAL ARTERY DISEASE

The risk factors associated with the incidence of PAD in this study were age, male gender, arterial hypertension, diabetes and low high-density lipoprotein (HDL) cholesterol, chronic kidney disease and their presence almost doubles the risk of presenting PAD during follow-up. Major physical activity limitations and active smoking also increased the risk of PAD incidence by 2.5 and 4 times, respectively. Age can be a risk factor probably because of the increase in the prevalence of different risk factors with age. The increased incidence in males may be explained by the increased prevalence of smoking in males or as suggested by some authors, because of genetic predisposition. However, paradoxically, in some studies fewer cardiovascular risk factors as well as a lower incidence of PAD events and contrarily, an increased predisposition for PAD were found in women. A study done by Eshcol J (2014) in south Indian diabetic patients, the overall incidence was 7.6 % of the study population (women—11.8%, men—5.1%).

Of these risk factors, age, smoking and diabetes pose the greatest risk for the development and progression of PAD. An elevated HbA1c being associated with progression of PAD stresses the need for strict control of diabetes.

A new analysis published by The Sage Group concludes that atherosclerotic PAD afflicts over 20 million in India. Both PAD and CLI represent major and significantly underestimated problems for the Indian economy and healthcare system. According to Mary L Yost, author of the report stated that PAD appears to afflict Indians at similar rates as those found in European and US populations in the same age and risk factor groups. According to the author older age is known as one of the most significant risk factors for both PAD and CLI. India has a much younger population with an average age of 23 years whereas the average age in the US is 35 years and in Germany 40 years. According to Mary L Yost, there are around 40 million diabetics in India and diabetics represent 60–80% of CLI patients. The prevalence of PVD in diabetics increases with age increasing from 3.2% in those below 50 years of age to 33% in those above 80 years of age. The prevalence of PVD in diabetics also increases with the duration of diabetes from 15% to 45% at 10–20 years respectively after the diagnosis of diabetes. As per the American Diabetes Association (ADA) 2012 consensus statement on PAD, those with a duration of diabetes >10 years have a higher risk for PAD. In India, the number of diabetic patients above the age of 80 years or with duration of diabetes more than 30 years is extremely low, thus explaining the low prevalence of PVD in diabetics.

Compared with PAD, Buerger's disease is relatively uncommon in India. It is estimated that fewer than 400,000 cases of CLI are caused by Buerger's disease.

VENOUS DISORDERS

Chronic venous disorder of the leg (CVDL) is an umbrella term that encompasses the various clinical presentations of chronic venous disease namely venous symptoms, edema, telangiectases, reticular vein, varicose veins, skin changes and venous ulcers. The CVDL is one of the most prevalent medical conditions in the general population. Varicose veins are common in females, but onset of the disease is earlier in males. The incidence of varicose veins ranged from 9 to 19 per 1,000 individual-years in men and from 19 to 26 per 1,000 individual-years in women in follow-up studies from Finland and the USA (Brand et al 1988, Makivaara et al 2004). The prevalence of varicose veins in different countries is as follows (males/females); USA 40–45/50–55%, UK 10–15/20–25%, Germany 36.3/47.4%, Portugal 20.7/40.8%.

In USA, the statistics shows approximately 1 in 22 or 4.5% or 12.2 million people are affected. It is also estimated that 41% of all women will suffer from abnormal leg veins by the time they are 50 years. The extrapolated prevalence rate of varicose vein in India providing warning is about 47,928,177 in statistics. According to another estimate 15–20% of population in India is suffering vein disease these days. The prevalence of varicose veins increases with age. Professions involving standing or sitting for prolonged periods of time have an increase risk of developing varicose veins, i.e. store clerks, waitresses, hair dressers, flight attendants, teachers, nurses, etc. The prevalence of varicose veins among school teachers in Kerala was 19%. Among those affected 89.5% had a history of standing for long duration and concluded as standing for longer hours was a major risk factors for prevalence of varicose veins and recommended health education and cutting down of working hours. The other reported risk factors are family history of varicose vein, obesity, lifestyle factors, multiparity (two or more pregnancy), female hormones, hormone replacement therapy, oral contraceptives, diet, smoking and alcoholic consumption.

Venous thromboembolic disease is a term used to denote deep venous thrombosis (DVT) and pulmonary embolism (PE). The DVT has always been under recognized, under diagnosed and under treated in India. There is a very high incidence of prothrombotic state in the Indian population; one condition that is easy to diagnose and treat is secondary polycythemia because of smoking. These patients present very late because of severe stasis changes. The incidence of venous thromboembolic disease in patients who underwent total hip or total knee arthroplasties and in patients who have sustained a fracture of the proximal end of the femur has ranged from 32% to 88% in the Western population. The use of routine thromboprophylaxis has shown to decrease the incidence to 15–30%. There are various factors that have been shown to be associated with high incidence of venous thromboembolism (VTE). They are malignancy, obesity, history of VTE in patient or family, hormonal replacement therapy, oral contraceptives, congestive heart failure, varicose veins, prolonged

immobilization, hematological disorders and chronic renal failure. The incidence of VTE in Indian patients was traditionally considered to be lower than the Western population. The genetic traits that may be possible explanations for this reduced incidence of VTE in Indian population include activated protein C resistance, decreased prevalence of homocystinemia, and a lesser prevalence of mutation known as factor V Leiden that is more common in Western population. Acquired traits that are thought to be risk factors for development of VTE and have been found to be less prevalent in Asians include obesity and heart failure. The overall incidence of obesity is lower in Indian patients as compared with the Western population.

MOTIVATION FOR AWARENESS AND INTEREST

Awareness and interest in PVD is growing in India because of the following reasons:

- Advancing age of the general population, resulting in increase prevalence of the PVD.
- Unwillingness of patients to accept the limitations and associated morbidity of vascular disease when therapeutic options are available.
- The realization that vascular disease in one system should prompt investigation of other areas for coexistent disease
- In the coming years with better disease care, longevity of our diabetics would significantly increase and it will not be surprising to see an increasing prevalence of PVD in Indian diabetics.
- Some of the worst sufferers of venous disorders come from the group involved in long-standing occupations such as grocery shop, textile, bakery, hotel, police who neither have the awareness nor the means to change occupation. They are forced to stand throughout their duty, which may be between 8 and 10 hours.

Wounds and particularly chronic wounds (ulcer), are a major concern for the Indian patient and clinician, chronic wounds affect a large number of patients and seriously reduce their quality of life. While there are few Indian studies on the epidemiology of chronic wounds, one study estimated the prevalence at 4.5 per 1,000 population. The incidence of acute wounds was more than double at 10.5 per as diabetes, atherosclerosis, tuberculosis and leprosy. Other major causes included venous ulcers, pressure ulcers, vasculitis and trauma. A study report stated that inappropriate treatment of acute traumatic wounds was the most common cause of the chronic wound. In India, as in other under-resourced nations, the problem of chronic wounds is compounded by other demographic factors, such as low literacy rates, poor access to health care, inadequate clinical manpower and a poor healthcare infrastructure. Inadequate education and clinical training in the fundamentals of basic wound care greatly magnify the problem in India. India has had its first wound healing program only in the last decade. Major textbooks on wound healing have only recently appeared on the shelves.

In India and the United Republic of Tanzania, the estimated prevalence of diabetes for the urban areas is between 12% and 14% and in rural areas about 1–2%. In the current estimates, on the advice of local experts, the prevalence of diabetes

in rural areas is assumed to be one-quarter that of urban areas for Bangladesh, Bhutan, India, Maldives, Nepal and Sri Lanka. By 2030, it is estimated that the number of people with diabetes >64 years of age will be >82 million in developing countries and >48 million in developed countries. By 2030 the number of diabetic patients project up to 87 million from an estimate of 50.8 million in 2010.

The diabetic foot syndrome encompasses a number of pathologies, including diabetic neuropathy, PVD, charcot neuroarthropathy, foot ulceration, osteomyelitis and potentially preventable end point amputation. Patients with the diabetic foot can also have multiple diabetic complications and caring for such patients may require attention to many different areas. Diabetic foot disease now results in major debilitating complications with severe morbidity and increased amputations. Diabetic foot complications have a large impact on the quality of life of patients and diabetic foot complications are a large economic problem, particularly if amputation results in prolonged hospitalization, rehabilitation, and an increased need for home care and social services. The 10.6% prevalence rate of diabetic foot syndrome (DFU) was reported in USA in a population based study, with 2.2% an annual rate on incidence. In the same year, 7.4% prevalence rate of DFU was reported by Walter et al, in UK. The average risk of foot ulcer development in peoples with diabetes is estimated to be 15%. Various risk factors for foot ulcer include peripheral neuropathy, PAD and foot deformity. A high prevalence of neuropathy promotes recurrence of foot lesions, more than 50% after 3 years. Unfortunately, these chronic wounds are often inadequately treated. India remains the nation with the highest and virtually unchanged new case detection rate of leprosy. Severe disabilities and ulceration are common, and the custom of begging and the prevailing caste system do little to improve rates of healing.

Chronic leg ulcers (CLU) affect 0.6–3% of those aged over 60 years, increasing to over 5% of those aged over 80 years. Estimate of annual incidence of leg ulcer in the UK and Switzerland are 3.5 and 0.2 per 1,000 individuals, respectively. It is thought that the incidence of ulceration is rising as a result of aging population and increased risk factors for atherosclerotic occlusion such as smoking, obesity and diabetes. The prevalence of ulcer is Ireland (0.12%) Brazil (1.5%), Australia (0.11%).

According to a study carried out in Germany, venous insufficiency was the dominating causative factor in 47.6% and arterial insufficiency in 14.5% and 17.6% of ulcers were because of combined arterial and venous insufficiency. Rarer causes included vasculitis (5.1%), exogenous factors (3.8%), and pyoderma gangrenosum (3.0%). Risks for venous ulcers are varicose veins, previous DVT in the affected leg, phlebitis in the affected leg, previous fracture, trauma or surgery, family history of venous disease and symptoms of venous insufficiency such as leg pain, heavy legs, aching, itching, swelling, skin breakdown, pigmentation and eczema. Risks for arterial ulcers are coronary heart disease, history of stroke or transient ischemic attack, diabetes mellitus, PAD including intermittent claudication, obesity and immobility.

4 Peripheral Vascular Diseases

DEFINITION

Peripheral vascular disease (PVD) refers to any disease or disorder of the circulatory system outside of the brain and heart. Peripheral vascular disease is the most common disease of the arteries.

GENERAL CLASSIFICATION

The PVD are classified into arterial, venous and lymphatic disorders. Arterial and venous disorders are classified again into acute and chronic disorders. All the PVD may fit into either acute or chronic disorders **(Fig. 4.1)**.

Classification of Arterial Disorders

Fontaine Stages According to Severity

This classification applies to only peripheral arterial disorders. There are four increasing stages of severity, which are as follows:

- *Stage I*: Asymptomatic
- *Stage II a*: Mild claudication (>200 meters)
- *Stage II b*: Moderate to severe claudication (<200 meters)
- *Stage III*: Ischemia rest pain
- *Stage IV*: Ulceration, necrosis or gangrene.

Rutherford Categories of Severity

This classification applies to only peripheral arterial disorders. There are seven increasing categories of severity, which are as follows:

- *Grade I (Category 0)*: Asymptomatic
- *Grade I (Category 1)*: Mild claudication
- *Grade I (Category 2)*: Moderate claudication
- *Grade I (Category 3)*: Severe claudication
- *Grade II (Category 4)*: Ischemia rest pain
- *Grade II (Category 5)*: Minor tissue loss, nonhealing ulcer, focal gangrene, diffuse pedal ischemia

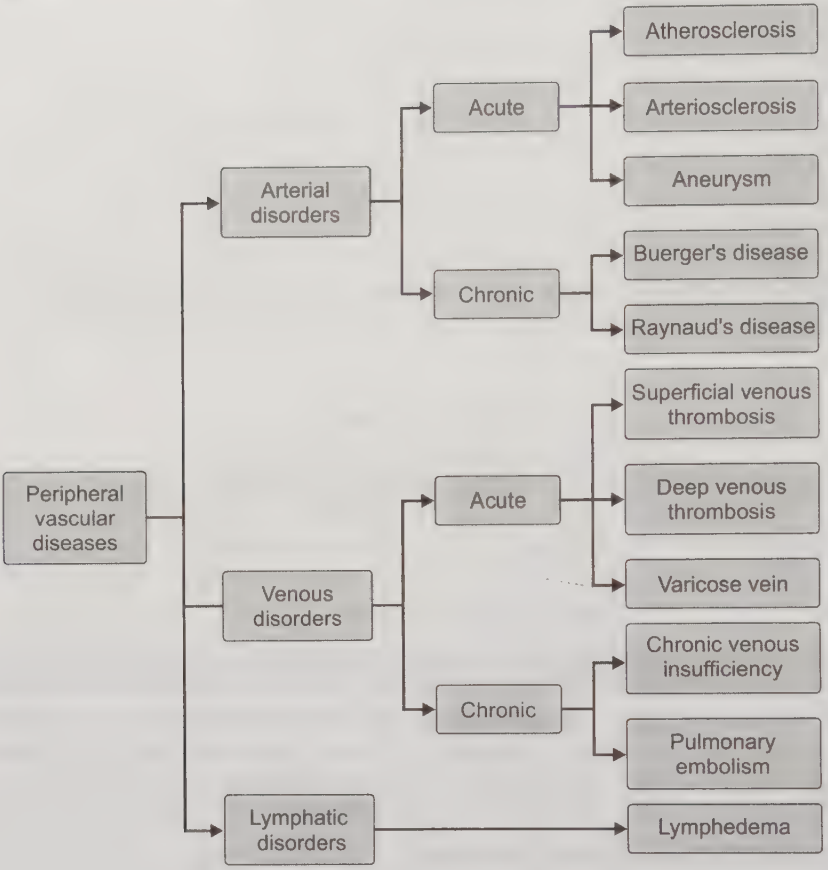


Fig. 4.1 General classification

- *Grade IV (Category 6)*: Major tissue loss extending above transmetatarsal level, foot no longer salvageable.

American Heart Association

The American College of Cardiology/American Heart Association (ACC/AHA) practice guidelines use the following divisions:

- *Asymptomatic*: Absence of leg claudication symptoms.
- *Claudication*: Inadequate blood flow during exercise, causing fatigue, discomfort or pain.
- *Critical limb ischemia*: Compromise of blood flow to extremity, causing limb pain at rest. Patients often have ulcers or gangrene.
- *Acute limb ischemia*: A sudden decrease in limb perfusion that threatens limb viability. These are associated with 5Ps:
  1. Pain
  2. Paralysis
  3. Paresthesias
  4. Pulselessness
  5. Pallor.

5 Arterial Disorders

5.1 ARTERIOSCLEROSIS

DEFINITION

It is a general term used to include all conditions with thickening and hardening of the arterial walls.

TYPES

- Hypertensive arteriosclerosis
- Monckeberg arteriosclerosis
- Atherosclerosis.

Hypertensive Arteriosclerosis

Subtypes are hyaline, hyperplastic and necrotizing arteriolitis.

Hyaline Arteriosclerosis

Causes

- Physiologically due to aging
- Pathologically due to hypertension or diabetes.

In this condition, visceral arterioles are involved. The vascular walls are thickened and the lumina narrows or even obliterated. The thickened vessel wall shows structure less, eosinophilic, hyaline material in the intima and media.

Pathogenesis

Permeability of the vessel wall is increased in hypertension diabetes, immunological reaction or due to aging. This results in the leakage of components of plasma across the vascular endothelium.

Hyperplastic Arteriosclerosis

Causes

- Malignant hypertension
- Hemolytic uremic syndrome
- Scleroderma
- Toxemia of pregnancy.

Pathogenesis

Systemic hypertension, which results in endothelial injury, hypoxia or immunologic damage leads to increased permeability. This is followed by proliferation of smooth muscle cells with fibrosis.

Necrotizing Arteriolitis

Necrotizing arteriolitis is a form of hyaline arteriosclerosis with necrotic changes.

Monckeberg Arteriosclerosis (Medial Calcific Sclerosis)

Monckeberg arteriosclerosis is the calcification of media of large- and medium-sized muscular arteries especially of the extremities and of the genital tract.

Causes

Age-related degenerative process.

Pathogenesis

Deposition of calcium salts in the media produces pipe stem-like rigid tubes without causing narrowing of the lumen.

For clinical features, medical management and surgical management—Refer Chapter 5.2 'Atherosclerosis.'

Atherosclerosis

Refer Chapter 5.2 'Atherosclerosis.'

5.2 ATHEROSCLEROSIS

DEFINITION

Atherosclerosis is a specific form of arteriosclerosis affecting primarily the intima of large- and medium-sized muscular arteries, and is characterized by fibrofatty plaques or atheroma.

INCIDENCE

The highest incidence occurs in the sixth and seventh decades of life. There is an increased prevalence of peripheral atherosclerotic disease in individuals with diabetes mellitus, hypercholesterolemia, hypertension or hyperhomocysteinemia and in cigarette smokers.

RISK FACTORS

Major Constitutional Risk Factors

- *Age*: Early lesions of atherosclerosis may be present in childhood. Clinical significant lesions are found with increasing age. Fully developed atheromatous plaques usually appear in the fourth decade.
- *Gender*: Incidence is more in men compared to women. Lower incidence in women especially in premenopausal age is due to high levels of estrogen and high density lipoproteins (HDL) both of which have antiatherogenic influence.
- *Genetic factors*: Hereditary genetic derangements of lipoprotein metabolism predispose the individual to high blood lipid level and familial hypercholesterolemia.
- *Familial factors*: It may be related to other risk factors such as diabetes, hypertension and hyperlipoproteinemia.
- *Racial factors*: Blacks have generally less severe atherosclerosis than whites.

Major Acquired Risk Factors

- *Hyperlipidemia*: Chronic hyperlipidemia may itself initiate endothelial injury and dysfunction by causing increased permeability. Increased serum concentration of low density lipoprotein (LDL) and very low density lipoprotein (VLDL) promotes formation of foam cells, while high serum concentration of HDL has antiatherogenic effect.
- *Hypertension*: It acts probably by mechanical injury to the arterial wall due to increased blood pressure.
- *Smoking*: Increased risk in smokers is due to reduced level of HDL and accumulation of carbon monoxide in the blood that produces carboxy hemoglobin and eventually hypoxia in the arterial wall favoring atherosclerosis. It also increases blood viscosity, increases fibrinogen there by increases the tendency to thrombosis.
- *Diabetes mellitus*: It is of both type I and type II accelerates the development of atherosclerosis, so that consequent atherosclerotic lesions appear earlier than in the general population. The cause of this atherosclerotic process is not known, but possible contributory factors are hyperlipidemia, reduced HDL levels, nonenzymatic glycosylation, increased platelet adhesiveness, obesity and associated hypertension in diabetes.

Minor Risk Factors

- *Environmental influences*: Higher incidence in developed countries and lower prevalence in under developed countries.

- *Obesity*: If the person is overweight by 20% or more is associated with increased risk.
- *Oral contraceptives*: Use of oral contraceptives by women has been shown to have increased risk of developing myocardial infarction or stroke.
- *Lack of physical activity*: Lack of exercise is associated with the risk of developing atherosclerosis and its complications.
- *Type A behavioral pattern*: The type A behavior pattern or stress characterized by aggressiveness, competitive drive, ambitiousness and sense of urgency is associated with enhanced risk of ischemic heart disease (IHD) compared with type B behavior of relaxed and happy-go-lucky type.
- *Alcoholism*
- *Viral infections*: It produces intimal cell proliferation and monoclonal cell proliferation in atheromatous lesion. The inflammation and scarring caused by infection can block, narrow or weaken blood vessels. Both salmonellosis (infection with *Salmonella* bacteria) and syphilis have been two infections traditionally known to infect and damage blood vessels.
- *Blood clot*: A blood clot can block a blood vessel (thrombus/emboli).
- *Inflammation of the arteries*: This condition is called arteritis and can cause narrowing or weakening of the arteries. Several autoimmune conditions can develop vasculitis, and besides the arteries, other organ systems are also affected.
- *Structural defects*: Defects in the structure of a blood vessel can cause narrowing. Most of these cases are acquired at birth, and the cause remains unknown. Takayasu disease is a vascular disease affecting the upper vessels of the body and affects usually Asian females.
- *Injury*: Blood vessels can be injured in an accident such as a car wreck, etc.
- People who have coronary heart disease or a history of heart attack or stroke generally also have an increased frequency of having peripheral vascular disease.

PATHOPHYSIOLOGY

Atherosclerosis is not caused by single etiologic factor, but is a multifactorial disease whose exact pathogenesis is still not known. A number of theories have been proposed.

Older Historical Theories of Atherosclerosis

- *Insudation hypothesis*: The concept hypothesized by Virchow in 1856 that atherosclerosis is a form of cellular proliferation of the intimal cells resulting from increased imbibing of lipids from the blood. This theory is also called lipid theory. Currently this theory is also called response to injury hypothesis.
- *Encrustation hypothesis*: The concept hypothesized by Rokitansky in 1852 that atheroma represented a form of encrustation on the arterial wall from the components in the blood forming the thrombi composed of platelets, fibrin and leucocytes. Currently it is believed that encrustation or thrombosis is not the sole factor in atherogenesis but the components of thrombus (platelets, fibrin and leukocytes) have a role in atheromatous lesions.

This theory has now been incorporated into the foregoing recent theory of response to injury.

Currently, the pathology of atherosclerosis is explained on the basis of the following two hypotheses.

1. Reaction to injury hypothesis.
2. Monoclonal theory.

Reaction to Injury Hypothesis

This theory incorporates aspects of two older historical theories of atherosclerosis, i.e. lipid theory of Virchow and thrombogenic theory of Rokitansky.

1. The original response to injury theory was first described by Ross in 1973 according to which the initial event in atherogenesis was considered to be endothelial injury followed by smooth muscle cell proliferation, so that the early lesions according to this theory consists of smooth muscle cells mainly.
2. The modified response to injury described subsequently in 1993 implicates lipoprotein entry into the intima as the initial event followed by lipid accumulation in the macrophages (foam cells), which according to the modified theory are believed to be the dominant cells in early lesions.

Following are the key components involved in atherogenesis:

Endothelial injury: It is the initial triggering event in development of lesions of atherosclerosis. Major risk factors, which acts together to produce endothelial injury are hemodynamic stress from hypertension, chronic hyperlipidemia and cigarette smoking.

Initial smooth muscle cell proliferation: Endothelial injury causes adherence, aggregation and platelet release reaction at the site of exposed subendothelial connective tissue. Proliferation of intimal smooth muscle cells is stimulated by various mitogens released from platelet adherence at both site of endothelial injury and monocytes. The most important of which is platelet-derived growth factor (PDGF); other are fibroblast growth factor (FGF); epidermal growth factor and transforming growth factor alpha (TGF-α). Proliferation can also be facilitated by loss of growth inhibitors such as TGF-β and heparin-like substances. Intimal proliferation of smooth muscle cells is accompanied by synthesis of matrix proteins such as collagen, elastic fiber, proteins and proteoglycans.

Role of blood monocytes: LDL appears in monocyte cytoplasm to form foam cell. Plasma LDL after entry into the intima undergoes oxidation. The oxidized LDL performs the following important function on monocytes and endothelium.

- For monocytes, oxidized LDL acts to attract, proliferate, immobilize and activate them as well as is readily taken up by scavenger receptor on the monocyte to transform it to a lipid-laden form cell.
- For endothelium, oxidized LDL is cytotoxic death of foam cell by apoptosis releases lipid to form lipid core of plaque.

Role of hyperlipidemia: Chronic hyperlipidemia initiate endothelial injury and dysfunction by causing increased permeability. Increase serum concentration of LDL and VLDL promotes formation of foam cell, while high serum concentration of HDL has antiatherogenic effect.

Thrombosis: Endothelial injury exposes subendothelial connective tissue resulting in formation of small platelet aggravates at the site and causing proliferation of smooth muscle cells. This causes mild inflammatory reaction, which together with foam cell is incorporated into the atheromatous plaque. The lesions enlarge by attracting fibrin and cells from the blood, so that thrombus becomes a part of atheromatous plaque.

Monoclonal Hypothesis

Monoclonal hypothesis is based on the postulate that proliferation of smooth muscle cell is the primary event and that this proliferation is monoclonal in origin similar to cellular proliferation in neoplasm. The monoclonal proliferation of smooth muscle cell in atherosclerosis may be initiated by mutation caused by exogenous chemicals (e.g. cigarette smoking), endogenous metabolites (lipoproteins) and some viruses (e.g. Marek's disease virus in chickens, herpes virus).

CLINICAL FEATURES

Symptoms

Symptoms are more common in the lower than in the upper extremities because of the higher incidence of obstructive lesions in the former region. The most common symptom seen in the legs is pain in one or both calves, thighs, or hips.

The iliac femoral, popliteal arteries are commonly affected and clinical features are as follows.

- The pain usually occurs, while walking or climbing stairs and stops when rest. This is because the muscles' demand for blood increases during walking and other exercise. The narrowed or blocked arteries cannot supply more blood, so the muscles are deprived of oxygen and other nutrients. This pain is called intermittent (comes and goes) claudication.
- It is usually a dull, cramping pain. It may also feel like a heaviness, tightness or tiredness in the muscles of the leg.
- Pins and needles, tingling or complete anesthesia may be present especially in hands or feet and increased by exercises.
- Buttock pain
- Numbness, tingling or weakness in the legs
- Burning or aching pain in the feet or toes, while resting
- An ulcer or wound on a leg or a foot that will not heal
- One or both legs or feet feel cold, or change color (pale, bluish, dark reddish)
- Impotence.

Signs

- Cramps in the legs have several causes, but cramps that start with exercise and stop with rest most likely are due to intermittent claudication. When the blood vessels in the legs are completely blocked, leg pain at night is very typical, and the individual almost always hangs his/her feet down to ease the pain. Hanging the legs down allows for blood to passively flow into the distal part of the legs.

- In patients with severe arterial occlusive disease, critical limb ischemia may develop.
- Frequently, these symptoms occur at night when the legs are horizontal and improve when the legs are in a dependent position.
- Important physical findings of peripheral arterial disease (PAD) include decreased or absent pulses distal to the obstruction, the presence of bruits over the narrowed artery, and muscle atrophy.
- With more severe disease, hair loss, thickened nails, smooth and shiny skin, reduced skin temperature, and pallor or cyanosis is frequent physical signs. In addition, ulcers or gangrene may occur.
- Elevation of the legs and repeated flexing of the calf muscles produce pallor of the soles of the feet, whereas rubor, secondary to reactive hyperemia, may develop when the legs are dependent.
- Patients with severe ischemia may develop peripheral edema because they keep their legs in a dependent position much of the time. Ischemic neuritis can result in numbness and hyporeflexia.

DIFFERENTIAL DIAGNOSES

- Mönckeberg's sclerosis
- Noninfectious arterial inflammation (Takayasu's arteritis, Kawasaki's disease)
- Periarteritis nodosa.

INVESTIGATIONS

- *Ultrasound* measurement of intima-media thickness using B-mode ultrasound.
- *Intravascular ultrasound* can detect atherosclerotic plaques not visible on angiography; may show up recent plaque disruption.
- *Computed tomography (CT): Multislice spiral CT or electron beam* CT to quantify coronary artery calcium.

TREATMENT

Treatment for atherosclerosis focuses on reducing symptoms and preventing further progression of the disease. In most cases, lifestyle changes, exercise and claudication medications are enough to slow the progression or even reverse the symptoms of atherosclerosis.

The goals of treatment include:

- Relieving symptoms
- Reducing risk factors in an effort to slow or stop the buildup of plaque
- Lowering the risk of formation of blood clots
- Widening or bypassing plaque-clogged arteries
- Preventing atherosclerosis-related diseases.

Medical Treatment

Cholesterol medications: Aggressively lowering LDL cholesterol, the 'bad' cholesterol, which can slow, stop or even reverse the buildup of fatty deposits

in the arteries. Boosting HDL cholesterol, the 'good' cholesterol, may help too. Cholesterol medications including drugs known as statins and fibrates may be given.

Antiplatelet medications: Aspirin reduce the likelihood that platelets will clump in narrowed arteries, form a blood clot and cause further blockage.

Beta blocker medications: They lower heart rate and blood pressure, reducing the demand on heart and often relieve symptoms of chest pain. Beta blockers like Atenolol, Metoprolol, and Propranolol reduce the risk of heart attacks and heart rhythm problems.

Angiotensin-converting enzyme (ACE) inhibitors: These medications can help slow the progression of atherosclerosis by lowering blood pressure and producing other beneficial effects on the heart arteries. ACE inhibitors can also reduce the risk of recurrent heart attacks, e.g. Quinapril, Enalapril.

Vasoactive drugs [Pentoxifylline and Nafronyl oxalate (Naftidrofuryl)]: Pentoxifylline enhances red blood cell flexibility and to decrease blood viscosity. Nafronyl oxalate enhances aerobic glycolysis and oxygen consumption in ischemic tissues.

Calcium channel blockers (Verapamil, Diltiazem, Nifedipine): These medications lower blood pressure and are sometimes used to treat angina.

Water pills (diuretics): High blood pressure is a major risk factor for atherosclerosis. Diuretics lower blood pressure.

Vitamin E (Vita-Plus E, Soft gels, Aquasol E): This antioxidant protects polyunsaturated fatty acids in membranes from attack by free radicals.

Omega-3 polyunsaturated fatty acid: The possible benefits of omega-3 polyunsaturated fatty acid in the treatment of atherosclerosis include effects on lipoprotein metabolism, hemostatic function, platelet/vessel wall interactions, antiarrhythmic actions, and the inhibition of proliferation of smooth muscle cells and therefore growth of the atherosclerotic plaque. Fish oil feeding has also been found to result in moderate reductions in blood pressure and to modify vascular neuroeffector mechanisms.

Surgical Treatment

Angioplasty: It is a nonsurgical procedure that can widen a narrowed or blocked artery. A thin tube (catheter) is inserted into an artery in the groin or arm, and advanced to the area of narrowing. A tiny balloon on the tip of the catheter is then inflated to enlarge the narrowing in the artery.

Cryoplasty: It is a newer form of angioplasty in which freezing is used to open a narrowed artery. In this procedure, the balloon on the catheter is filled with liquid nitrous oxide, which freezes and destroys plaques within the artery.

Endarterectomy: This is the removal of an atheromatous occlusion by stripping it out together with the tunica interna and part of the media inside the artery of affected leg.

Bypass surgery: It involves using a vein from the body or a portion of synthetic vessel (known as grafts) to create a detour around the blockage. One end of the graft is sewn to the damaged artery above the blockage and the other end is sewn below the blocked area. Blood flow is then able to bypass the area of narrowing or blockage.

5.3 ANEURYSM

DEFINITION

An aneurysm is defined as a permanent abnormal dilation of blood vessel occurring due to congenital or acquired weakening or destruction of vessel wall. Aneurysms can occur in both veins and arteries, but they are much more common in arteries. Most commonly aneurysm involves large elastic arteries, specially the aorta and its major branches. The abdominal and thoracic (chest) aortas are the most frequent locations for arterial aneurysms.

INCIDENCE

- The peak incidence is in the sixth and seventh decades
- Men are more affected than women with a ratio of 2:1.

CLASSIFICATION

- *Classification based on number of layers involved:*
  - True aneurysm is one in which all the three layers of arterial wall in the dilatation or aneurysm. There will be 50% increase in the normal diameter of the vessel.
  - False aneurysm is one in which has a single layer of fibrous tissue as the wall of the sac and does not contain the three layers of the arterial wall as the covering of aneurismal sac.
  - Dissecting aneurysm is one in which the blood enters the separated or dissected wall of the vessel. Dissecting aneurysm occurs when there is tear in the intima and a column of blood is forced to enter between the inner and outer layers of tunica media.
- *Classification based on shape:*
  - Saccular having large spherical out pouching from one side of the arterial wall.
  - Fusiform having slow spindle-shaped dilatation.
  - Cylindrical with a continuous parallel dilatation.
  - Serpentine or varicose, which has tortuous dilation of the vessel.
  - Racemose having mass of intercommunicating small arteries and vein.

- *Classification based on pathogenic mechanism:*
  - Atherosclerotic aneurysms.
  - Syphilitic aneurysm.
  - Mycotic aneurysm.
  - Berry aneurysm.
- *Classification based on the site:*
  - Central aneurysms occur in the proximal blood vessels such as abdominal aorta, thoracic aorta and cerebral blood vessels.
  - Peripheral aneurysms—Aneurysms that occur in arteries other than the aorta and the brain arteries are called peripheral aneurysms. Common locations for peripheral aneurysms include the popliteal, femoral and carotid arteries.
- *Classification based on involvement of blood vessels:*
  - Arterial.
  - Venous.
  - Arteriovenous.

ETIOLOGY

Aneurysms are caused by weakness in the wall of a cerebral artery or vein, aortic artery, or peripheral artery. The disorder may result from defects present at birth (congenital) or from acquired.

- *Congenital (Berry aneurysm)*: Occurs in the circle of Willis. Connective tissue defects similar to Marfan's syndrome may cause congenital aneurysm. Even congenital arteriovenous fistula can lead to aneurysm.
- Acquired can be broadly classified into three groups, i.e. traumatic degenerative and infective.
  1. *Traumatic*: Majority of aneurysm is false aneurysms and are fusiform in shape. Direct trauma such as penetrating wound to the artery may cause such aneurysm. Indirect trauma may cause aneurysm, e.g. at the subclavian artery distal to the point where it crosses the cervical rib.
  2. Degenerative cause is more common. Atherosclerosis is associated with degenerative changes and if mechanical strains developed due to local anatomic factors such as lumbar lordosis and the repeated flexion and extension at knee may predispose to aneurysm.
  3. Infections such as syphilis, nonspecific fungal infection can cause aneurysm.

RISK FACTORS

High blood pressure, high cholesterol levels and cigarette smoking may raise the risk of abdominal aortic aneurysms. Pregnancy is often associated with the development and rupture of splenic artery aneurysms. Inherited conditions that affect the connective tissues of the body, such as Marfan's syndrome, also increase the risk of developing certain types of aneurysms.

PATHOPHYSIOLOGY

The occurrence and expansion of an aneurysm in a given segment of the arterial tree involves local hemodynamic factors and factors intrinsic to the arterial segment itself. The aorta is a relatively low-resistance circuit for circulating blood. The lower extremities have higher arterial resistance, and the repeated trauma of a reflected arterial wave on the distal aorta may injure a weakened aortic wall and contribute to aneurysmal degeneration. Systemic hypertension compounds the injury, accelerates the expansion of known aneurysms and may contribute to their formation. Aneurysm formation is probably the result of multiple factors affecting that arterial segment and its local environment. Increasing aneurysmal dilatation leads to increasing arterial wall tension or stress.

CLINICAL FEATURES

Aneurysms can develop slowly over many years. Many people have no symptoms, while others may experience a number of symptoms that vary in intensity among individuals depending on the location, rate of growth, and size of the aneurysm.

Common Symptoms of an Carotid Aneurysm

Aneurysms typically do not produce symptoms. However, if an aneurysm ruptures, any of these aneurysm symptoms may occur and can be severe:

- Blurred or double vision
- Confusion or loss of consciousness for even a brief moment
- Dilated pupil
- Drooping eyelid
- Increased sensitivity to light
- Nausea with or without vomiting
- Pulsing sensation
- Seizures and tremors
- Stiff neck
- Difficulty swallowing
- Slurred speech or inability to speak
- Numbness, weakness or paralysis of one side of the face
- Rapid breathing (tachypnea) or shortness of breath
- Rapid heart rate (tachycardia)
- Worst headache.

Symptoms of Aortic Aneurysm

- *Due to compression of the adjacent tissue:*
  - Chest pain
  - Shortness of breath
  - Cough
  - Hoarseness
  - Dysphagia.

- *Due to aneurismal dilatation of ascending aorta:*
  - Congestive heart failure as a consequence of aortic regurgitation.
- *Due to the compression of superior vena cava:*
  - Congestion of head and neck, and upper extremities.

DIFFERENTIAL DIAGNOSIS

- Appendicitis
- Arterial embolism
- Hemothorax
- Intestinal ischemia
- Intestinal obstruction
- Ischemic heart disease
- Myocardial infarction
- Pain related to acute chest trauma
- Pneumothorax
- Gastrointestinal ulcer
- Unstable angina
- Cerebral hemorrhage
- Cerebrovascular accident (CVA)
- Congenital arteriovenous malformation in the brain
- Migraine headache
- Tumor (neoplasm).

INVESTIGATIONS

- *Blood*: Hemoglobin level, full blood count, erythrocyte sedimentation rate, blood grouping and cross matching, blood lipids, electrolytes, liver function tests and renal function test should be performed.
- *Urine analysis*: For screening diabetes.
- ECG should be performed.
- *Aortagraphy*: To confirm about the extent of suspected extensive lesion and to exclude presence of small aneurysm.
- *Ultrasound scanning*: The most important investigation for a popliteal aneurysm is an ultrasound scan. This confirms that an aneurysm is present and at the same time can determine the size of the aneurysm.
- *Angiogram*: This will provide a road map of the arteries so that a bypass operation may be planned. An angiogram is usually not useful in assessing the size of a popliteal. The aneurysm as it can only assess the inside of the aneurysm and not the outer wall.
- *X-rays and ultrasound*: Routine X-rays may show popliteal artery aneurysms if deposits of calcium exist in the arteries. An aneurysm that has calcified is a sign of hardening of the arteries (atherosclerosis).
- *Computed tomography (CT)*: A CT scanner uses X-ray scanners to create detailed images of the arteries and blood vessels in the leg. CT angiography may be done using injected dyes that show the flow of blood. Special software may be used to create detailed three-dimensional (3D) images of the aneurysm.

- *Magnetic resonance imaging (MRI)*: In MRI, a cylindrical machine scans the body using magnetic fields to create computer images of the body's soft tissues. Like CT angiography, MR angiography may be done using dyes that show the flow of blood and special software that creates 3D images of the aneurysm.

MEDICAL MANAGEMENT

Medical therapy should be initiated as soon as the diagnosis is considered. The patient should be admitted to an intensive care unit for monitoring hemodynamic and urine output. Unless hypotension is present, therapy should be aimed at reducing cardiac contractility and systemic arterial pressure, and thereby shear stress. For acute dissection (unless contraindicated) adrenergic blockers should be administered parenterally, using intravenous Propanolol, Metoprolol or the short-acting esmolol to achieve minimum heart rate of 60 beats per minute. This should be accompanied by sodium nitroprusside infusion to lower systolic blood pressure to 120 mm Hg. Labetalol, a drug with both adrenergic blocking properties, is also used as a parenteral agent in the acute therapy of dissection. The calcium channel antagonists, verapamil and diltiazem, may be used intravenously if nitroprusside or labetalol cannot be employed. The addition of a parenteral angiotensin-converting enzyme (ACE) inhibitor, such as enalaprilat, to adrenergic blocker may also be considered. Isolated use of direct vasodilators, such as diazoxide and hydralazine, is contraindicated because these agents can increase hydraulic shear and may propagate dissection.

SURGICAL MANAGEMENT

- *Arterial ligation*: The different methods of ligation are:
  - The ligature is applied just proximal the sac
  - *Brasdor's method*: The ligature is applied just distal to the sac.
  - *Hunter's method*: The ligature is applied immediately above a branch of the artery
  - *Wardrop's method*: The ligature is applied immediately below a branch of the artery
  - *Antylus method*: Two ligatures are applied one proximal and another distal to the aneurysmal sac.
- *Wiring of the aneurysmal sac*: A long fine thread of stainless steel wire is introduced into the sac with a hypodermic needle. So the wire gets coiled within the sac and this lead to clotting, thrombosis followed by fibrosis.
- *Wrapping of aneurysmal sac*: A strip of fascia lata, polythene or cellophane sheet may be wrapped around the aneurysmal sac to strengthen its wall and prevent rupture.
- *Aneurysmorrhaphy*: This is particularly suitable in case of saccular aneurysm of arteries such as femoral or popliteal. The aneurysmal sac is totally excised and the defect in the arterial wall is closed by suturing of the adjacent healthy arterial wall.
- *Excision and grafting*: Dacron graft or autogenous vein is used for grafting.

- *Excision and end-to-end suturing*: This is only possible in case of peripheral aneurysm, when after excision of the aneurysm to cut and can be approximated by mobilization for end-to-end anastomosis.

COMPLICATIONS

An aneurysm can cause various ill effects such as thrombosis, thromboembolism and alteration in the flow of blood, rupture of the vessel and compression of its neighboring structures.

5.4 RAYNAUD'S DISEASE

DEFINITION

Vasospasm of the acral or distal vessels often triggered by cold stress and relieved by warmth or pharmacological agents. It is characterized by episodic digital ischemia, manifested clinically by the sequential development of digital blanching, cyanosis, and rubor of the fingers or toes following cold exposure and subsequent rewarming. Emotional stress may also precipitate Raynaud's phenomenon.

TYPES

Basically, it is of two types.

1. *Primary Raynaud's*: Often, the cause of it is not known. This type of Raynaud's is also called Raynaud's disease.
2. *Secondary Raynaud's*: Sometimes a disease, condition, or other factor causes Raynaud's. This type of Raynaud's is called Raynaud's phenomenon or secondary Raynaud's.

INCIDENCE

- Incidence of Raynaud's disease is higher in colder climates.
- Primary Raynaud's affects about 3% of general population, according to The National Institute of Arthritis and Musculoskeletal and Skin Diseases. In most cases, it occurs between the ages of 18 years and 30 years.
- Secondary Raynaud's occurs between the ages of 35 years to 40 years. Approximately, 90% of patients with scleroderma, 30% of patients with lupus, 20% of patients with rheumatoid arthritis, and 30% of patients with Sjögren's syndrome develop secondary Raynaud's.

RISK FACTORS

The risk factors for primary Raynaud's include:

- *Age*: Primary Raynaud's usually develops before the age of 30.

- *Gender*: Women are more likely to have primary Raynaud's than men. (More emotional stress, estrogens vascular reactivity to colder temperature, genetic carriers and auto immune disease more common).
- *Family history*: Primary Raynaud's may occur in members of the same family.
- *Living in a cold climate*: Cold temperatures can trigger Raynaud's attacks.

The risk factors for secondary Raynaud's include:

- *Age:* Secondary Raynaud's usually develops after the age of 30.
- Certain diseases and conditions that directly damage the arteries or nerves that control the arteries in the hands and feet may cause secondary Raynaud's.
- Injuries to the hands or feet.
- Exposure to certain workplace chemicals such as vinyl chloride (used in the plastics industry).
- Repetitive actions with the hands, such as typing or using vibrating tools.
- Certain medicines such as migraine, cancer, cold/allergy, or blood pressure medicines.
- Smoking.
- Living in a cold climate.

ETIOLOGY

The etiology of primary Raynaud's is not exactly known. There are multiple factors causing secondary Raynaud's. Examples include:

- Diseases and conditions that directly damage the arteries or damage the nerves that control the arteries in the hands and feet.
- Repetitive actions that damage the nerves that control the arteries in the hands and feet.
- Injuries to the hands and feet.
- Exposure to certain chemicals.
- Medicines that narrow the arteries or affect blood pressure.

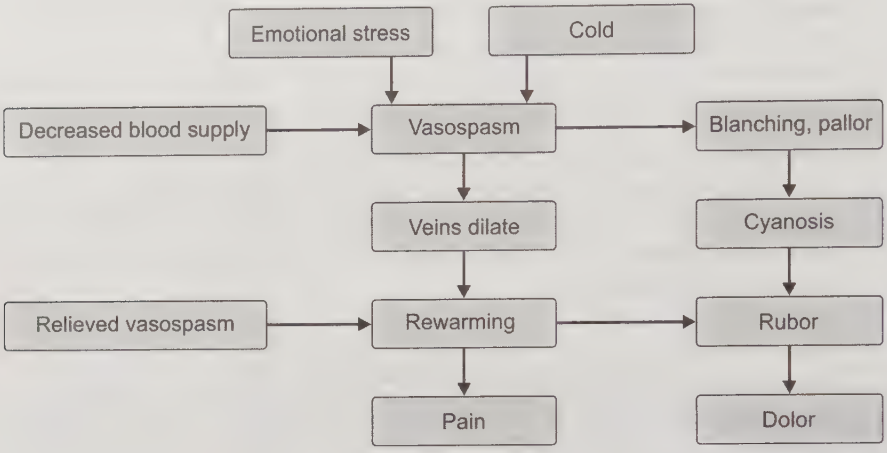
PATHOPHYSIOLOGY (FLOW CHART 5.4.1)

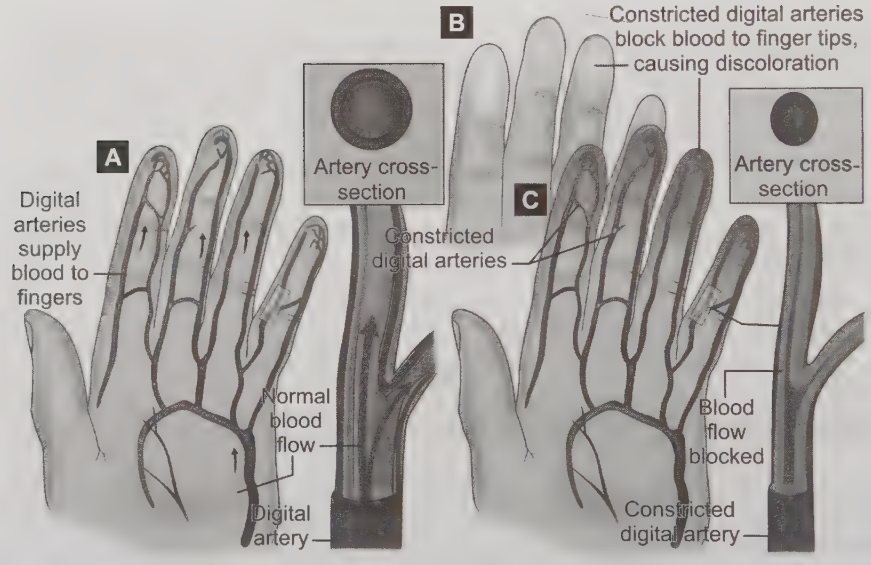
The color changes are usually well demarcated and are confined to the fingers or toes, which results from vasospasm of digital arteries. The capillaries and venules dilate, and cyanosis results from the deoxygenated blood that is present in these vessels. With rewarming, the digital vasospasm resolves, and blood flow into the dilated arterioles and capillaries increases dramatically. This "reactive hyperemia" imparts a bright red color to the digits. In addition to rubor and warmth, patients often experience a throbbing, painful sensation.

CLINICAL FEATURES (FIGS 5.4.1A TO C)

- Tricolor sequence of color changes in the skin in response to cold or stress. Tricolor phenomenon is characterized by white (initial paleness with paresthesia, Fig. 5.4.1B), then blue (cyanosis due to venostasis, Fig. 5.4.1C and finally red (hyperemia due to vasodilation, Fig. 5.4.1A)
- Numb, prickly feeling or stinging pain upon warming or relief of stress.

Flow chart 5.4.1 Pathophysiology of Raynaud's phenomenon





Figs 5.4.1A to C (A) Arteries in the fingers (digital arteries) with normal blood flow. The inset image shows a cross-section of a digital artery; (B) Fingertips that have turned white due to blocked blood flows; (C) Narrowed digital arteries causing blocked blood flow and blue fingertips. The inset image shows a cross-section of a narrowed digital artery
(For color version, see Plate 1)

- Look pallor due to lack of blood flow.
- Redness and throbbing or tingling sensation as blood returns to the affected area.
- One fingertip temporarily turns white and may start to ache; the finger turns white with increasing frequency. Other fingers begin turning white, but the

thumb is not usually affected. After several fingers turn white, the disease is probably irreversible.

Most people who have Raynaud's have no long-term tissue damage or disability. However, people who have severe Raynaud's can develop skin sores or gangrene from prolonged or repeated Raynaud's attacks.

At first during an attack of Raynaud's, affected areas of skin usually turn white. Then the areas often turn blue, feel cold and numb and the sensory perception is dull. The affected skin may look slightly swollen. As circulation improves, the affected areas may turn red, throb, tingle or swell. The order of the changes of color isn't the same for all people, and not everyone experiences all three colors.

Occasionally, an attack affects just one or two fingers or toes. Attacks do not necessarily always affect the same digits. Although Raynaud's most commonly affects your fingers and toes, the condition can also affect other areas of body such as nose, cheeks, ears and even tongue. An attack may last less than a minute to several hours. Over time, attacks may grow more severe.

People who have Raynaud's accompanied by another disease may also have symptoms related to their underlying condition. Raynaud's attacks can last less than a minute or as long as several hours. Attacks can occur daily or weekly.

INVESTIGATIONS

- *Liver function tests/thyroid function tests* can be used as basic screening tests in appropriate patients. The presence of specific autoantibodies, e.g. anti-nuclear antibodies, may suggest underlying inflammatory disease. Plasma glucose should be checked for possible diabetes.
- *Urine/serum protein electrophoresis*, cold agglutinins and fibrinogen levels may identify hyper viscosity states.
- *Chest X-ray* (thoracic outlet views) may show a cervical rib.
- *Cold stimulation test*: A cold stimulation test can be used to trigger Raynaud's symptoms. For this test, a small device that measures temperature is taped to the fingers. The hands are then exposed to cold (they are usually briefly put into ice water). The hands are then removed from the cold, and the device measures how quickly the fingers return to their normal temperature. If one has Raynaud's, it may take more than 20 minutes for the fingers to return to their normal temperature.

MEDICAL MANAGEMENT

If lifestyle changes do not control Raynaud's, medicines are prescribed. Medicines improve blood flow to the fingers and toes.

- *To reduce vascular tone:* Calcium channel blockers, nitroglycerin.
- *To increase vasodilatation:* Alpha adrenergic antagonists (prazosin, phenoxybenzamine, angiotensin-converting enzyme inhibitors (captopril and lisinopril).
- *To block mediators of platelet adhesion and vasoconstriction:* Prostaglandins, thromboaxane synthetase inhibitors, serotonin inhibitors.
- *To reduce blood viscosity:* Plasmaphoresis, hemodilution, removal of fibrinogen.

Calcium channel blockers are traditionally first-choice prescription medications to treat RS. These drugs block calcium channels in the smooth muscle of vessel walls, thereby preventing contraction. Nifedipine is considered the standard for treatment. These drugs improve symptoms only moderately in the treatment of secondary Raynauds syndrome.

Adrenergic blocking agents such as reserpine have been shown to increase nutritional blood flow to the fingers. The postsynaptic adrenergic antagonist prazosin has been used with favorable responses. Doxazosin and Terazosin may also be effective.

Vasodilator such as Cilostazol (a relatively new medicine) has shown promise in the treatment of RS (Rajagopalan S et al. 2003). Sildenafil has also been used with some effectiveness.

Prostaglandin E1 containing Transdermal patches improves blood flow to skin capillaries and reduced the number of episodes of RS in subjects who had RS secondary to scleroderma.

Vitamins supplements must be provided as these help in synthesis of collagen and improves blood flow. The use of antioxidants in treating Raynauds syndrome is based on research studies that showed that levels of important antioxidants such as Vitamin E, Vitamin C and Selenium are depleted in patients who have Raynauds syndrome.

PROPHYLAXIS

- Wearing mittens or heated gloves
- Protection from cold and moisture
- Avoid vibrating tools (power saws, jack hammer)
- Often changing duties at work is required
- Make regular repeated fist
- Medications which could be triggers, such as beta blockers, ergot-containing substances and oral contraceptives should be avoided.

SURGICAL MANAGEMENT

Rarely, people who have severe Raynaud's may develop skin sores or gangrene. If this happens, antibiotics or surgery to cut out the damaged tissue may be needed.

Surgical cervical sympathectomy is helpful in some patients who are unresponsive to medical therapy, but benefit is often transient.

Another treatment for severe Raynaud's is to block the nerves in the hands or feet that control the arteries. This can help prevent Raynaud's attacks. This treatment is done using surgery or shots. The surgery often relieves symptoms, but sometimes for only a few years. Shots may need to be repeated if symptoms persist or come back.

5.5 BUERGER'S DISEASE

DEFINITION

Buerger's disease is an inflammatory occlusive vascular disorder involving small and medium-sized arteries and veins in the distal upper and lower extremities. It is characterized by the absence or minimal presence of atheroma, segmental vascular inflammation, vaso-occlusive phenomenon and involvement of small- and medium-sized arteries and veins of the upper and lower extremities.

INCIDENCE

Most patients with Buerger's disease are aged 20–45 years. This disorder develops most frequently in men under the age of 40 (male-to-female ratio is 3:1).

PREVALENCE

Prevalence is higher in Asians and individuals of eastern European descent.

ETIOLOGY

While the cause of thromboangiitis obliterans (TAO) is not known, there is a definite relationship to cigarette smoking in patients with this disorder. It has been postulated that Buerger's disease is an 'autoimmune' reaction (one in which the body's immune system attacks the body's own tissues) triggered by some constituent of tobacco.

PREDISPOSING FACTORS

It has been proposed in Japan that the presence of a gene linked to some human leukocyte antigen (HLA) might control the susceptibility to the disease. The notion that the condition is linked to tobacco exposure is supported by the fact that the disease is more common in countries with heavy use of tobacco and is perhaps most common among natives of Bangladesh who smoke a specific type of cigarettes, homemade from raw tobacco, called '*bidi*'.

Socioeconomic conditions and working environment may also play a role in etiology as the disease is seen more in outdoor and manual workers. Hypercoagulable state has been observed in association with the disease. Hepatitis B virus (HBV) and rickettsiosis may contribute to pathogenesis, but this role is uncertain.

PATHOPHYSIOLOGY

In the initial stages of thromboangiitis obliterans, polymorphonuclear leukocytes infiltrate the walls of the small and medium-sized arteries and veins. The internal elastic lamina is preserved and thrombus may develop in the

vascular lumen. As the disease progresses, mononuclear cells, fibroblasts and giant cells replace the neutrophils. Later stages are characterized by perivascular fibrosis and recanalization.

CLINICAL FEATURES

- The symptoms mainly include a triad of the affected extremity, which is characterized by:
  - Intermittent claudication
  - Raynaud's phenomenon
  - Migratory superficial vein thrombophlebitis.

  Patient may experience pain during walking, which will relieve by rest. Most patients (70–80%) with Buerger's disease present with distal ischemic rest pain.
- *Weakness in hands and feet:* The lack of blood flow to the arms, legs, hands and feet can cause pain and weakness in the affected limbs.
- *Swelling*: Patients with Buerger's disease experience swelling in the hands and feet.
- *Sores*: As blood flow decreases, the skin becomes ulcerated. Without blood flow to the affected areas, the skin will decay and gangrene occurs **(Fig. 5.5.1)**.
- *Discoloration*: Hands and feet are sensitive to cold temperatures in patients with Buerger's disease. The hands and feet may turn white, blue and then red in these patients.
- *Paresthesias* (numbness, tingling, burning, hypoesthesia) of the feet and hands.
- The hands and feet of patients with the disease are usually cool.
- *Impaired distal pulses* in the presence of normal proximal pulses are usually found in patients with the disease.

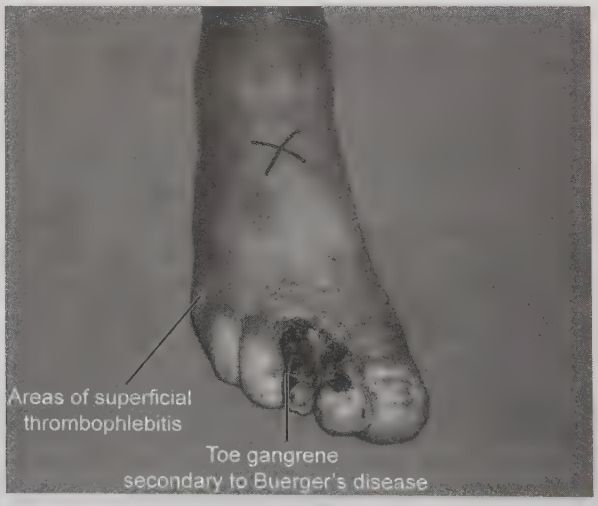


Fig. 5.5.1 A patient's leg with Buerger's disease
Source: www.angiologist.com
(For color version, see Plate 1)

Box 5.5.1 Olin's diagnostic criteria

- Age younger than 45 years
- Current (or recent) history of tobacco use
- Presence of distal extremity ischemia (indicated by claudication, pain at rest, ischemic ulcers or gangrene) documented by noninvasive vascular testing
- Exclusion of autoimmune diseases, hypercoagulable states and diabetes mellitus (DM) by laboratory tests
- Exclusion of a proximal source of emboli by echocardiography and arteriography
- Consistent arteriographic findings in the clinically involved and noninvolved limbs.

DIAGNOSIS

Because a firm diagnosis of Buerger's disease is difficult to establish, a number of different diagnostic criteria have been proposed (Shionoya 1998, Olin 2000, etc.). Olin's diagnostic criterion is given in the **Box 5.5.1**.

DIFFERENTIAL DIAGNOSIS

- *Atherosclerosis*: The distal nature of thromboangiitis obliterans and involvement of the legs and arms help to differentiate it from atherosclerosis.
- *Systemic vasculitis*: Internal elastic lamina and the media are preserved in patients with thromboangiitis obliterans where as in systemic vasculitis there will be disruption of this lamina.
- *Scleroderma, crest syndrome (calcinosis cutis, Raynaud phenomenon, sclerodactly and telangiectasia, repetitive trauma, emboli, hypercoagulable states and vasculitis*: These disorders primarily involves in hand however in thromboangiitis obliterans primary involvement in the hand.
- *Raynaud disease*: This disease primarily affects the arm and usually it is triggered by emotional stress or cold.

INVESTIGATIONS

- *Laboratory studies*: Erythrocyte sedimentation rate (ESR), which is usually elevated in certain autoimmune diseases.
- Angiography/Arteriography findings in patients with Buerger's disease are nonatherosclerotic, segmental occlusive lesions of the small- and medium-sized vessels (e.g. digital, palmar, plantar, tibial, peroneal, radial and ulnar arteries) with formation of distinctive small-vessel collaterals around areas of occlusion known as 'corkscrew collaterals'.
- *Echocardiography*: Echocardiography should always be performed in patients thought to have Buerger's disease in order to exclude a proximal source of emboli as the cause of distal vessel occlusion.
- Excisional biopsy and pathological examination of an involved vessel disorders.
- *X-ray*: Presence of cervical rib or carpal tunnel syndrome should be excluded by X-ray.
- *Doppler ultrasound*: It gives quantitative information about the degree of stenosis.

- *Plethysmography*: Method of assessing changes in volume due to arterial supply to that particular part. Recently segmental plethysmography has been introduced by placing venous occlusion cuff around the thigh calf and ankle. The cuffs are inflated to 65 mm Hg and the pulsation is the quantitative measure to the arterial disease.
- *Phonoangiography*: Vibrations of low frequency in the arterial wall due to disturbance in the blood can be analyzed audibly.
- *Isotope technique*: Xenon 133 injected intramuscularly and its clearances used to study the blood flow in the calf muscles. Recently technetium has been used as a choice of isotope. Gamma camera is used to picture out blood flow in the limb.
- *Magnetic resonance angiography (MRA)*: To detect flow-related phenomenon.

MEDICAL MANAGEMENT

- Absolute discontinuation of tobacco
- *Vasodilators*: To increase circulation
- *Analgesics*: To relieve pain
- *Thrombolytic therapy*: Streptokinase
- *Skin hygiene*: To prevent wound infection
- Antibiotics for wound infection
- Recently treatment with intravenous iloprost (a prostaglandin analog), an expensive therapy has been shown to be somewhat effective in improving symptoms (relief of pain), accelerating resolution of distal extremity trophic changes, and reducing the amputation rate among patients with Buerger's disease.

SURGICAL MANAGEMENT

- *Upper thoracic or lumbar sympathectomy*: To relax the arterial muscle and increase the vessel lumen thereby improving the blood supply to the extremities.
- Spinal cord stimulator (SCS) implantation relieves pain by pain gate theory and increases vasodilation. SCS may be a useful therapeutic option in BD, particularly for pain control and wound healing and may delay the need for amputation in selected patients, who have exhausted all other therapeutic options. Vasodilation is due to inhibition of sympathetic vasoconstriction improves the peripheral microcirculation. Nitric oxide and gamma-amino butyric acid systems in the spinal cord may be important intermediaries in SCS-induced pain relief. In arterial insufficiency SCS have been shown to decrease rest pain (Fontaine class III), improve claudication distance, raise skin temperature, and increase transcutaneous oxygen tension in the forefoot, with values of >10 mm Hg before treatment being associated with significantly better outcome.
- Arterial bypass of the larger vessels may be used in selected instances.
- Ultimate surgical therapy for refractory Buerger's disease is distal limb amputation for nonhealing ulcers, gangrene or intractable pain.

- Recently, Isner and colleagues reported improved healing of ischemic ulcers and relief of rest pain in a small series of patients with Buerger's disease using intramuscular gene transfer of vascular endothelial growth factor.

COMPLICATIONS

- Ulcerations
- Gangrene
- Infection
- Need for amputation.

PREVENTION OF COMPLICATIONS

- Use of well-fitting protective footwear to prevent foot trauma and thermal or chemical injury.
- Early and aggressive treatment of extremity injuries to protect against infections.
- Avoidance of cold environments.
- Avoidance of drugs that lead to vasoconstriction.

6 Venous Disorders

6.1 SUPERFICIAL VEIN THROMBOSIS

DEFINITION

Superficial vein thrombosis (SVT) is defined as thrombosis and inflammation of inner walls of the greater or lesser saphenous veins or their tributaries.

ETIOLOGY

- Trauma to the vessel wall—a drip needle or pressure externally due to tight garments or position of a limb.
- Circulating toxins from septic wounds.
- In association with deep venous thrombosis (DVT).
- It is associated with intravenous catheters and infusions.
- Occurs with varicose veins.
- Migrating SVT is often a marker for a carcinoma.
- May also occur in patients with vasculitides, such as thromboangiitis obliterans (TAO).

PATHOLOGY

Irritation produced changes in the tunica intima causing a thrombus to form. The thrombus becomes attached to the vein wall and rarely produces an embolus.

CLINICAL FEATURES

It can easily be distinguished from those of DVT. Patients complain of pain localized to the site of the thrombus. Examination reveals a reddened, warm and tender cord extending along a superficial vein. The surrounding area may be red and edematous. As the condition resolves, the skin become pigmented (brown) along the course of the vein.

INVESTIGATIONS

Phlebography is used to find out the thrombosis.

TREATMENT

Treatment is primarily supportive:

- Initially, patients can be placed at bedrest with leg elevation.
- Application of compression bandage in the form of crepe bandage or stockings from the toes to beyond the upper limit of the affected area.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) may be provided to relieve pain and inflammation.
- Antibiotics in case of infective phlebitis.
- *Anticoagulant therapy*: If a thrombosis of the greater saphenous vein develops in the thigh and extends toward the saphenofemoral vein junction and to prevent extension of the thrombus into the deep system and a possible pulmonary embolism.

6.2 DEEP VENOUS THROMBOSIS

DEFINITION

The presence of thrombus within a deep vein and the accompanying inflammatory response in the vessel wall is termed as *deep venous thrombosis* (DVT) or *thrombophlebitis*.

INCIDENCE

Deep vein thrombosis occurs less frequently in the upper extremity than in the lower extremity, but the incidence is increasing, because of greater utilization of indwelling central venous catheters.

ETIOLOGY

The factors that predispose to venous thrombosis were initially described by Virchow in 1856 and include stasis, vascular damage and hypercoagulability.

RISK FACTORS

- Recent surgery
- Neoplasms
- Trauma
- Fractures
- Immobilization
- Acute myocardial infarction (MI), congestive heart failure (CHF), stroke

- Postoperative convalescence
- Pregnancy
- Estrogen use (for replacement or contraception)
- Hypercoagulable states
- Previous DVT.

PATHOGENESIS

Damage to the intima causes platelets to be deposited on the vein wall. Venous stasis increases the accumulation of platelets, which adds to the size of the thrombus resulting in occlusion of the vessel lumen. Initially, the thrombus is composed principally of platelets and fibrin. Red cells become interspersed with fibrin and the thrombus tends to propagate in the direction of blood flow. The inflammatory response in the vessel wall may be minimal or characterized by granulocyte infiltration, loss of endothelium and edema. There is further extension of the thrombus (propagated thrombus) along the vessel to the next junction with a vein. A portion may break off giving rise to a pulmonary embolus or the thrombus may become organized and firmly attached to the vessel wall. Gradually, it is recanalized and circulation is re-established, but the valves are often destroyed leaving chronic venous insufficiency (CVI).

CLINICAL FEATURES

- Most common complaint is cramp-like pain in the calf.
- Unilateral leg swelling (edema around the joint distal to the area).
- Local warmth.
- Erythema.
- Tenderness may be present along the course of the involved veins.
- Palpable cord.
- Increased tissue turgor.
- Distention of superficial veins.
- Appearance of prominent venous collaterals.
- In some patients, deoxygenated hemoglobin in stagnant veins impart a cyanotic hue to the limb, a condition called *phlegmasia cerulea dolens.*
- In markedly edematous legs, the interstitial tissue pressure may exceed the capillary perfusion pressure, causing pallor, a condition designated as *phlegmasia alba dolens.*
- Unexplained systemic features, e.g. mild pyrexia, pleuritic pain, tachycardia in a patient recovering from surgery.
- Severe pulmonary embolus giving signs of extreme distress, breathlessness and shock may be the first indication of DVT.

Examination may reveal posterior calf tenderness, warmth, increased tissue turgor or modest swelling and rarely a cord. Cuff test, Homan's sign and Mose's sign will be positive.

INVESTIGATIONS

- D-Dimer, a degradation product of cross-linked fibrin is often elevated in patients with venous thrombosis.

- *Duplex venous ultrasonography:* By imaging the deep veins, thrombus can be detected either by direct visualization or by inference when the vein does not collapse on compressive maneuvers.
- The Doppler ultrasound measures the velocity of blood flow in veins. This velocity is normally affected by respiration and by manual compression of the foot or calf. Flow abnormalities occur when deep venous obstruction is present.
- *Magnetic resonance imaging (MRI):* It is useful in patients with suspected thrombosis of the superior and inferior vena cava or pelvic veins.
- *Venography*: Contrast medium is injected into a superficial vein of the foot and directed to the deep system by the application of tourniquets. The presence of a filling defect or absence of filling of the deep veins is required to make the diagnosis.

DIFFERENTIAL DIAGNOSIS

Deep vein thrombosis must be differentiated from a variety of disorders that cause unilateral leg pain or swelling, including muscle rupture, trauma or hemorrhage; a ruptured popliteal cyst and lymphedema. It may be difficult to distinguish swelling caused by the postphlebitic syndrome from that due to acute recurrent DVT. Leg pain may also result from nerve compression, arthritis, tendinitis, fractures and arterial occlusive disorders.

TREATMENT

- Bedrest with a cradle and the end of the bed elevated (15–22 cm) until all the local signs subside may be up to 7 days.
- *Anticoagulants prevent thrombus propagation and allow the endogenous lytic system to operate:*
  - This includes either unfractionated heparin or low-molecular-weight heparin (LMWH).
  - A direct thrombin inhibitor, such as lepirudin or argatroban may be used as initial anticoagulant therapy for patients in whom heparin is contraindicated, because of heparin-induced thrombocytopenia (HIT).
  - Warfarin is administered during the first week of treatment with heparin and may be started as early as the first day of heparin treatment.
- *Thrombolytics*: Thrombolytic drugs such as streptokinase, urokinase and tissue plasminogen activator (tpA) may also be used.
- *Vena cava filter* (*Greenfield filter*): To prevent pulmonary embolism.

PROPHYLAXIS

Prophylaxis should be considered in clinical situations where the risk of DVT is high.

- Low-dose unfractionated heparin (5,000 units 2 h prior to surgery and then 5,000 units every 8–12 h postoperatively).
- Warfarin.

- External pneumatic compression (TED antiembolic stockings) applied to the legs are used to prevent DVT.

COMPLICATIONS

- Pulmonary embolism
- Chronic venous insufficiency.

6.3 VARICOSE VEINS

DEFINITION

Varicose veins are dilated, tortuous superficial veins that result from defective structure and function of the valves of the saphenous veins.

INCIDENCE

- The most common in 40–50 years
- Females are more affected than males.

TYPES

Varicose veins can be categorized as primary or secondary. Primary varicose veins originate in the superficial system and occur two to three times as frequently in women as in men. Approximately half of patients have a family history of varicose veins. Secondary varicose veins result from deep venous insufficiency and incompetent perforating veins or from deep venous occlusion causing enlargement of superficial veins that are serving as collaterals.

ETIOLOGY

- Failure of development of valve in the vein
- Damage to the valve due to thrombosis
- From intrinsic weakness of the vein wall
- From high intraluminal pressure
- Rarely from arteriovenous fistulas.

PREDISPOSING FACTORS

- *Pregnancy*:
  - Compression of pelvic vein due to enlarged womb **(Fig. 6.3.1)**
  - Estrogens relax the muscles in the veins and this also increases the tendency of the veins to expand.
- Occupation necessitating constant standing, e.g. shop assistant, traffic police, teachers, etc.

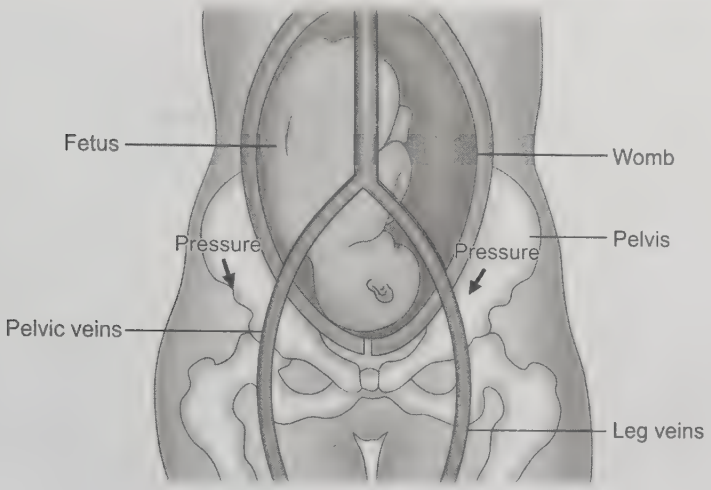


Fig. 6.3.1 Uterus pressing the pelvic veins

- Low-fiber diet, which results in constipation. If the bowel is loaded with constipated stool it compresses the large (iliac) veins in the pelvis, which in turn dilates the veins of the leg.
- *Constipation:* Staining during constipation builds up pressure and transmits the pressure to the legs. This increased pressure build-up in the leg veins over the years will lead to a deterioration of the valves and hence, will lead to varicose veins.
- Tight corsets or garters.
- Contraceptive pills which have estrogen.
- Family history.
- Secondary to DVT.

PATHOLOGY (FIG. 6.3.2)

The vein wall dilates at weak areas and the valves become incompetent. Normally as the calf muscle contracts, there is pressure on deep veins which forces the blood proximally. This pressure is not transmitted to the superficial veins, because of valves in communicating veins. When these valves become incompetent the pressure pushes the blood into a superficial vein, which dilates and lengthens. A vicious cycle is set up, the ineffectual valves permitting regurgitation and the increasing amount of blood thus left in the veins still further dilating them and making the valves more incompetent.

During standing, the force of gravity tends to keep the blood in lower parts of the body, aggravating the condition. There is loss of elastic tissue, muscle atrophy of the media layer and hypertrophy of the outer layer. Dilated veins and abnormally high pressure in the capillaries increases exudation of lymph, which results in edema.

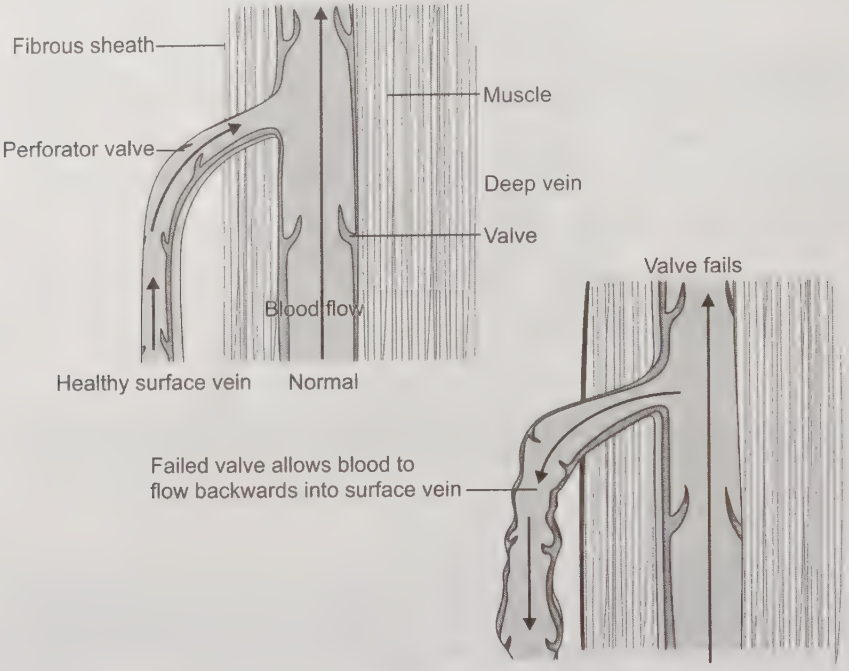


Fig. 6.3.2 Varicose vein caused by valve failure

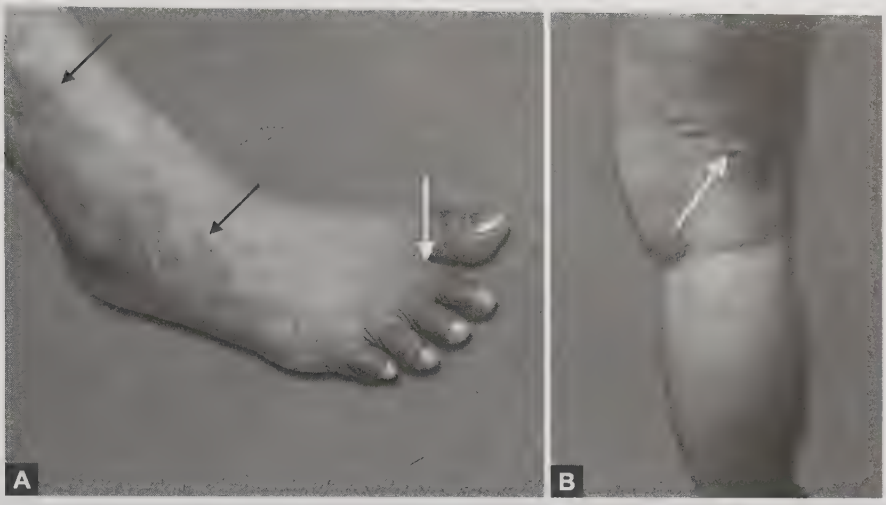
CLINICAL FEATURES

- Patients with venous varicosities are often concerned about the cosmetic appearance of their legs.
- Patient may complain of a dull ache, pain or pressure sensation in the legs after prolonged standing; it is relieved with leg elevation.
- Fatigue in the legs with difficulty in walking.
- Cramp in the calf muscles, especially at night.
- The legs feel heavy and mild ankle edema develops occasionally.
- The skin of the leg may become pigmented and indurated **(Figs 6.3.3A and B)**.
- Extensive venous varicosities may cause skin ulcerations near the ankle.
- Superficial venous thrombosis may be a recurring problem.
- Rarely, a varicosity ruptures and bleeds.

Visual inspection of the legs in the dependent position usually confirms the presence of varicose veins.

INVESTIGATIONS

- *Doppler ultrasound scan:* It can easily identify reflux or back-flow of blood in the veins.
- *Varicogram*: A cuff will be placed around the lower calf and the dye injected into the veins on the back of the foot. Usually, the site of connection of the varicose vein to the deep venous system need to be identified and the



Figs 6.3.3A and B (A) Varicose vein on female's leg, black arrows: dilated vein; white arrow: skin changes; (B) Varicose vein on female's thigh, white arrow: spider vein *(For color version, see Plate 2)*

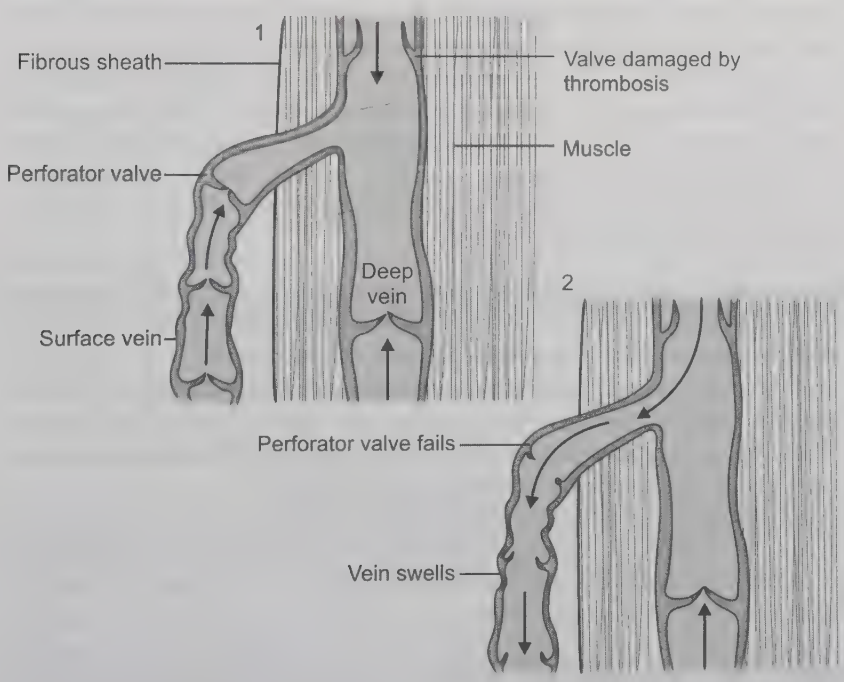


Fig. 6.3.4 Varicose veins caused by deep vein damage

needle is then placed within the varicosity. If the test is done to look at whether the valves in the leg are working, the needle may be placed either in the foot or in the groin, and the table will be tilted to see if the dye passes backwards through the valves **(Fig. 6.3.4)**.

CONSERVATIVE TREATMENT

- External compression stockings has to be worn which provide a counter-balance to the hydrostatic pressure in the veins.
- Walking is encouraged.
- Avoid prolonged standing.
- Symptoms often decrease when the legs are elevated periodically.
- *Laser therapy*: The laser pulse is of a very short duration and destroys the veins, which are broken into very small particles. These are then removed by the body's immune system. There may be some reaction to the laser for the first 24 hours, with redness at the site, but this resolves quite quickly. This treatment is given on an out-patient basis and does not require any anesthesia. The complications of this therapy include pigmentation at the site and loss of pigment at the site. There is usually loss of hair growth in the area if the laser treatment is repeated. Very occasionally, there may be some crusting of the skin together with blistering. But these side-effects do not last long.
- *Endovenous obliteration of the saphenous vein*: A newer treatment for varicose veins is to insert a long, thin catheter that emits energy (most commonly heat, radiowaves or laser energy). The released energy collapses and scleroses the vein. A variety of techniques and protocols are used. Because it is easier to insert a catheter through a vein in the same direction that the valves open, the catheter is most commonly inserted into a more distal portion of the vein and threaded proximally. Energy is released from the catheter tip. As the catheter is pulled out, the vein lumen collapses. Bruising, tightness along the course of the treated vein, recanalization and paresthesia are possible complications.

SURGICAL MANAGEMENT

Surgical therapy may also be indicated for cosmetic reasons:

- *Sclerotherapy*: It is a procedure in which a sclerosing solution is injected into the involved varicose vein and a compression bandage is applied. The sclerosant solution produces inflammation in the vein so that no blood can pass through causing the lumen to be obliterated.
- Surgical therapy usually involves extensive ligation and stripping of the greater and lesser saphenous veins and should be reserved for patients, who are very symptomatic, suffer recurrent superficial vein thrombosis, and/or develop skin ulceration.

COMPLICATIONS

- *Hemorrhage*: Bleeding following rupture of a vein.
- *Ulceration*: Venous ulcer due to devitalized skin.
- *Phlebitis*: Superficial venous thrombosis.
- Edema particularly of the foot and ankle.
- Pigmentation.

6.4 PULMONARY EMBOLISM

DEFINITION

The term 'pulmonary embolism' implies clinically significant obstruction of a part or the whole of the pulmonary arterial tree, usually by thrombus that becomes detached from its site of formation outside the lung and is swept downstream until arrested at points of intrapulmonary vascular narrowing.

TYPES

- Thrombotic pulmonary embolism
- Nonthrombotic pulmonary embolism.

Thrombotic Pulmonary Embolism

As a complication of deep venous thrombosis: If a thrombosis breaks off in a deep vein it travels in the venous system to the right side of the heart, where it enters the pulmonary artery and passes into the pulmonary circulation where it blocks a vessel, the lumen of which is too narrow to let it pass through. The factors that predispose to a deep vein thrombosis also predispose to a pulmonary embolism. Stasis being the most important initiating factor in initiating and venous thrombi rather than any damage to vessel wall.

Other: A small proportion of pulmonary emboli may arise from the pelvic veins including the prostatic venous plexus in men and also from the right side of the heart (following myocardial infarction and in right ventricular failure).

Septic pulmonary emboli may arise from bacterial endocarditis in patients with septal defects, from the tricuspid valve in drug abusers, and from foreign material such as central venous lines, ventriculoatrial and arteriovenous shunts, internal cardiac defibrillators and pacemaker wires.

Nonthrombotic Pulmonary Embolism

Fat embolism: It is probably a common subclinical event following bony trauma. Following such trauma, neutral fat may pass into the circulation from injured long bones, be carried to the lungs and become lodged in the pulmonary vasculature.

Tumor emboli: Pulmonary vessels are occluded by macroscopic emboli, but it differs in its lack of responsiveness to anticoagulation. Tumors that have been implicated most frequently include carcinoma of the breast, stomach, colon and cervix, hepatomas, choriocarcinomas and hypernephromas.

Air embolism: It may be the result of faulty cannulation of the neck veins, therapeutic insufflation of air into a fallopian tube or intrauterine manipulations, including criminal abortion in which a frothy solution may be introduced into the uterus under pressure. Small amount of air are reabsorbed without harm,

but large amounts can cause mechanical obstruction of the pulmonary circulation and death. The subject of air embolism in diving is also another case.

Amniotic fluid embolism: It is a serious complication of pregnancy. It usually arises during labor or cesarian section, but is not predictable or recognized as occurring because of any particular mishap. It causes respiratory distress with pulmonary edema and shock.

PATHOPHYSIOLOGICAL EFFECTS

- Increased pulmonary vascular resistance due to vascular obstruction or platelet secretion of neurohumoral agents including serotonin. This increases the afterload on the right ventricle, which further results in right ventricular failure. This produces a decline in pulmonary blood flow and in turn reduced filling of the left ventricle, which is no longer able to maintain a normal systolic blood pressure.
- *V/Q mismatch and impaired gas exchange*: Pulmonary embolism produces mismatching of ventilation and perfusion by preventing blood in the pulmonary artery from reaching the ventilated lung (increased alveolar dead space) and paradoxically by interfering with the ventilation of lung that is still perfused (increased intrapulmonary shunting). Due to increased alveolar dead space, right-to-left shunting from vascular obstruction, impaired carbon monoxide transfer due to loss of gas exchange surface leads to impaired gas exchange.
- Alveolar hyperventilation due to reflex stimulation of irritant receptors.
- Increased airway resistance due to constriction of airways distal to the bronchi.
- Decreased pulmonary compliance due to lung edema, lung hemorrhage or loss of surfactant.

Increased right ventricular wall tension also compresses the right coronary artery, and may precipitate myocardial ischemia and right ventricular infarction. The interventricular septum bulges, which under fills of the left ventricle results in fall of left ventricular output and systemic arterial pressure, thereby provoking myocardial ischemia due to compromised coronary artery perfusion. Eventually results in circulatory collapse and death.

CLINICAL SYNDROMES

Types of pulmonary embolism:

- *Massive pulmonary embolism*:
  - Systemic hypotension
  - Wide spread thromboembolism
  - Abnormal right heart function.
- *Moderate to large pulmonary embolism*:
  - Normal systemic arterial blood pressure
  - Right ventricular hypokinesia (abnormal right heart function).
- *Small to moderate pulmonary embolism*:
  - Normal right heart function

  - Normal systemic blood pressure
  - But pulmonary infarction present.
- *Chronic pulmonary embolism*:
  - Chronic occlusion of pulmonary microvasculature
  - Abnormal right heart function.

CLINICAL FEATURES

Patients with a history of active cancer, paralysis or immobilization of a leg, more than 3 days in bed or major surgery within 4 weeks, and a strong family history of venous thrombosis; major signs are tenderness over the leg veins and swelling of calf and thigh, or more than 3 cm swelling of calf.

Symptoms of pulmonary embolism may be immediately preceded by straining, such as during defecation, or by other minor physical exertion, such as that involved in getting out of bed or even laughing. These patients are dyspneic, restless, anxious, confused and cyanosed.

- *Dyspnea* is the most frequent symptom of pulmonary embolism (PE). The possible cause of dyspnea is stimulation of pressure receptors in pulmonary vasculature or right atrium. It is of sudden onset. Breathlessness is present both lying and sitting, but is associated with no auscultatory signs in the lung, a most important negative feature. Shoulder will be raised to allow the increased lung air flow.
- *Chest pain and chest tightness*: It may be abrupt onset lasts for several minutes to few hours, pleuritic often lateral on the side of embolism. Pain worse with deep breath or cough. Chest pain due to pulmonary embolism is believed to be due to distention of the pulmonary artery or infarction of a segment of the lung adjacent to the pleura. Massive pulmonary emboli may lead to substernal pain that is suggestive of acute myocardial infarction. Central chest pain as a result of reduced coronary artery perfusion may also occur. More commonly, smaller emboli lead to focal pulmonary infarctions which cause pain, i.e. lateral and pleuritic.
- *Syncope* occurs in up to 10% of patients with massive pulmonary embolism.
- *Cough* could be nonproductive, or productive of purulent or bloody sputum.
- *Hemoptysis* following the acute onset of pleuritic chest pain and dyspnea is suggestive of pulmonary embolism. If hemoptysis occurred, the sputum typically was blood-streaked, but can be pure blood or blood tinged.
- *Palpitation*: Patient may often compliant of palpitation.
- *Tachypnea* is its most frequent sign.
- *Hypotension* in case of massive pulmonary embolism.
- *Sweating, light headedness.*
- *Low grade fever*: Pyrexia may be present and is very occasionally the dominant finding until anticoagulation is commenced.
- *Jugular venous engorgement and neck vein distention*
- *Auscultation*:
  - An accentuated pulmonary component of the second heart sound depends on the degree of pulmonary hypertension produced

- Diminished or absent breath sounds
- Pleural friction rub
- Occasionally, wheeze may be heard as a result of secondary bronchoconstriction.

Wells criteria for pulmonary embolism and Geneva score for pulmonary embolism are used as tools to objectify the risk of pulmonary embolism.

INVESTIGATIONS

Blood test: White blood cell (WBC) count (leukocytosis), bleeding time, clotting time (to check hypercoagulable states).

Serological tests, D-dimer (ELISA): Commonly used as a screening test in patients with a low and moderate probability clinical assessment, on these patients. A normal D-dimer excludes pulmonary embolism, no further testing is required. A raised D-dimer is seen with pulmonary embolism but has many other causes and is therefore nonspecific; it indicates the need for further testing, if pulmonary embolism is suspected.

Chest X-ray: Pulmonary embolism may give rise to a variety of radiographic appearances such as:

- *Fleischner sign*: Enlarged pulmonary artery
- *Hampton hump*: Peripheral wedge of airspace opacity and implies lung infarction
- *Westermark's sign*: Hyperlucency due to regional oligemia
- *Knuckle sign*: Refers to the abrupt tapering or cutoff of a pulmonary artery secondary to embolus
- *Opacities*: Horizontal linear opacities, bilateral in lower zone
- *Signs of pleural effusion*: Blunting of costophrenic angles.

Arterial blood gases: Classical finding shows a reduced PaO_2 and a normal or low $PaCO_2$ (respiratory alkalosis). A mixed acidosis (respiratory and metabolic) may also be seen in acute massive pulmonary embolism with cardiovascular collapse.

Electrocardiogram (ECG) findings McConnell's sign: The QRS axis may shift to the right, sometimes in concert with the so-called S1Q3T3 pattern (prominence of the S wave in lead I, Q wave in lead III, with T-wave inversion in lead III). T-wave inversion may be found in the anterior chest leads implying right ventricular strain. In severe cases, this pattern is associated with prolongation of the QRS complex, mimicking right bundle branch block.

Echocardiography (ECHO): Show changes of acute right heart strain with ventricular hypokinesia and tricuspid regurgitation from which pulmonary artery pressure may be estimated.

Ventilation/Perfusion scan: Pulmonary thromboembolism usually produces one or more regions of ventilation-perfusion mismatch, i.e. a region in which there is a defect in perfusion that follows the distribution of a vessel and that is not accompanied by a corresponding defect in ventilation.

Pulmonary angiography: The pulmonary arterial system can be visualized by pulmonary angiography in which radiopaque contrast medium is injected through a catheter previously threaded into the pulmonary artery. When performed in cases of pulmonary embolism, pulmonary angiography demonstrates the consequences of an intravascular clot—either a defect in the lumen of a vessel (a 'filling defect') or an abrupt termination (cutoff) of the vessel.

MEDICAL AND SURGICAL MANAGEMENT

Supplemental oxygen therapy: All patients who are hypoxic should be given supplementary oxygen at high concentration (enough to restore normal PO_2). In the early stages, continuous monitoring of arterial oxygen tension by pulse oximetry is advised.

Resuscitation: The patients presenting with massive pulmonary embolism may have circulatory collapse. The aim is to support the circulation until measures designed to deal with the embolus (thrombolysis) can be applied and take effect.

Anticoagulants: The anticoagulants such as heparin, LMWH, and fondaparinux (new parenteral synthetic anticoagulant), which are administered subcutaneously or intravenously; and coumarin compounds, which are given orally. Fibrinolytics are also administered.

Thrombolytic therapy: It is not indicated for the routine treatment of pulmonary embolism. Hypotension and continuing hypoxemia, while receiving high fractions of inspired oxygen (FiO_2) are indications for intervention. Right ventricular dysfunction on the echocardiogram may also be an indication. Streptokinase, urokinase or tissue plasminogen activator are commonly administered.

Pharmacologic support: Diuretics, dobutamine, digoxin should be administered to support cardiovascular support. The use of prostaglandins such as epoprostenol (prostacyclin) as endogenous vasodilators or iloprost therapy has dramatically improved exercise performance, symptoms and prognosis.

Respiratory support: Patients are intubated and mechanical ventilator may be needed to support respiratory system.

Inferior vena cava occlusion: An inferior vena cava filter is recommended in a patient with proximal deep venous thrombosis or pulmonary embolism, if anticoagulants are contraindicated or pulmonary embolism has recurred, while on adequate anticoagulant therapy or pulmonary embolism is severe.

Catheter interventions: Catheter-tip devices for the extraction or the fragmentation of embolus have the potential of producing immediate relief from massive pulmonary embolism. Such interventions may be particularly useful in patients in whom there is a contraindication to thrombolytic therapy.

Pulmonary embolectomy: It is a procedure to remove one or more large blood clots (emboli) from the lungs. Indications are massive pulmonary embolism, hemodynamic instability (shock) despite heparin therapy and resuscitative efforts, and failure of thrombolytic therapy or a contraindication to its use.

Pulmonary thromboendarterectomy: It is a surgery that removes organized clotted blood (thrombus) from the pulmonary arteries. Patients who develop chronic pulmonary hypertension due to prior pulmonary embolism may become severely dyspneic at rest or with minimal exertion are considered for pulmonary thromboendarterectomy.

PREVENTION

Mechanical measures such as stockings, early ambulation and medicinal measures already described in chapter DVT.

PHYSIOTHERAPY MANAGEMENT

Problem List

- Exercise limitation
- Shortness of breath
- Oxygen deprivation
- Limited endurance capacity.

Goals

- To improve oxygenation
- To ambulate more without much breathlessness.

Intervention

- Oxygen therapy
- *Rest*: Bedrest with the end of the bed elevated. Patient is allowed up when all symptoms have disappeared
- Smoking cessation techniques
- Inspiratory muscle training
- Aerobic, strength, flexibility, posture, breathing to return to prior level of functioning
- As the physiotherapist can recognize the signs of a pulmonary embolus, so that treatment can be instigated early, physiotherapy in the form of active exercises to the lower limb and early ambulation are important preventative measures.

6.5 CHRONIC VENOUS INSUFFICIENCY

DEFINITION

The term chronic venous insufficiency (CVI) describes a condition that affects the venous system of the lower extremities with venous hypertension causing various pathologies including pain, swelling, edema, skin changes and

ulcerations. Although the term CVI is often used to exclude uncomplicated varicose veins, varicose veins have incompetent valves with increased venous pressure leading to progressive dilation and tortuosity.

RISK FACTORS

Risk factors found to be associated with CVI include age, sex, a family history of varicose veins, obesity, pregnancy, phlebitis, and previous leg injury. There also may be environmental or behavioral factors associated with CVI such as prolonged standing and perhaps a sitting posture at work.

PATHOGENESIS

Venous pathology develops when venous pressure is increased and return of blood is impaired through several mechanisms. This can result from valvular incompetence of the deep or superficial or perforator vein, venous obstruction, or a combination of these. These factors are exacerbated by muscle pump dysfunction in the lower extremity. These mechanisms serve to produce venous hypertension particularly with standing or ambulation.

With failure of the valves of the deep veins, normal blood volume is pumped out of the extremity, but refill occurs by both arterial inflow and pathological retrograde venous flow. The venous pressure immediately after ambulation may be slightly elevated or even normal, but veins refill quickly with the development of high venous pressure without muscle contraction.

Dysfunction or incompetence of the valves in the superficial venous system also allows retrograde flow of blood and increased hydrostatic pressures. Failure of valves located at the junctions of the deep and superficial systems, most notably at the saphenofemoral and saphenopopliteal junctions, allows high pressure to enter the superficial veins. In this situation, venous dilatation and varicose veins form, and propagate from the proximal junction site down the extremity.

High pressure also can enter the superficial system because of failure of the valves in the communicating perforator veins. Perforator valve incompetence allows blood to flow from deep veins backward into the superficial system and the transmission of the high pressures generated by the calf muscle pump. This local high pressure can produce excessive venous dilatation and secondary failure of superficial vein valves. As a result, a cluster of dilated veins develops at this site and appears to ascend up the leg.

Obstruction of the deep veins may limit the outflow of blood, causing increased venous pressure with muscle contraction and secondary muscle pump dysfunction. Obstruction may occur because of an intrinsic venous process, such as previous DVT with inadequate recanalization or venous stenosis, or because of extrinsic compression, as in May-Thurner syndrome (compression of the left common iliac vein as it traverses between the right common iliac artery and the lumbosacral region). Venous outflow obstruction appears to play a more significant role in the pathogenesis of CVI and its clinical expression than previously appreciated.

Changes in the macrocirculatory hemodynamics of the large veins of the lower extremity are transmitted into the microcirculation and eventually result in the development of venous microangiopathy. Features of this microangiopathy include elongation, dilation, and tortuosity of capillary beds, thickening of basement membranes with increased collagen and elastic fibers, endothelial damage with widening of inter endothelial spaces, and increased pericapillary edema. The abnormal capillary with increased permeability and high venous pressure leads to the accumulation of fluid macromolecules, and extravagated red blood cells into the interstitial space. In addition to changes in the blood vessels and connective tissue, alteration in the lymphatic network and nervous system may occur. Fragmentation and destruction of microlymphatics may further impair drainage from the extremity, whereas dysfunction of local nerve fibers may alter regulatory mechanisms. Venous hypertension may result in dermal changes with hyperpigmentation, subcutaneous tissue fibrosis (lipodermatosclerosis), and eventual ulceration.

Several mechanisms for the development of venous microangiopathy have been postulated, including fibrin cuff formation, growth factor trapping, and white blood cell trapping.

- The fibrin cuff theory involves the accumulation of fluid containing fibrin into the pericapillary space. This cuff with impaired fibrinolysis is speculated to increase the diffusion barrier, inhibit the repair process, and maintain the inflammatory process.
- A related mechanism is the trapping of growth factor by fibrin and other macromolecules, making them unavailable to facilitate healing.
- Another theory involves the trapping of white blood cells in the capillaries or postcapillary venules. The adhesion of white blood cells with activation releases inflammatory mediators and proteolytic enzymes with endothelial damage that may increase permeability or impede flow leading to occlusion.

CLINICAL FEATURES

The CVI represents a spectrum of conditions ranging from simple telangiectasia or reticular veins to more advanced stages such as skin fibrosis and venous ulceration. It is important to realize that the same clinical manifestations may result from the varied pathogenic mechanisms (i.e. incompetent valves alone, venous obstruction alone, muscle pump dysfunction alone or a combination).

The major clinical features of CVI are dilated veins, edema, leg pain and cutaneous changes.

- Varicose veins are dilated superficial veins that become progressively more tortuous and larger. They are prone to develop bouts of superficial thrombophlebitis.
- Edema begins in the perimalleolar (or goiter) region, but ascends up the leg with dependent accumulation of fluid.
- The leg pain or discomfort is described as heaviness or aching after prolonged standing and relieved by elevation of the leg. Edema presumably produces the pain by increasing intracompartmental and subcutaneous volume and pressure.

- *Venous claudication:* Obstruction of the deep venous system may lead to venous claudication or intense leg cramping with ambulation.
- There may be tenderness along varicose veins from venous distention.
- Cutaneous changes include skin hyperpigmentation from hemosiderin deposition and eczematous dermatitis.
- Fibrosis may develop in the dermis and subcutaneous tissue (lipodermatosclerosis).
- There is an increased risk of cellulitis, leg ulceration and delayed wound healing.
- Long-standing CVI also may lead to the development of lymphedema, representing a combined disease process.

The manifestations of CVI may be viewed in terms of a well-established clinical classification scheme. The clinical, etiology, anatomic, pathophysiology (CEAP) classification provides a basis for uniformity in reporting, diagnosing, and treating CVI **(Table 6.5.1)**.

The clinical classification has seven categories (0–6) and is further categorized by the presence or absence of symptoms.

The etiologic classification is based on congenital, primary and secondary causes of venous dysfunction. Congenital disorders are those that are present at birth, although they may be recognized later in life, including the well-recognized Klippel-Trenaunay (varicosities and venous malformations, capillary malformation, and limb hypertrophy) and Parkes-Weber (venous and lymphatic malformations, capillary malformations, and arteriovenous fistulas) syndromes. The cause of primary venous insufficiency is uncertain, whereas secondary venous insufficiency is the result of an acquired condition.

The anatomic classification describes the superficial, deep, and perforating venous systems, with multiple venous segments that may be involved.

Table 6.5.1 CEAP classification of chronic venous disease

Classification	*Type*	*Description /Definition*
Clinical	0	No venous disease
	1	Telangiectasia
	2	Varicose veins
	3	Edema
	4	Lipodermatosclerosis or hyperpigmentation
	5	Healed ulcer
	6	Active ulcer
Etiologic	Congenital	Present since birth
	Primary	Undetermined etiology
	Secondary	Associated with post-thrombotic, traumatic
Anatomic distribution (alone or in combination)	Superficial	Great and short saphenous veins
	Deep	Cava, iliac, gonadal, femoral, profunda, popliteal, tibial, and muscular veins
	Perforator	Thigh and leg perforating veins
Pathophysiological	Reflux	Axial and perforating veins
	Obstruction	Acute and chronic
	Combination of both	Valvular dysfunction and thrombus

The patho physiological classification describes the underlying mechanism resulting in CVI, including reflux, venous obstruction, or both.

EXAMINATION FINDINGS

Observation shows hyperpigmentation, stasis dermatitis, atrophic blanching (white scarring at the site of previous ulcerations with a paucity of capillaries) or lipodermatosclerosis. The distribution of varicose veins may follow the course of the affected superficial vein, such as the great or short saphenous veins. Inspection and palpation may reveal visual evidence for chronic venous disease.

Palpation also may reveal tenderness of the dilated veins. The surface of the skin is examined for irregularities or bulges to suggest the presence of varicose veins.

Examination should include an evaluation of the patient in the upright posture to allow maximal distention of the veins and from multiple directions. The presence of edema and its severity are assessed. Edema is usually pitting; however, early evidence may be calf fullness or increased limb girth, so the calf muscle consistency should be assessed and measurement of the limb girth should be performed. Long-standing edema may become more resilient to palpation with 'brawny' edema. Active or healed ulcers are seen with more advanced disease. The venous ulcers usually occur in the medial supramalleolar area at the site of major perforating veins and the greatest hydrostatic pressure. Trendelenberg test may be performed to determine the distribution of venous insufficiency, this test does not help determine the extent or severity of disease or provide information about the cause.

INVESTIGATIONS

Continuous wave Doppler studies: The presence and direction of flow in the veins may be determined after maneuvers, such as the Valsalva maneuver or the sudden release of thigh or calf compression. Minimal signal should be detected toward the feet with these maneuvers.

Venous duplex imaging: It is used to confirm the diagnosis of CVI and assess its etiology and severity. The direction of flow may be assessed in a 30° reverse Trendelenberg position during provocative maneuvers, such as the Valsalva maneuver or after augmenting flow with limb compression. The use of a cuff inflation-deflation method with rapid cuff deflation in the standing position is preferred to induce reflux.

Photoplethysmography (PPG): It may be used to establish a diagnosis of CVI. Relative changes in blood volume in the dermis of the limb can be determined by measuring the backscatter of light emitted from a diode with a photosensor. A PPG probe is placed on the foot with maneuvers to empty the foot with calf muscle contraction. Then return of blood is detected by increased backscatter of light and the refill time may be calculated. The venous refill time is the time required for the PPG tracing to return to 90% of the baseline after cessation of

calf contraction. A venous refill time less than 18–20 seconds, depending on the patient's position during the study, is indicative of CVI.

Air plethysmography (APG) has the ability to measure each potential component of the pathophysiological mechanisms of CVI—reflux, obstruction, and muscle pump dysfunction. A normal venous filling index is less than 2 mL/s, whereas higher levels of 4–7 mL/s have been found to correlate with the severity of CVI.

Phlebography or venography: It may be either ascending or descending. Ascending phlebography involves the injection of contrast in the dorsum of the foot with visualization of contrast traveling up the lower extremity in the deep venous system. Descending phlebography involves proximal injection of contrast in a semivertical posture on a tilt table with the use of the Valsalva maneuver.

Ambulatory venous pressure (AVP) monitoring: It is the hemodynamic gold standard in assessing CVI. The technique involves insertion of a needle into the pedal vein with connection to a pressure transducer. The pressure is determined at rest and after exercise is performed, usually in the form of toe raises. The pressure also is monitored before and after the placement of an ankle cuff to help distinguish deep from superficial reflux. AVP has been shown to be valuable in assessing the severity and clinical outcomes in CVI. The ambulatory venous pressure (normal range of 20–30 mm Hg) and refill time (normal range of 18–20 seconds) are the most useful measurements.

DIFFERENTIAL DIAGNOSIS

There is a broad differential for the common presenting complaint in limb swelling and discomfort seen with CVI. Acute venous problems such as DVT need to be excluded. Systemic causes of edema need to be considered such as heart failure, nephrosis, liver disease, endocrine disorders, or a side effect of a medication, such as calcium channel blockers, nonsteroidal anti-inflammatory agents, and oral hypoglycemic agents. Other regional considerations include a ruptured popliteal cyst, soft tissue hematoma or mass, chronic exertional compartment syndrome, a gastrocnemius tear, or lymphedema.

TREATMENT

Conservative

- Elevating the legs to minimize edema and reducing intra abdominal pressure should be advocated.
- *Compressive leg garments*: A number of compression garments are available including graded elastic compressive stockings, paste gauze boots, layered bandaging, and adjustable layered compression garments. The use of graded elastic compressive stockings (with 20–50 mm Hg of tension) is well established in the treatment of CVI. The prescription for elastic compression stockings in CVI includes information about the tension and length.

The tension is based on the clinical severity, with 20 to 30 mm Hg for CEAP classes 2 to 3, 30 to 40 mm Hg for CEAP classes 4 to 6, and 40 to 50 mm Hg for recurrent ulcers. The most common length is knee-length stockings because patient adherence is greater and symptom relief is adequate. The use of thigh- or waist-high stockings may be necessary in patients with edema extending above the knee, but these stockings are more difficult to use.

Pharmacological Therapy

Four groups of drugs have been evaluated in the treatment of CVI including coumarins (alpha benzopyrones), flavonoids, saponosides (horse chestnut extracts), and other plant extracts. These drugs have venoactive properties. The principle for the use of venoactive drugs in CVI is to improve venous tone and capillary permeability, although a precise mechanism of action of these drugs is not known.

Wound and Skin Care

Because progressive CVI may lead to compromised skin integrity, it is important to keep the affected area well moisturized to reduce the risk of skin breakdown and possibility of infection. The development of stasis dermatitis needs to be treated with a topical steroid. With venous ulcers, bacterial overgrowth control and aggressive wound care are required to minimize infectious complications. A variety of hydrocolloids and foam dressings are available to control wound fluid drainage and resultant maceration of the adjacent skin. In the presence of an infected ulcer bed, silver-impregnated dressings have been effective in controlling infection and restoring tissue integrity.

SURGICAL MANAGEMENT

Sclerotherapy

Venous sclerotherapy is a treatment modality for obliterating telangiectasia, varicose veins, and venous segments with reflux. Sclerotherapy may be used as a primary treatment or in conjunction with surgical procedures in the correction of CVI. Sclerotherapy is indicated for a variety of conditions including spider veins (<1 mm), venous lakes, varicose veins of 1–4 mm in diameter, bleeding varicosities, and small cavernous hemangiomas (vascular malformation).

Ablative Therapy with Endovenous Radiofrequency and Laser

A recent advance in venous ablative surgery is the use of thermal energy in the form of radiofrequency or laser to obliterate veins. The heat generated causes a local thermal injury to the vein wall leading to thrombosis and eventual fibrosis.

Endovascular Therapy

Endovascular therapy in the treatment of CVI has become increasingly important to restore outflow of the venous system and provide relief of obstruction.

Ligation and Stripping and Venous Phlebectomy

Removing the saphenous vein with high ligation of the saphenofemoral junction is considered durable and the standard for many patients with CVI. In addition, large venous varicose clusters that communicate with the incompetent saphenous vein can be avulsed at the same setting by a technique called stab phlebectomy.

Subfascial Endoscopic Perforator Surgery (SEPS)

However, provides a means to ligate incompetent perforator veins by gaining access from a remote site on the leg that is away from the area with lipodermatosclerosis or ulcers.

Valve Reconstruction

An open technique for repairing the femoral vein valve that renders the valve leaflets competent has been described. Closed techniques for venous repair developed with transcommissural valvuloplasty.

Vein Transplant

Other procedures for reconstructing nonfunctioning venous valves resulting from post-thrombotic valve destruction include transposition of the profunda femoris vein or saphenous vein valve and axillary vein valve transplantation to the popliteal or femoral vein segments. Cryopreserved vein valve allografts also have been used; however, early thrombosis, poor patency and competency, and a high patient morbidity have precluded their use as a primary intervention.

7 Lymphedema

DEFINITION

Lymphedema is defined as excessive accumulation of lymphatic fluid in the interstitial spaces due to impaired function of lymphatic circulation. According to Foldi et al (1985), lymphedema is chronic and progressive swelling caused by a low output failure of the lymphatic system, resulting in the development of a high-protein edema in the tissues.

CLASSIFICATION AND ETIOLOGY

According to the Cause of its Development (Kinmonth et al. 1957)

Primary Lymphedema

Primary lymphedema arises from an abnormality occurring within the lymphatic system. Primary lymphedema includes:

- Idiopathic indicating that the lymphedema is of unknown origin
- Intrinsic indicating that the lymphedema is due to an abnormality within the lymphatic system
- Spontaneous indicating that the lymphedema occurred without intervention.

Primary lymphedema is also associated with some chromosomal and single gene abnormalities, which result in a wide range of lymphatic abnormalities, and differing presentations. These are classified as genetically determined abnormalities or acquired abnormalities and include clinical syndromes such as Turner's syndrome, Noonan's syndrome, Klippel-Trenaunay syndrome and Milroy's disease.

Secondary Lymphedema

Secondary lymphedema arises from the influence of an external factor, which affects the function of the lymphatic system. Causes of secondary lymphedema include infection, inflammation, trauma, cancer and its treatment.

Infection: Filariasis parasite impedes the flow of lymph through the lymphatic vessels they occupy, to cause lymphedema. Repeated bacterial infection involving the superficial layers of the skin and the subcutaneous tissues can cause local signs of inflammation and subsequent damage to the lymphatic channels through fibrosis.

Inflammation: Chronic conditions such as arthritis, psoriasis and dermatitis cause inflammatory changes to occur within the skin lymphatics. These then become obstructed with debris from the inflammatory process, placing the individual at risk of the development of lymphedema.

Trauma: The function of the lymphatic vessels and glands may be impeded by trauma caused by accidental or self-inflicted injury and some surgical procedures. Trauma may occur as a result of burns or severe wounds with extensive tissue loss.

Cancer and its treatment: Surgical excision of lymph node reduction or damage to the lymphatic vessels as a result of the surgery can lead to the development of lymphedema. Radiation therapy damage normal lymph cells within the irradiated area and inflict damage to the lymph channel.

According to the Age of Onset of the Swelling (Kinmonth et al.)

- *Congenital lymphedema*: Present at birth or within 2 years.
- *Lymphedema praecox*: Appearing after birth, but before 35 years of age.
- *Lymphedema tarda*: Appearing after the age of 35 years.

According to the Lymphographic Appearance of Lymph Vessels Established During Lymphography Scanning Procedures, (Kinmonth et al. 1950)

- *Aplasia*: No collecting lymph vessels detected
- *Hypoplasia*: A lower than normal number of lymph vessels detected
- *Hyperplasia*: An increased number of lymphatic vessels detected.

According to Both Clinical and Lymphographic Abnormalities (Browse and Stewart)

- Distal obliteration, which constitutes approximately 80% of primary lymphedemas, described as total absence or reduction in number of distal lymphatics; usually presents as mild edema of both ankles and lower leg.
- Proximal obliteration (10%) usually involves the entire limb and is unilateral.
- Congenital (10%) usually presents at birth or an early stage and can be unilateral or bilateral in presentation.

RISK FACTORS FOR DEVELOPMENT OF LYMPHEDEMA

Risk factors can be considered in three areas, which are summarized in **Table 7.1**.

Table 7.1 Risk factors for development of lymphedema	
Disease-related factors	*Cancer status*: Cancer stage at diagnosis
Treatment-related factors	*Surgery*: Involving lymph nodes in the axilla, groin or pelvis *Radiotherapy*: Involving lymph nodes in the axilla, breast or groin *Postoperative events*: Seroma formation, cording, thrombosis, venepuncture, infection
Patient-related factors	Concurrent medical conditions and medications *Demographic issues*: Age, weight, activities involving the limb *Skin conditions*: Psoriasis, eczema and trauma

Disease Related

Lymphedema can develop as a result of metastatic tumor in lymph node areas causing an obstruction in the lymphatic channels and the appearance of swelling.

Treatment Related

Surgery or radiotherapy to any lymph node area can reduce the number of functioning lymphatic vessels in the area and interrupt the transport capacity of remaining lymphatics. Tender cord-like structures of varying thickness can develop postoperatively and extend from the axilla along the inner aspect of the arm. The presence of cords can result in arm swelling of an acute nature. The thrombosis can lead to increased venous pressure and a subsequent increase in lymph load. Therapeutic access to veins in a limb where there is impaired lymph drainage may cause damage to surrounding tissues and result in increased capillary filtration.

Patient Related

Concurrent conditions such as kidney or cardiac disease, diabetes mellitus or hypertension may cause an increase in lymphatic load and increase the risk of lymphedema. There are several medications known to cause fluid retention in some patients for whom they are prescribed. Arthritis, psoriasis, dermatitis can produce inflammation on superficial lymphatics. Patient who are immobile due to lack of muscular inactivity can develop lymphedema. Weight gain, excessive use of the limb, or exercises, which cause increased capillary filtration, can add to the lymph load of the limb. Air travel may increase the risk of lymphedema following breast cancer treatment due to a reduction in barometric pressure. Trauma sustained by the 'at-risk' limb will initiate an inflammatory response, which adds to the fluid load of the limb.

PATHOPHYSIOLOGY

Increased Permeability of the Capillary Walls

This is due to a release of histamine and other substances, which result from burns, trauma and allergic reactions leading to inflammation and infection.

Decreased Plasma Osmotic Pressure

This problem is found in malnutrition, liver disease, profuse serous drainage and hemorrhage. A low albumin level in the blood may result from decreased albumin synthesis or nutritional protein deficiency, and result in lower limb and ankle swelling.

Increased Plasma Hydrostatic Pressure

This causes fluid to be squeezed into the tissues, due to circulatory overload. This type of edema is commonly seen in conditions such as heart failure (due to salt and water retention), kidney failure (due to salt and water retention) and excessive infusions of isotonic or other sodium, containing solutions. It may also result from venous obstruction, venous disease or immobility where continuous dependency of the legs leads to increased capillary pressure related to increased venous pressure.

Reduced Lymphatic System Transport Capacity

A reduction in the lymphatic system transport capacity can develop when damage or obstruction occurs in the lymphatic system. This reduces the available drainage routes for lymph fluid and lymphedema can appear. It can occur following cancer-related treatment in lymph node areas of the body or when advanced or recurrent tumor obstructs lymph node regions.

CLINICAL FEATURES

Location

When lymphedema develops, it is most often apparent in the distal extremities, particularly over the dorsum of the foot or hand (**Fig. 7.1**). The term dependent edema describes the accumulation of fluids in the peripheral aspects of the limbs, particularly when the distal segments are lower than the heart. In contrast, lymphedema also can manifest more centrally, for example in the axilla, groin or even the trunk.

Severity

The lymphedema is described by the severity of changes that occur in skin and subcutaneous tissues. The three categories, i.e. pitting, brawny and weeping edema.

1. *Pitting edema*: Pressure on the edematous tissues with the fingertips causes an indentation of the skin that persists for several seconds after the pressure is removed. This reflects significant, but short-duration edema with little or no fibrotic changes in skin or subcutaneous tissues.
2. *Brawny edema*: Pressure on the edematous areas feels hard with palpation. This reflects a more severe form of interstitial swelling with progressive, fibrotic changes in subcutaneous tissues.

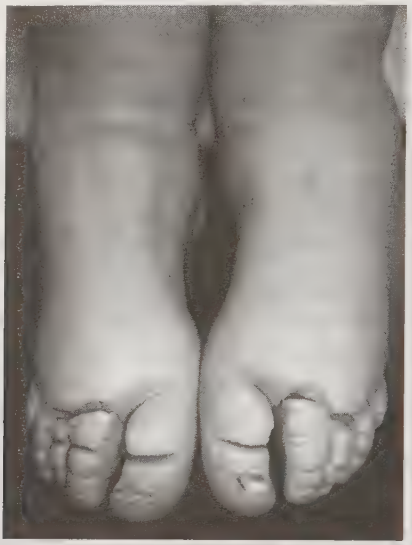


Fig. 7.1 A patient with severe, chronic lymphedema of both lower extremities

3. *Weeping edema*: This represents the most severe and long-duration form of lymphedema. Fluids leak from cuts or sores; wound healing is significantly impaired. Lymphedema of this severity occurs almost exclusively in the lower extremities.

   Although all three types reflect a significant degree of lymphedema.

Increased Size of the Limb

As the volume of interstitial fluid in the limb increases, so does the size of the limb (weight and girth). Increased volume, in turn, causes tautness of the skin and susceptibility to skin breakdown.

Sensory Disturbances

Paresthesia (tingling, itching or numbness) or occasionally a mild aching pain may be felt particularly in the fingers or toes. In many instances, the condition is painless and the patient perceives only a sense of heaviness of the limb. Fine finger coordination also may be impaired as the result of the sensory disturbances.

Stiffness and Limited Range of Motion

Range of motion (ROM) decreases in the fingers, wrist or toes, ankle or even in the more proximal joints, which leading to decreased functional mobility of the involved segments. Decreased resistance to infection wound healing is delayed; and frequent infections (e.g. cellulitis) may occur. These apparent skin infections are recognized by signs and symptoms including acute pain, swelling, anorexia, fevers, vomiting and rigors.

Table 7.2 International Society of Lymphology (ISL) lymphedema staging	
Stage 0	A subclinical state where swelling is not evident, despite impaired lymph transport. This stage may exist for months or years before edema becomes evident
Stage 1	This represents early onset of the condition where there is a accumulation of tissue fluid that subsides with limb elevation. The edema may be pitting at this stage
Early stage 2	Limb elevation alone rarely reduces swelling and pitting is manifest
Late stage 2	There may or may not be pitting as tissue fibrosis is more evident
Stage 3	The tissue is hard (fibrotic) and pitting is absent. Skin changes such as thickening, hyperpigmentation, increased skin folds, fat deposits and warty overgrowths develop

Psychological Effects

The psychosocial impact of living with lymphedema can be profound, resulting in negative feelings such as embarrassment, loss of self-esteem, and increased feelings of anxiety and depression.

Staging of lymphedema is based on International Society of Lymphology (ISL) lymphedema staging (**Table 7.2**).

MEDICAL MANAGEMENT

Diuretics
The administration of diuretics results in an increased excretion of water from the body.

Benzopyrones
These are a group of naturally occurring substances, some of which have been found to be beneficial in the treatment of high-protein edemas.

Diethylcarbamazine
For filariasis.

SURGICAL MANAGEMENT

Indications
- To reduce the weight of the bulky part
- To improve the shape of the limb, making it possible for patients with large limbs to wear normal clothes again
- To reduce the incidence of repeated infections
- To improve the texture of the skin.

Surgical procedures used in lymphedema management can be divided into three areas:
1. Reduction procedures
2. Lymphatic bypass procedures
3. Liposuction.

Reduction Procedures

Several surgical procedures are Homan's reducing operation, Sistrunk's reducing operation, Charles' reducing operation and Thompson's buried dermis flap operation. These procedures remove excess, edematous, subcutaneous tissue. In Homans operation, residual flaps are sutured together. But in Charles operation, the defects are covered with split thickness grafts.

Lymphatic Bypass Procedures

Obstructed lymphatic vessels have been anastomosed to vessels in the venous system in an attempt to drain the swollen limb and normal healthy lymph vessels have been transplanted into areas where lymphedema exists in order to connect poorly functioning lymph vessels with normal ones.

Liposuction

The procedure involves several incisions along the length of the limb through which a cannula is inserted. The subcutaneous fat is sucked out through the cannula under vacuum. Liposuction does not correct lymph drainage and results are only maintained through the continued use of high compression garments.

COMPLICATIONS

- Recurrent cellulitis occurs due to entry of organism through skin defects.
- Lymphangitis, which is inflammation of lymph vessels is common
- Lymphangiosarcoma is the malignancy due to long standing swelling
- *Lymphostatic hemangiopathy*: Due to edema, dermal microcirculation gets affected and surrounding tissues are then infiltrated by number of inflammatory cells. The condition is known as lymphostatic hemangiopathy.

8 Assessment of Patients with Peripheral Vascular Disease

8.1 EXAMINATION AND TREATMENT OF PERIPHERAL VASCULAR DISEASE

EXAMINATION

The examination is the initial component in the management of the patient and includes patient history, review of systems and data collection using specific tests, and measures. A thorough examination of a patient with the diagnosis of peripheral vascular disease (PVD) can differentiate arterial disorders from venous disorders.

Subjective Examination

The subjective examination addresses the current symptoms including presentation, behavior and factors that either increase or decrease symptoms.

Patient History

As with any patient examination, the initial step is to obtain a complete history. If available, prior to the patient interview, requisition of medical records can be useful in understanding the patient's previous medical condition:

- *Age and sex*: Atherosclerosis is a disease of old age. It affects men more than women. Buerger's disease is commonly seen in men between 20 and 40 years of age.
- *Limbs affected*: Buerger's disease commonly affect lower limbs but Raynaud's disease commonly affect upper limbs.
- *Bilateral or unilateral*: In Buerger's disease and Raynaud's disease is bilateral. Atherosclerosis may be unilateral to start with, but often ends as bilateral disease.

- *Pain*: Assess in the OPQRST format as mentioned below; Origin/Onset, Pattern/Position, Quality/Quantity, Radiating/Referred, Symptoms/Signs and Treatment. Further description is beyond the scope of this book. The two types of pain are noticed, i.e. intermittent claudication and rest pain.
  1. *Intermittent claudication*: The word *claudication* is derived from the Latin word *claudico* meaning 'to limp'. The current definition of intermittent claudication (IC) is pain or discomfort of the lower extremity brought on by walking and relieved by rest—hence the intermittent nature. **Table 8.1.1** differentiates intermittent claudication from other disorders. It is often described as pain, cramping, aching, numbness, fatigue or weakness in the muscles of the leg, thigh or buttocks that occurs during exercise and abates in a short time with rest. Pain develops only when the muscles are working. The pain disappears when the exercise stops. The location of the discomfort with exercise may help localize the site of the stenosed or obstructed artery. Symptoms typically occur in the muscle groups immediately distal to the diseased vessel. Obstruction at the level of the superficial femoral artery, the most common site of atherosclerosis in the lower limbs, causes calf muscle symptoms. However, as during walking the gastrocnemius musculature has the greatest workload and

Table 8.1.1 Comparison of intermittent claudication to other conditions with lower extremity pain

Characteristics	Intermittent claudication	Spinal stenosis	Chronic compartmental syndrome	Venous insufficiency
Location of pain or discomfort	Calf muscle	Hip, thigh, buttocks (follows dermatome)	Calf muscles	Entire leg, but usually worse in thigh and groin
Characteristic of discomfort	Cramping pain	Weakness, pain	Tight, bursting pain	Heavy feeling
Onset relative to exercise	After same degree of exercise	After walking or standing for some length of time	After sustained exercise (e.g. jogging)	After walking
Effect of rest	Quickly relieved	Relieved by stopping only if position is changed	Subsides very slowly	Subsides slowly
Effect of body position	None	Relief by lumbar spine flexion	Relief speeded by elevation	Relief speeded by elevation
Distance walked	Constant	Variable	Variable	Variable
Other characteristics	Reproducible	Frequent history of back problems provoked by increased intra-abdominal pressure	Typically muscular athletes edema	History of iliofemoral DVT*, signs of venous congestion

*DVT, deep vein thrombosis

highest oxygen consumption of any muscle group in the leg, calf pain is common even with more proximal disease. Patients with proximal disease, such as aortoiliac occlusive disease, may develop claudication in a buttock, thigh or even lower back. The classic presentation of aortoiliac disease, termed Leriche syndrome, is bilateral 'high' claudication accompanied by impotency and atrophy of the lower extremity muscles. Obstruction in the tibial or peroneal arteries may cause discomfort in the ankle or foot, which in diabetics may be difficult to distinguish from diabetic neuropathy.

2. *Rest pain*: The pain is continuous and aching in nature. The pain is worse at night its gets aggravated by elevation of the leg above the level of the heart and is relieved by hanging the leg below the level of the heart. It usually affects the most distal part first that means the tip of the toes. The painful part becomes very sensitive and any movement or pressure causes an acute exacerbation.

- *Effects of heat and cold:* Application of warmth will increase symptoms of arterial occlusion. Raynaud's phenomenon is always associated with cold.
- *Paresthesia:* When the muscle pain begins, the patient often feels numbness, pins and needle sensation.
- *History of superficial phlebitis:* This is characterized by swelling, redness and minor pain in the affected part.
- *Involvement of other arteries:* If there is complain of fainting, transient black out, chest pain, weakness or paresthesia in the upper limbs, blurred vision and abdominal pain involves occlusion of other arterial disease anywhere in the body.
- *Impotence:* Due to failure in erection in case of bilateral iliac arterial occlusion.
- *Past history:* Interview with the patient should include questions concerning past medical history (e.g. history of diabetes mellitus, hypertension or any cardiac condition). Information should be gathered about medications and any prior treatment for the current problem (s), as well as history of relevant surgical or medical treatment procedures involving the vascular system.
- *Personal history:* It can reveal details of lifestyle patterns such as use of tobacco, dietary habits and use of alcohol. A history of onset of symptoms and mechanism of injury should be included.
- *Family history:* Atherosclerosis and varicose vein are often familial.
- *Occupational history:* Information should also be gathered about the individual's work environment and job skills required that may contribute to the pathology and impairments.

Objective Examination

The objective section of the initial examination should begin with observation of the patient. Any evidence of cellulitis or edema should be noted. Cyanosis or pallor, loss of hair, evidence of wound or a previous amputation need also to be recorded.

Observation

Change in Color

Marked pallor is a remarkable feature of sudden arterial occlusion. Congestion and purple-blue cyanosed appearance are the characteristic features of severe ischemia. Presence of discolorations of the skin such as hemosiderin staining in case of venous disorders should also be documented.

Signs of Ischemia

The signs of ischemia are thinning of skin, diminished growth of hair, loss of subcutaneous fat, shininess, trophic changes in the nail, which become brittle and transverse ridges and minor ulcerations.

Palpation

Skin temperature

Temperature of the skin can be assessed with a variety of such as a radiometer, thermostat; test tubes or basic palpation skills. The temperature is best felt with the back of fingers. Feel the temperature of whole limb to find out the zone where the temperature changes from the normal warm temperature to cold skin of the ischemic site. Altered temperatures of the affected limb versus the uninvolved side or more proximal body part can provide important insight into vascular dysfunction. In cases of arterial insufficiency, skin temperature will be reduced according to severity of disease. With venous insufficiency, there may be little or no change in temperature of the affected limb. The radiometer or thermistor provides quantitative data and can be compared to information gained from palpation of the extremity.

Capillary refilling

The tip of the nail or the pulp of a toe or a finger is pressed for a few seconds and pressure is released the time taken for the blanched area to turn pink after the pressure has been released is a crude indication of capillary blood flow. This time is definitely longer in ischemic limb.

Palpation of blood vessels

The presence or absence of pulses should be addressed. With PVD, palpation of the distal arteries is typically more important than monitoring the larger vessels of the neck and chest. Palpation should focus on the more distal arteries along with the femoral, popliteal, dorsalis pedis and posterior tibial arteries. In case of ischemic limb, there will be disappearance of arterial pulsations below the level of occlusion. Expansile arterial pulsations indicate presence of aneurysm. The arteries, which commonly assessed are dorsalis pedis artery, posterior tibial artery, popliteal artery, femoral artery, radial artery, ulnar artery, brachial artery. Each site is often assigned a subjective rating using grading schemes. One such scheme is to grade the pulses as 0 if absent, 1+ if diminished and 2+ if normal.

Motor sensory status

A gross motor examination to assess range of motion (ROM) and strength should be included in the initial examination. These tests can provide information about the effectiveness of muscular activity in promoting blood flow as well as general mobility of the patient. Sensory testing using light touch and pressure should be included as well as the use of the Semmes-Weinstein monofilaments. These monofilaments can detect different cutaneous sensory levels. The instrument consists of a handle with nylon projections of various thicknesses that require different forces to bend the material. The 5.07 monofilament indicates protective sensation if felt by the patient.

Palpation of Edema

The classic clinical assessment described by Seidel et al. is an unvalidated, subjective measure of edema. The clinical assessment evaluated in this study, an adaptation of this classic technique, was repeated at three anatomical locations (the lower calf at 7 cm proximal to the midpoint of the medial malleolus, behind the medial malleolus and the dorsum of the foot). The scoring definitions were modified to capture pit depth and recovery separately. Pit depth was estimated visually and scored as follows:

0 = No clinical edema
1 = Slight pitting (2 mm depth) with no visible distortion
2 = Somewhat deeper pit (4 mm) with no readily detectable distortion
3 = Noticeably deep pit (6 mm) with the dependent extremity full and swollen
4 = Very deep pit (8 mm) with the dependent extremity grossly distorted. Recovery time was recorded in seconds.

Examination

Circumferential (Girth) Measurements

Circumferential measurements of an extremity are essential to identifying and monitoring the extent of, and documenting changes in, edema. Girth measurements should be taken in reference to bony prominences to allow consistency for tracking changes during subsequent measurements. For consistent ankle circumference measurements, each ankle was marked with a semipermanent marker at approximately 7 cm proximal to the midpoint of the medial malleolus described by Mora et al.

Figure of Eight Method

Eight methods developed by Perry S Esterson, which use eight landmarks on the ankle and foot to measure ankle circumference in centimeters **(Figs 8.1.1A to E)**:

1. Midway between the tibialis anterior tendon and the lateral malleolus
2. Distal to the tuberosity of the navicular
3. Proximal to the base of the fifth metatarsal
4. Tibialis anterior tendon
5. Distal to the distal tip of the medial malleolus
6. Achilles tendon

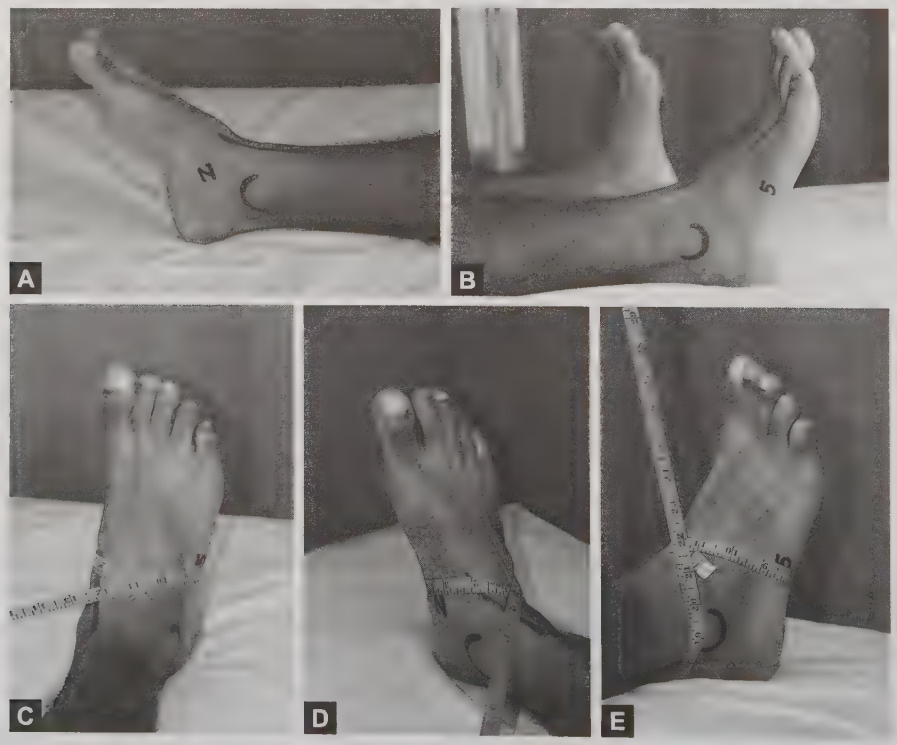
7. Distal to the distal tip of the lateral malleolus, and
8. Back to origin.

A tension-controlled measuring tape, rather than a standard measuring tape, was wrapped around the ankle/foot following the eight landmarks.

The beginning of the tape is placed midway between the tibialis anterior tendon and lateral malleolus then the tape is drawn medially across the instep and placed just distal to the tuberosity of the navicular bone. Pull the tape across the arch and up just proximal to the base of the fifth metatarsal and cross the tibialis anterior tendon. Continue the tape around the ankle joint just distal to the distal tip of the medial malleolus and pull the tape across the Achilles tendon; place the tape just distal to the distal tip of the lateral malleolus and back to starting point.

Volumetric Measurement

It is a quantitative system to measure edema, can be used. This process can be time consuming and cumbersome, but is a very accurate method of determine method of determining changes in edema. The volumeter, a clear acrylic rectangular box (13" × 5" × 9") with a spout at the top of one of the short sides was filled with water until water rushed out of the spout. Once the water level was stable, the patient placed one foot in the volumeter and the displaced water



Figs 8.1.1A to E Assessment of edema
(For color version, see Plate 2)

collected and measured in a graduated cylinder. The amount of water displaced in milliliters equals the volume of the foot/ankle.

Auscultation

Auscultation is performed by listening to the vessel with a stethoscope. This assessment is useful in identifying a bruit. Bruit is created by turbulence as blood flows through a stenotic lesion. Detection of a bruit may indicate the possibility of partial blockage of that artery. A systolic bruit is common when there is obstructive disease proximal to the site auscultated. Systolic murmur can also be heard over an aneurysm. When a diastolic component is also present, it means the collateral circulation is inadequate to even allow diastolic pressure distal to the diseased area to rise to the systemic diastolic pressure and thus stop flow during diastole. Bruits may be heard in the abdominal, femoral, carotid, subclavian and even popliteal locations.

Blood Pressure

Blood pressures of both arms are measured to exclude affection of subclavian, brachiocephalic or axillary artery.

Gait Assessment

Characteristics of the gait pattern should be observed and can provide clues into areas that need to be addressed during treatment.

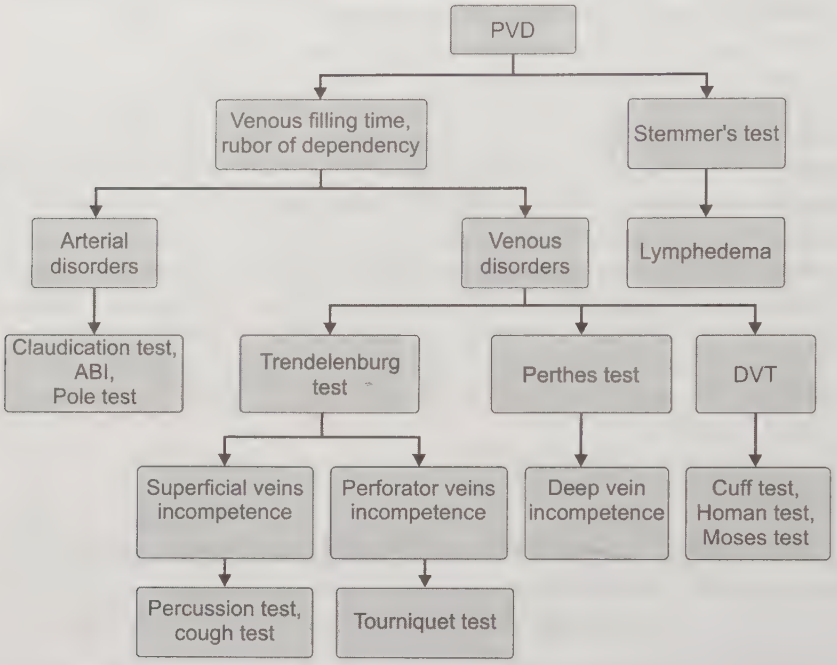
8.2 SPECIAL TESTS IN PERIPHERAL VASCULAR DISORDERS

Two tests that differentiate between arterial and venous disorders are venous filling time and rubor of dependency. **Flow chart 8.2.1** has to be followed to differentiate between different types of peripheral vascular disorders.

VENOUS FILLING TIME

In this test the patient is positioned supine and the affected extremity is elevated to empty blood from superficial veins of the limb. Next, the patient hangs the extremity over the edge of the treatment table into a dependent position. Time is recorded when the veins on top of the foot have refilled. Normal filling time is approximately 15 seconds. A time greater than 15 seconds is indicative of potential arterial disease whereas a time below 15 seconds would suggest venous insufficiency.

Flow chart 8.2.1 Special tests for PVD



Abbreviations: DVT, deep vein thrombosis; PVD, peripheral vascular disease; ABI, ankle-brachial index

RUBOR OF DEPENDENCY

The rubor of dependency test can provide information concerning the state of both the arterial and venous systems. The test is performed in similar fashion to the venous filling test. The patient is positioned supine and the color of both feet is examined. The affected limb is then elevated for several seconds, and lowered back to the original position. The time is recorded for color of the tested foot to match the stationary foot. If arterial disease is present, it may take longer than 20–30 seconds for color to return and will usually be bright red. If the color returns immediately, this may denote the presence of venous insufficiency.

CLAUDICATION TEST

The claudication test provides information about arterial involvement. One of the earliest signs of arterial disease is intermittent claudication. As discussed, claudication is a painful cramping most often occurring in the lower leg muscle owing to an insufficient supply of blood to working muscles. The patient begins to ambulate using a treadmill or an unobstructed level surface. The claudication test is recorded as the time or distance at which this painful symptom occurs.

CUFF TEST

Whenever a patient is referred to physical therapy with a vascular disease, it is important to assess for the presence of a DVT. Usually a through work up by a physician should detect the potential for development of a DVT, but occasionally it can be overlooked as wound or musculoskeletal pain. This test is performed by placing a blood pressure cuff around the lower leg and inflating cuff. If the patient is unable to tolerate cuff pressure greater than 40 mm Hg, there is a high probability of an active DVT.

HOMAN'S TEST

The test can be augmented with a forceful squeeze of the calf region or passive dorsiflexion of the calf by the examiner. A positive result is severe pain expressed by the patient is indicative of DVT.

MOSES TEST

Tap the calf muscle side to side elicits pain.

PERCUSSION TEST

Percussion of a major superficial vein of the lower extremity can be useful in determining valve competency. With the lower extremity in a dependent position, the greater saphenous vein is palpated distal to the knee with one hand while the vein is tapped approximately 6–8 inches proximal to the knee with the other if a wave of fluid is detected under the distal palpation site; this indicates an incompetent valve or valves.

COUGH TEST

Patient coughs, palpate for reflux as above. There should be no reflux of blood.

TRENDELENBURG TEST

The Trendelenburg test is a simple way to assess if the valves are functioning properly, especially within the perforation system. The patient is positioned supine and the lower extremity is elevated to approximately 75° to allow venous blood to empty. A tourniquet is placed around the thigh to prevent superficial venous backflow. The lower extremity is then placed in a dependent position. The process of venous filling is observed. Should the superficial veins fill quickly the valves of the perforator vein have become incompetent. If immediate filling is noted with release of the tourniquet, the superficial system has incompetent valves. Normal venous filling would demonstrate both situation, and the veins would demonstrate neither situation, and the veins would fill approximately 25–30 seconds after the lower extremity is placed in a dependent position. The test can be repeated with the tourniquet at different levels (above knee and below knee) to further pinpoint the level of valvular incompetence. This is called as torniquet test.

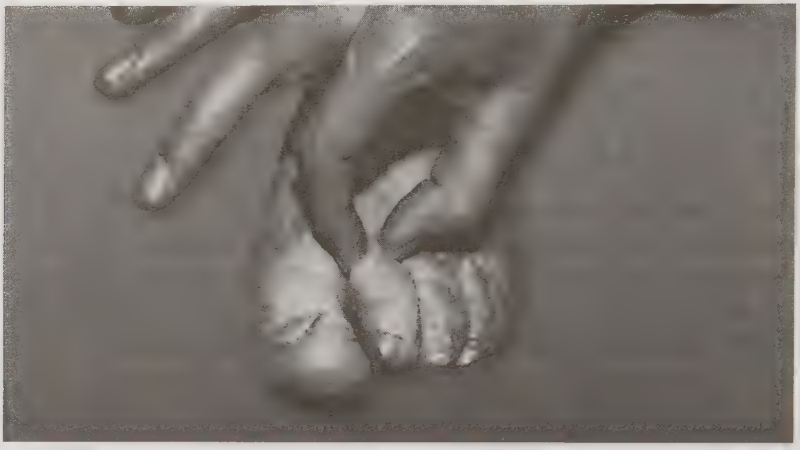


Fig. 8.2.1 Stemmer's sign

STEMMER'S TEST

This test can be performed to clarity the pressure or evidence of lymphedema or lymphostasis. Stemmer's sign is the inability to pick up a fold of skin at the base of the second toe of the affected extremity **(Fig. 8.2.1)**.

PERTHES TEST

The test used to assess vein patency, calf muscle pump and incompetence. Patient stands, a tourniquet is placed on thigh and they walk for 5 minutes. If deep veins patent and perforators competent, then saphenous veins collapse below the tourniquet (calf muscle pump intact). A positive Perthes test means the superficial veins become further dilated, tortous and the patient complaints of severe bursting pain in the leg. This suggests an occluded deep veins due to previous DVT.

8.3 INVESTIGATIONS

ARTERIAL STUDIES

Doppler Ultrasound (Figs 8.3.1 and 8.3.2)

Locate dorsalis pedis artery or post-tibial artery pulse. Pressure cuff is placed around calf and inflated until cessation of audible sound. Take measurement at this point. Same procedure is repeated at upper extremity cuff over arm (brachial artery). Doppler is placed at radial artery then ankle brachial index (ABI) is calculated, i.e. ankle pressure/arm pressure.

Fig. 8.3.1 Ankle brachial index measurement for lower limb
(For color version, see Plate 3)

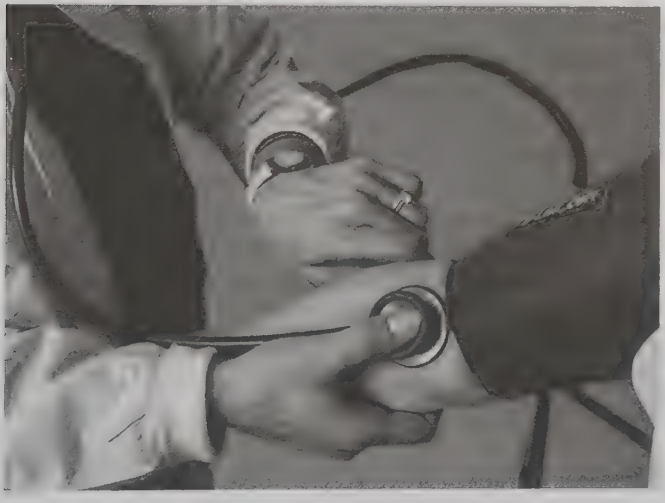


Fig. 8.3.2 Ankle brachial index measurement for upper limb

Resting Ankle Brachial Index

Normal ratio of the ankle to the brachial systolic blood pressure ankle brachial index (ABI) is greater than 0.95 for screening on atherosclerosis in the legs (detection of symptomatic PAD or confirmation of symptomatic PAD). Pressures in each leg were measured and the ABIs calculated separately for each leg. The total ABI is the lowest of both legs. Normal values are mentioned in **Table 8.3.1**.

Table 8.3.1 Ankle brachial index	
Normal	Normal = one or slightly higher
>1.2	Falsely elevated-arteriosclerosis, DM
1.2–0.95	Normal
0.95–0.75	Mild arterial + claudication
0.74–0.5	Moderate arterial + rest pain
<0.50	Severe arterial

Pole Test

This test was performed for falsely elevated ABI. In patients with leg ischemia level of elevation at which foot takes significant pallor correlates to level at which Doppler test was performed. Height of ankle elevation calibrated to mm Hg, i.e. 13 cm intervals to 10 mm Hg. Upper extremity measurement is determined as mentioned above. If pole is not available, distance between ankle and bed at which sound disappear is taken and multiply 735 to get pressure.

Postexercise Ankle Brachial Index

In patients with atherosclerotic lesions which are not yet hemodynamically significant at rest, postexercise hyperemia with changes in blood flow and other parameters could reveal PAD. Thus, in symptomatic patients with normal resting ABI, it is recommended to measure the post exercise ABI to differentiate arterial claudication from nonarterial claudication (pseudoclaudication). A 15–20% drop in postexercise ABI compared to resting ABI is indicative for the PAD diagnosis. Patients were requested to undergo standard exercise testing in accordance with the physician advice. According to the protocol, the patient was requested to perform 30 active pedal plantar flexions (or to stop earlier if symptoms occurred). Immediately after exercise the patient was asked to lie in a supine position. Then only the blood pressure of the arm and the blood pressure of the tibial posterior arteries were measured. Hence, the total ABI is the ratio of the lower of the right and left tibial posterior artery pressures and the brachial artery pressure.

Toe Systolic Pressure Index

This index is a useful clinical method, especially in diabetes because digital vessels are usually spared form medial sclerosis. It is especially useful in detecting individual at high risk of gangrene, ulceration and infection associated with occlusive arterial disease (arterial BP at toes is approximately 60% systolic BP at ankle).

Air Plethysmography

Air plethysmography (APG) is a noninvasive vascular examination of both the arterial and venous systems. This clinical tool has the capability to detect minute

changes in leg volume and can be performed during static or postural changes as well as during light exercise. It is also a reliable predictor of the presence or reoccurrence of venous ulcers.

Arteriography

It is a common contrast dye studies that may be ordered by a physician to further examine a patient's vascular system.

X-ray

It can show calcified arteries that are associated with low ABI levels.

Pulse Volume Recording

This is based on the principle that the contour of the pulse wave changes distal to a stenosis. There is loss of dicrotic notch, a slow rise, a rounded peak and a slow descent. Plethysmographic instruments are used to measure volumetric changes in the limb when the transducer is placed at different segments of the limbs.

Doppler Velocimetry

The Doppler wave form changes if the probe is placed distal to an arterial stenosis. Normal Doppler wave form has forward flow during systole; transient flow reversal during early diastole and slow forward flow during shows a late diastole. Abnormal Doppler wave forms—are deceleration of systolic flow, loss of early diastolic reversal and diminished peak velocities.

Duplex Imaging

Methods include gray scale imaging, Doppler pulse and continuous wave spectral imaging and Doppler color flow imaging. Gray scale and color flow imaging are useful in localizing the diseased segment while spectral imaging is used to assess the severity of the lesion. A greater increase in peak systolic velocity at the site of stenosis indicates 50% or more stenosis. Doppler signals are absent if artery is totally occluded.

Digital Contrast Angiography

It is the gold standard for identifying PVD. It is indicated to visualize the arterial anatomy prior to a revascularization. The ability to store information in standardized Digital Imaging and Communication in Medicine (DICOM) format, allows remote reading and serial comparison, use of telemedicine applications and universal access in healthcare.

MR Angiography

It is a noninvasive test to visualize the peripheral arteries. Gadolinium enhanced MRA has shown to have sensitivity and a specificity of 96–100%.

It is done to evaluate symptomatic patients and helps in decision making prior to endovascular intervention. It is also of special use in evaluating arterial dissection and hematoma.

Intravascular Ultrasound

Widely used as an adjunct to peripheral vascular intervention. It avoids drawbacks of transcutaneous duplex, e.g. shadowing artifacts, wall calcification, and acoustic impedance from interposed tissue.

VENOUS STUDIES

Noninvasive Testing

Venous Duplex Imaging

Venous duplex imaging is a method to diagnosis DVT and CVI. Venous duplex imaging combines imaging of the deep and superficial veins with pulsed Doppler assessment of flow. This provides information about the anatomic extent of disease involving the deep and superficial systems, as well as perforators. The direction of flow may be assessed in a 30° reverse Trendelenburg position during provocative maneuvers, such as the Valsalva maneuver, or after augmenting flow with limb compression. The use of a cuff inflation-deflation method with rapid cuff deflation in the standing position is preferred to induce reflux. The presence of reflux is determined by the direction of flow because any significant flow toward the feet is suggestive of reflux.

The duration of reflux is known as the reflux time (replacing the commonly used valve closure time). The longer the duration of reflux or the greater the reflux time implies more severe disease. Other parameters such as the reflux velocity and even the calculated reflux volume have been used to assess the severity of reflux.

Photoplethysmography

Photoplethysmography (PPG) may be used to establish a diagnosis of CVI. Relative changes in blood volume in the dermis of the limb can be determined by measuring the back scatter of light emitted from a diode with a photo sensor. A PPG probe is placed on the foot with maneuvers to empty the foot with calf muscle contraction. Then return of blood is detected by increased backscatter of light and the refill time may be calculated. The venous refill time is the time required for the PPG tracing to return to 90% of the baseline after cessation of calf contraction. A venous refill time <18 to 20 seconds, is indicative of CVI. A venous refill time >20 seconds suggests normal venous filling. The use of a tourniquet or low-pressure cuff allows for distinguishing superficial from deep venous disease. Correction of an abnormal refill time with a low-pressure thigh cuff is indicative of great saphenous vein disease. Failure to correct rapid venous refill time with a low-pressure cuff is indicative of deep venous disease. The test provides information about regional venous function, not about specific anatomic distribution. Although a shorter rapid refill time suggests more severe disease.

Air Plethysmography

Air plethysmography (APG) has the ability to measure each potential component of the pathophysiological mechanisms of CVI—reflux, obstruction, and muscle pump dysfunction. Changes in limb volume are measured by air displacement in a cuff surrounding the calf during maneuvers to empty and fill the venous system. Venous outflow is assessed during rapid cuff deflation on an elevated limb that has a proximal venous occlusion cuff applied. The outflow fraction at 1 second (or venous outflow at 1 second expressed as a percentage of the total venous volume) is the primary parameter used to evaluate the adequacy of outflow. The limb is then placed in the dependent position to evaluate the venous filling. The rate of refill is used to determine the presence and severity of reflux. The key parameter is the venous filling index, which is calculated by measuring 90% of the venous volume and dividing this by the time required to fill 90% of the venous volume after resuming an upright position. A normal venous filling index is < 2 mL/s, whereas higher levels (>4 to 7 mL/s) have been found to correlate with the severity of CVI. The ability of the calf muscle pump to eject blood is determined after single and 10 repetitive contractions during toe raises. The volume of blood ejected with 1 tiptoe maneuver divided by the venous volume is the so-called ejection fraction. This technique provides quantitative information about several aspects of global venous function.

Light Reflection Rheography

Light reflection rheography (LRR) has been developed as a simple, quick and noninvasive test of venous function which reproduces the hemodynamic parameters of venous pressure measurements by recording changes in dermal blood content of the lower limb during exercise. It uses the same principles as photoplethysmography (PPG) but is simpler to use in a clinical context.

Other Techniques

Other techniques such as strain gauge plethysmography and foot volumetry also may be used. Both techniques provide physiological information about global venous function and may correlate better with the clinical severity than doe's duplex imaging. Strain gauge plethysmography is the standard mode for testing endothelial dysfunction and reactive hyperemia. It detects venous outflow to detect deep venous thrombosis in the legs.

Invasive Testing

Phlebography

Phlebography or venography may be either ascending or descending. Ascending phlebography involves the injection of contrast in the dorsum of the foot with visualization of contrast traveling up the lower extremity in the deep venous system. Although ascending phlebography is considered the gold standard to determine the patency of veins, it has been largely replaced by

noninvasive imaging. It does provide details of venous anatomy that may be useful with surgical interventions and can help to distinguish primary from secondary disease. Descending phlebography involves proximal injection of contrast in a semivertical posture on a tilt table with the use of the Valsalva maneuver. It is most useful to identify reflux in the common femoral vein and at the saphenofemoral junction, but it may be used to evaluate other locations. A grading scheme has been developed based on the anatomic extent of reflux. This modality has been largely replaced by duplex scanning. It is now performed if deep venous reconstruction is being contemplated or with an inconclusive duplex scan before other venous surgery.

Ambulatory Venous Pressure

Ambulatory venous pressure (AVP) monitoring is the hemodynamic gold standard in assessing CVI. The technique involves insertion of a needle into the pedal vein with connection to a pressure transducer. The pressure is determined at rest and after exercise is performed, usually in the form of toe raises. The pressure also is monitored before and after the placement of an ankle cuff to help distinguish deep from superficial reflux. AVP has been shown to be valuable in assessing the severity and clinical outcomes in CVI. The mean ambulatory venous pressure (normal range of 20–30 mm Hg) and refill time (normal range of 18–20 seconds) are the most useful measurements. AVP provides information on the global competence of the venous system; however, there is concern about the failure of pressure to accurately reflect the pressure within the deep system. Because of the invasive nature and alternative diagnostic modalities, AVP seldom is used in clinical practice. Attempts to determine AVP by noninvasive means have been evaluated in small studies and are not widely used.

ASSESSMENT OF EDEMA

Clinical Assessment

The classic clinical assessment described by Seidel et al. and further details are described in Chapter 8.1.

Patient Questionnaire

Patient-reported edema was collected using a standardized, unvalidated questionnaire. The questionnaire was examiner-administered and included five questions to assess the presence and severity of self-reported edema over the past week and one question to assess the ease of completing the five questions. Only those patients responding they had experienced edema in the last week were asked about edema severity and frequency.

Ankle Circumference

Ankle circumference was measured in centimeters at a single location as described by Mora et al. For consistent ankle circumference measurements, each ankle was

marked with a semipermanent marker at approximately 7 cm proximal to the midpoint of the medial malleolus. Unlike the method outlined by Mora et al. a tension-controlled measuring tape (Gulick II, Lafayette Instrument Company, Lafayette, IN), rather than a standard measuring tape, can be used to minimize measurement error due to differences in the amount of tension applied.

Figure-of-Eight Method

Further details are described in Chapter 8.1.

The Leg-O-Meter

The Leg-O-Meter is designed to measure the circumference of the ankle or calf. This has high interobserver reliability and is easy to use. Furthermore, it is not invasive or costly.

The Leg-O-Meter, an instrument designed to measure the ankle or calf circumference. The Leg-O-Meter consists of a tape measure fixed to a stand attached to a small board on which the patient is in standing position. The tape measure of the Leg-O-Meter was fixed at 10 cm from the board in order to standardize all measurements. Leg-O-Meter gives a standardized and reliable measure of the circumference of the ankle.

Edema Tester (Fig. 8.3.3)

Cesarone and coworkers developed the edema tester. This involves applying a plastic plate with either protrusions or holes over the swollen area, applying pressure, and measuring the marks made. It may allow the differentiation between primary and secondary lymphedema, although it is only recommended at present as a screening tool. The edema tester is a 5 cm × 10 cm flexible plastic plate with two parallel rows of seven holes ranging from 2 mm to 12 mm in diameter arranged by size, with one row of holes increasing in size and the other

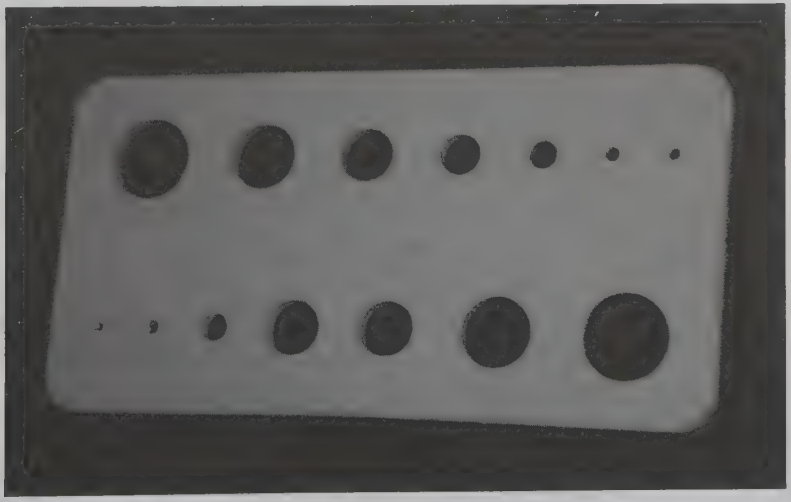


Fig. 8.3.3 Edema tester

line decreasing in size (ACI Medical, San Marcos CA, USA). The edema tester was placed with the long axis in the vertical plane and placed superior to the medial malleolus. A standard blood pressure cuff was placed over the tester and inflated to a standard pressure. No standard guidelines exist for the optimal amount or duration of pressure applied. In the original article, a pressure of 50 mm Hg was held for 2 minutes. Several thresholds of pressure and duration were evaluated for use in this study. Applied pressures of 100 mm Hg and 150 mm Hg, held for 3 seconds, were also used in literature. The cuff and edema tester were then removed. The numbers of impressions left by the holes were counted and the time for the last impression to disappear was measured in seconds. A large number of impressions and a long recovery time indicate more severe edema. A new version is called as Modified Edema Tester is similar to the edema tester except the holes were replaced by bumps in an attempt to mimic the standard clinical assessment (ACI Medical). Several modified edema testers were developed (**Fig. 8.3.4**).

Indirect Measure of Leg Volume

Leg volume was calculated indirectly using the disk model. A series of ankle and leg circumferences was used to calculate the volume of each cross-section in millimeters. The sum of the disk volumes provides an estimate of total leg volume. For consistent measurements, each extremity was marked with a semipermanent marker at 3 cm intervals beginning 3 cm proximal to the midpoint of the medial malleolus to the tibial medial condyle. A tension-controlled measuring tape, instead of a standard measuring tape, was used to minimize measurement error.

Water Displacement (Fig. 8.3.5)

Foot/ankle volume was measured through water displacement using a commercially available foot volumeter (Baseline, Fabrication Enterprises Inc.,

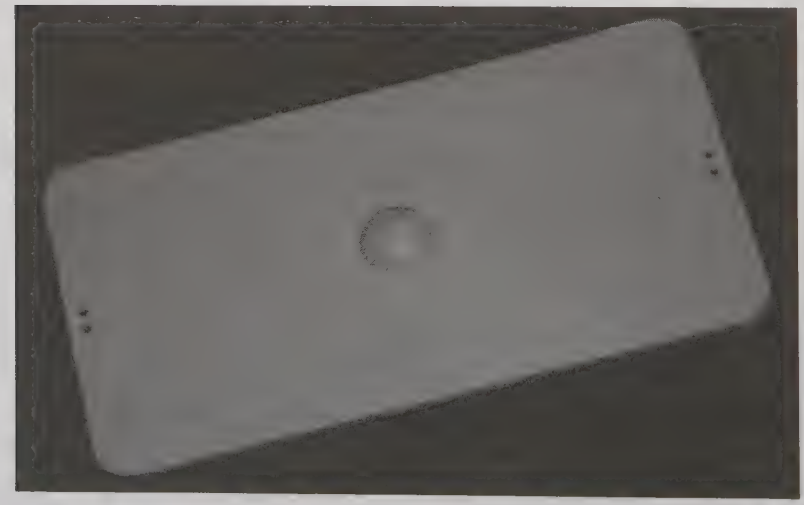


Fig. 8.3.4 Modified edema tester

Fig. 8.3.5 Volume displacement
(For color version, see Plate 3)

White Plains, NY). The volumeter, a clear acrylic rectangular box (13" × 5" × 9") with a spout at the top of one of the short sides was filled with water until water rushed out of the spout. Once the water level was stable, the patient placed one foot in the volumeter and the displaced water collected and measured in a graduated cylinder. The amount of water displaced in milliliters equals the volume of the foot/ankle.

Optoelectronic Volumetry

This is the most sophisticated method for assessing edema. The leg passes through a four-sided rigid frame which can be moved along a rail in the long axis of the limb. The frame is equipped with infrared-detecting diodes emitting an infrared beam which allows precise measurement of the lower limb volume. Markers placed on the leg allow the identification of the upper and the lower reference point.

INVESTIGATIONS IN LYMPHEDEMA

Lymphangiogram

Before lymphoscintigraphy became the gold standard, this was the main technique used for visualizing the lymphatics. It involves direct cannulation of the lymphatics through a skin incision and may lead to infection, local inflammation, and fibrosis. It is technically demanding, painful, and time-consuming, with an increased risk of hypersensitivity reactions and emboli. As a diagnostic tool, the technique has largely been abandoned. However, it is still useful if operative intervention (i.e. bypass procedure) is to be undertaken.

Lymphoscintigram

This is now the gold standard for assessing the lymphatics. The radio labeled protein used is usually technetium Tc 99m–labeled colloid, including antimony sulfur and albumin. It allows measurement of lymphatic function, lymph movement, lymph drainage, and response to treatment. To aid in the measurement of lymph flow, the patient should take an oral dose of heptaminol adenosine phosphate to increase lymphatic flow. A lymphoscintigram may be sufficient if any bypass procedure is intended but some patients may also require a contrast Lymphangiogram to fully elucidate the lymphatic anatomy. The amount of time that the lymphatics are visualized is equally important. If lymphatics are not imaged within the first hour after isotope injection, the diagnosis may be missed. In some patients, the 1-hour image may show normal lymphatics, while only delayed films (2–24 hours post injection) may show the true abnormality.

A lymphoscintigram will also differentiate between lymphedema and edema of venous origin. In patients with venous leg ulcers, lymphscintigraphy reveals significantly reduced lymph drainage in both the affected and the nonulcerated leg compared with controls. It is also lower in patients with varicose veins, especially if deep vein incompetence is present. This suggests that chronic venous insufficiency is also associated with lymphatic insufficiency. In post-thrombotic disease, there is reduction in the subfascial lymphatic flow whereas the epifascial flow remains normal. In lymphedema, both epifascial and subfascial lymphatics are abnormal. Therefore, both epifascial and subfascial compartments must be evaluated to differentiate between post-thrombotic disease and lymphedema. Lymphoscintigraphy also shows impairment of lymphatic drainage or lymphatic disruption following arterial reconstruction.

Fluorescence Microlymphangiography

It involves visualization of the superficial network of lymphatics with a fluorescence microscope following intracutaneous injection fluorescein isothiocyanate dextran. It can confirm clinical diagnosis of lymphedema and can distinguish various forms of edema.

Ultrasound

The ultrasound features of lymphedema are volumetric changes (a minimal increase in the thickness of the dermis, an increase in the subcutaneous layer, and an increase, decrease, or no change in the muscle mass) and structural changes (hyperechogenic dermis and hyperechogenic subcutaneous layer). It allows an assessment of soft tissue changes but does not give information about the truncal anatomy of the lymphatics.

Duplex Ultrasound

In patients with lymphedema, there is gradual impedance of venous return, which then aggravates the edema. The duplex ultrasound may be a useful investigation in patients with lower limb swelling. In one series, a combination of a duplex scan

and lymphoscintigram was able to diagnose the cause of the unexplained limb edema in 82% of patients.

Computed Tomography

Computed tomography (CT) scanning can be used to confirm the diagnosis and monitor the effect of treatment. The common CT findings in lymphedema include calf skin thickening, thickening of the subcutaneous compartment, increased fat density, and thickened perimuscular aponeurosis. A typical honeycomb appearance is seen in most patients. In patients with chronic venous disease, there is enlargement of the subcutaneous compartment and skin thickening but no honeycomb appearance. Computed tomographic scans of patients with DVT show an increase in the subcutaneous layer, with signs of lymphedema, as well as an increase in the cross-sectional muscle area and enlarged superficial veins. However, if calf swelling is not present following DVT, there will be no change in the muscle and so CT becomes an unreliable investigation.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) can differentiate among lymphedema, lipedema, and phlebedema. Features of lymphedema on MRI include circumferential edema, increased volume of subcutaneous tissue, and a honeycomb pattern above the fascia between the muscle and subcutis, with marked thickening of the dermis. It is, however, generally difficult to differentiate primary from secondary lymphedema using MRI. Following reconstructive surgery, MRI shows the edema to be located around the entire circumference of the limb but restricted to the subcutaneous tissue, increasing the leg volume by a mean of 26%. In DVT, there is edema of the leg muscles, particularly in the posterior compartments, with an increase in the leg volume of 23%. In chronic lymphedema, there is an increase in leg volume of 40%. Magnetic resonance imaging in lipedema will confirm that peripheral lymphatics are normal, soft tissue swelling consists solely of fat, and subcutaneous edema is absent.

8.4 PHYSICAL THERAPY FINDINGS IN VASCULAR DISORDERS

PHYSICAL THERAPY FINDINGS OF ARTERIAL INSUFFICIENCY

History

The patient's history may be significant for diabetes mellitus and hypertension. Previous surgical history may include bypass graft or amputation of lower extremity. The patient will often have a significant smoking habit.

Subjective Examination

The patient may present with complaints of pain on ambulation, rest pain and pain in lower extremities with elevation. The patient may report coldness of feet and hand, and may describe color change of digits.

Objective Examination

The examiner may observe pale appearance of extremity and Temperature of distal extremities will be decreased. Edema may or may not present in the affected extremity. The vascular examination will yield absent or diminished pulses; possibly the presence of bruit and the ankle brachial index (ABI) will be decreased or falsely elevated. If the ABI is falsely elevated the pole test results will presumably be decreased. The special tests such as rubor of dependency and venous filling time will have extended time frames. The Trendelenburg test, percussion test and cuff test should all be negative.

PHYSICAL THERAPY FINDINGS OF VENOUS INSUFFICIENCY

History

During the history taking, the patient may report the presence of diabetes, hypertension and or congestive heart failure. The occurrence of a previous leg ulcer or DVT may be reported as well as prevalence of varicosities.

Subjective Examination

The patient rarely reports evidence of intermittent claudication or rest pain. Edema if present can be decreased with elevation with no painful side effects. The patient may report the skin on the affected lower extremity as darker than normal.

Objective Examination

Hemosiderin staining of the lower extremities may be present. All involved extremities will be warm to touch and may be significant for edema. Distal pulses should be strong. Reflux may be detected with the Doppler and also reveal a normal ABI. Rubor of dependency and venous filling time results will indicate immediate filling of the veins. There may be positive percussion and Trendelenburg tests. The cuff test is usually negative except in the presence of active, deep vein thrombosis (DVT). If an air plethysmography (APG) is performed it will reveal an elevated venous filling index, decreased ejection fraction and increased residual volume in the affected extremity.

Differential Diagnosis of Lymphedema

Subjectively the patient typically reports similar information found with venous disorders. There may be history of malignant disorders reported in the patient history. One major distinction is that differentiates venous disease from lymph edema is the rate at which edema resolves. With venous insufficiency,

edema resolves within several hours of elevation, but may require several days to resolve in a patient with lymphedema.

Objectively, inspection of the extremities and trunk for generalized aberrations from normal skin texture and color can reveal deepened natural skin creases, thickened cutaneous folds, pronounced soft local swelling, as well as fibrosis of the skin. There is usually no evidence of skin ulceration, which is a common finding with venous insufficiency. A positive Stemmers' sign and columnar deformation of the legs can also be seen. Another way to differentiate between lymphedema and venous insufficiency is to use Doppler ultrasound to rule out venous reflux. Palpation can be used to identify local or generalized swelling, including subcutaneous lymph node enlargement.

8.5 OUTCOME MEASUREMENTS IN PERIPHERAL VASCULAR DISEASE

MEASUREMENT OF SEVERITY IN ARTERIAL DISORDERS

Presence of Claudication

The Edinburgh Claudication Questionnaire (Leng GC, Fowkes FRG, 1992) consists of series of six items. A positive questionnaire diagnosis of claudication is made only if the 'correct' answer is given to all questions.

MEASUREMENT OF SEVERITY IN VENOUS DISORDERS

Venous Severity Score

The venous severity scoring provides a numeric score based on three components—the venous clinical severity score, the anatomic segment disease score, and the disability score. The venous clinical severity score consists of 10 attributes (pain, varicose veins, venous edema, skin pigmentation, inflammation, induration, number of ulcers, duration of ulcers, size of ulcers and compressive therapy) with four grades (absent, mild, moderate, severe). The venous anatomic segmental score assigns a numerical value to segments of the venous system in the lower extremity that account for both reflux and obstruction. The venous disability score comes from the ability to perform normal activities of daily living with or without compressive stockings. The venous severity scoring has been shown to be useful to evaluate the response to treatment.

Deep Vein Thrombosis Risk Assessment Tool

The prevention of deep vein thrombosis (DVT) is important and can be achieved by comprehensive DVT risk assessment undertaken on admission,

followed by the most appropriate form of prophylaxis. Two DVT patient-risk assessment tool are thrombosis risk factor (TRF) assessment tool and the Autar DVT risk assessment scale. This tool categorizes the patient into low, moderate and high risk. The prophylactic measures can be administered based on the severity of risks.

ASSESSMENT OF PHYSICAL PERFORMANCE IN ARTERIAL DISORDERS

Treadmill Testing

Treadmill testing is a common way to quantify the grade of functional impairment. Treadmill testing is an objective measure of walking performance and thus is the measurement most frequently used in both research and clinical settings to measure change after a therapeutic intervention.

The treadmill protocols advocated for use in the peripheral arterial disease (PAD) population are different than those used to assess exercise performance in coronary artery disease (CAD). The typical exercise test used in CAD rapidly increase both speed and grade to symptom limited maximum exercise capacity.

Patients with PAD are often limited in walking speed. The treadmill protocols used in PAD are either graded (where the speed is fixed at 2 or 3 mph and the grade increases with each stage) or constant load, where both speed and grade are fixed.

Measurements

- *Initial claudication distance (ICD)* is the distance walked at the onset of claudication pain, also known as the initial claudication distance or pain free walking distance.
- *Absolute claudication distance (ACD)* is the distance at which claudication pain becomes so severe that the patient is forced to stop, also known as the maximal walking distance.
- *Functional claudication distance (FCD)* is the distance at which a patient prefers to stop because of claudication pain.

Lower Extremity Performance

Patients with PAD have decreased static balance in side by side, tandem and semi-tandem positions. Time for five chair stands, tandem walk along a 2 m line and number of rapid step ups in 10 seconds have been used as measures of lower extremity performance.

Walking Speed

Walking speed is measured by asking the patient to walk at a comfortable pace over a specified distance. In people with PAD, low ABI values are associated with slower walking at both usual and maximal speeds.

The 6-minute Walk Test Distance

When treadmill testing is not practical, the 6-minute walk test may be substituted as a useful measure of community-based walking ability. The 6-minute walk distance correlates well with maximal claudication time during treadmill testing. Normally 6-minute walk test will be repeated twice, as the second test distance has been identified as significantly greater than first trial. But in PAD subjects, the 6-minute walk test can be completed only once in subjects with PAD.

Walking Impairment Questionnaire

The walking impairment questionnaire (WIQ) is the most commonly used PAD-specific tool to assess self-reported walking ability. The distance score of the WIQ correlates with the peak treadmill walking time and is sensitive to changes in performance after exercise training. Difficulty walking quickly was correlated with peak TM walk time. The WIQ also is sensitive to change after intervention. The three domains are walking distance, walking speed and stair climbing. In each domain score ranges from 0 to 100 with lower scores indicating lower performance. The overall score is the average of all three subscores and combined scores are the average of two distinct subscales (e.g. distance and speed, distance and stairs, speed and stairs).

Physical Activity Measurement

The level of physical activity may vary from patient to patient and may be an important contributor to walking speed and physical function. Several tools have been used to assess physical activity in subjects with PAD.

The Stanford 7-day Physical Activity Recall

It asks patients to review activities performed over the previous week and are guided to report the duration of low, moderate and hard activity. This questionnaire (PAR) has been adapted for use with patients with PAD and low activity levels.

Leisure-time Physical Activity Questionnaire

Participants are asked to report the number of hours spent in each of 15 specified activities. Leisure-time physical activity questionnaires (LTPAQ) provide an estimate of weekly energy expenditure; either in total kcal expended per week or metabolic equivalent (MET) level per week.

Quality-of-Life

Quality-of-life (QOL) can be defined as the functional effect of an illness and its consequent therapy upon a patient as perceived by the patient. These functional effects are usually has limitations in physical, psychological and social functioning. Instruments can be classified into generic instruments and disease

specific instruments. Generic equipment allow comparison across populations of patients with different diseases whereas disease-specific instruments are sensitive to key dimensions of quality of life that are impaired by specific diseases.

Global quality-of-life measures are infrequently used for measuring medical interventions, because global measures may be influenced by factors other than health. Generally, the factors that contribute to quality-of-life include aspects of health (or disease), physical abilities, as well as an individual's personal beliefs and their social supports.

Health-related quality-of-life (HRQOL) measures are based on the premise that health is the most important contributor to QOL. It can be generic or disease specific. A generic tool is used to assess outcomes is that the results may be compared to other patients population or existing norms. Disease-specific instruments are primarily useful as a pre- and post-intervention measurement. These instruments are not useful when the goal is to compare populations with two different diseases or when evaluating multidisciplinary interventions.

Generic QOL Instruments in Arterial and Venous Disorders

SF-36, Nottingham health profile, WHOQOL, EuroQOL are used for both arterial and venous disorders.

SF-36

The SF-36 is widely used in subjects with arterial and venous disorders. It has nine domains with 36 items. In subjects with PAD, SF-36 scores are lower than population norms. SF-36 scores are not directly related to the level of physical function, in that QOL scores are not correlated with treadmill walking. However, the physical component score (PCS) is correlated with some walking tests. Intermittent claudication appears to impair all aspects of life; however, disease severity most significantly affects the physical component score.

Nottingham Health Profile

Nottingham health profile (NHP) is a generic self-report HRQOL tool designed to measure perceived health problems and the effect on daily activities. The six dimensions measured include energy, pain, sleep, physical mobility, social isolation and emotional response. Higher scores indicate more health problems.

World Health Organization Quality-of-Life Tool

World health organization quality-of-life tool (WHO-QOL) is a 100 item questionnaire used as a generic, multidimensional HRQOL tool. Results of this tool in subjects with PAD indicates that a greater number of co-morbid conditions produced lower QOL scores, specifically in the areas of overall and general health as well as fatigue. The more severe the IC, the lower the QOL scores, especially in independence, activities of daily living (ADL), and work capacity.

EuroQOL

It is another generic HRQOL instrument used in PAD. This scale consists of five dimensions, which measures physical and psychological status.

These are mobility, self-care, usual activities, pain and anxiety. Subjects with PAD had significant changes in scores following an intervention of vascular surgery. This suggests that this tool is responsive to change and may also be useful in evaluating the effects on noninvasive interventions.

Disease-specific Quality-of-Life Tool Instruments in Arterial Disorders

Claudication Scale

Claudication scale (CLAU-S) has five domains including pain, emotional health, psychological status, daily living and social limitations and it is responsive to treatment.

Sickness Impact Profile

Sickness impact profile scale is based on sickness-related behavior. Twelve of the 136 items applicable to IC were used to produce an IC scale called the sickness impact profile-intermittent claudication (SIP-IC).

VascuQOL

VascuQOL tool has 25 items contain five domains and they are pain, symptoms, activities and social and emotional response.

Peripheral Arterial Disease Quality-of-Life-Tool

Peripheral arterial disease quality-of-life-tool (PADQOL) tool has 60 items and asks subjects to agree or disagree with a statement and also to rank the importance of each statement.

Disease-specific Quality-of-Life Instruments in Venous Disorders

Freiburger Questionnaire of Quality-of-Life

Freiburger Questionnaire (FLQA) tool contains 83 items with four dimensions (physical, psychological, social, other) such as physical complaints, everyday life, emotional status, social life, therapy, satisfaction and occupation.

Tübingen Questionnaire for Chronic Venous Insufficiency (TLQ-CVI)

Tübingen questionnaire for chronic venous insufficiency (TLQ-CVI) tool has four dimensions (physical, psychological, social, other) with components such as physical condition, functional status, psychological well-being, social repercussions and general health.

VEINES-QOL

This tool has 26 items with three dimensions (physical, psychological, other) contains questions on symptoms, limitation in activities, psychological impact, changes in the past year, time of day of highest symptom severity.

Chronic Lower Limb Venous Insufficiency Questionnaire

Chronic lower limb venous insufficiency questionnaire (CIVIQ) have 20 items with dimensions on pain, physical functioning, psychological functioning and social functioning.

Disease-specific QOL Instruments Especially for Varicose Vein

Clinical varicose veins questionnaire is a 15 item questionnaire with dimensions on symptoms, pain, interference with daily activities and work, treatment and concern about appearance.

Disease-specific QOL Instruments in Venous Thrombosis

Health-related quality of life questionnaire is for deep vein thrombosis. It contains three dimensions such as physical, psychological and others.

Disease-specific QOL Instruments in Venous Ulcer

Charing cross venous ulcer questionnaire, self-report QOL questionnaire for patients with leg ulcers and leg ulcer measurement tool (LUMT). Charing cross venous ulcer questionnaire contains 32 items, which has dimensions on domestic activities, emotional status, social functioning and cosmetic appearance. LUMT contains 29 items with dimensions on functional limitations, dysphoric mood and treatment.

OUTCOME MEASUREMENT IN WOUND ASSESSMENT

Wound Tracing Method

Wound tracing can be accomplished by outlining the wound circumference onto a transparent film applied directly over the wound. The wound surface area can be determined from wound tracing using a planimeter. The percentage of decrease in wound surface area from wound size (% WSA) can be followed up.

Photographic Wound Assessment Tool

Wounds will be photographed with a camera that can adjust variations in adjusts in lighting, allow close up images. The photographic wound assessment tool (PWAT) is a pen and paper tool, which has six domains that assess composition of the wound bed and viability of the wound edge and periulcer skin. Each domain score can range between 0 and 4, and total score between 0 and 24. The lower score indicates better healed wound.

Pressure Sore Status Tool

Pressure sore status tool (PSST) has 13 domains that measures characteristics of wound—size, depth and wound bed composition, wound exudates, viability

of wound edge and periulcer skin on a scale from 1 to 5. Score can range from 13 to 65. The lower score indicates better appearance. This is now replaced by Bates-Jensen wound assessment tool.

Pressure Ulcer Scale for Healing Tool

The pressure ulcer scale for healing tool (PUSH) is a tool originally developed for measuring the healing of pressure ulcer. The PUSH tool involves assessment of three wound characteristics—surface area measurements, exudate amount and surface appearance. Use of the tool therefore involves measuring size, evaluating exudate and categorizing tissue type. The clinician measures the size of the wound, using length and width to calculate surface area (length × width) and chooses the appropriate size category on the tool (there are 10 size categories, from 0 to 10). Exudate is evaluated as none (0), light (1), moderate (2) or heavy (3). Tissue type choices include-closed (0), epithelial tissue (1), granulation tissue (2), slough (3) and necrotic tissue (4). The three subscores are then summed for a total score (17) and can be plotted on a graph. PUSH scores are responsive to change in ulcer status and can differentiate between ulcers that heal and those that do not. The PUSH tool has also been validated for use in monitoring healing in diabetic foot ulcers and venous ulcers.

Bates-Jensen Wound Assessment Tool

Bates-Jensen wound assessment tool (BWAT) is a measuring instrument used to assess and monitor healing in pressure ulcers and other chronic wounds. It uses a numerical scale to rate wound characteristics from best to worst possible. Two items are nonscored and they are location and shape. The remaining 13 are scored items. These are size, depth, edges, undermining or pockets, necrotic tissue type, necrotic tissue amount, exudate type, exudate amount, surrounding skin color, peripheral tissue edema, peripheral tissue induration, granulation tissue and epithelialization. Each scored item appears with characteristic descriptors rated on a scale (one indicating best for that characteristic and five indicating worst). Once a lesion has been assessed for each item on the BWAT, the 13 item scores can be summed to obtain a total score for the wound. Total scores range from 9 (wound closure) to 65 (profound tissue degeneration).

Vancouver Scar Scale

Vancouver scar scale as a method for assessing burn-related scars. This scale uses the variables of pigmentation, vascularity, pliability and height of the scar to describe the current status of the tissue. Pigmentation can be normal, hypopigmented or hyperpigmented. Vascularity can be normal, red, pink and purple. Pliability can range from normal to contracture. Height can be normal to raised (> 5 mm). Scores are assigned based on variances of these variables from normal, with normal being zero. A higher score represents a worse scar with a maximum score of 13.

9 Physiotherapy Management of Vascular Disorders

9.1 PHYSIOTHERAPY MANAGEMENT FOR ACUTE ARTERIAL DISORDERS

In acute arterial disorders usually immediate medical attention is required followed by surgical intervention. Commonly surgeries performed for these disorders include thrombectomy, bypass graft, embolectomy, endarterectomy.

GOALS

Physiotherapy treatment goals include:

- To decrease ischemia by restoration or improvement of blood flow
- To protect limb and skin care.

Box 9.1.1 Contraindication for physiotherapy

Contraindication to exercise in acute arterial occlusion

- Graded ambulation or bicycling is discontinued if the leg pain increases rather than decreasing
- Patient with resting pain should not participate in active or passive exercises
- If leg pain increases rather than decreases
- Presence of skin irritation, an ulceration, a wound or a fungal infection of the feet

General contraindication in acute arterial occlusion

- Prolonged positioning during bed rest, which could cause pressure on and potential breakdown of skin
- Local, direct heat on the involved extremity because of the potential for a burn to the ischemic tissue
- Use of support hose, which may increase peripheral resistance to blood flow
- Restrictive clothing that could compromise blood flow

TECHNIQUE

Physical intervention to improve peripheral blood flow, while patient is on bed rest include warming the limb by reflex heating of opposite extremity or elevating head of the bed slightly. Rest pain can be minimized by sleep with the legs in a dependent, but supported position over the edge of the bed. Contraindication for physiotherapy is mentioned in the **Box 9.1.1**. Pressure on skin can be minimized by special mattress and turning schedule.

9.2 PHYSIOTHERAPY MANAGEMENT FOR CHRONIC ARTERIAL DISORDERS

PROBLEM LIST

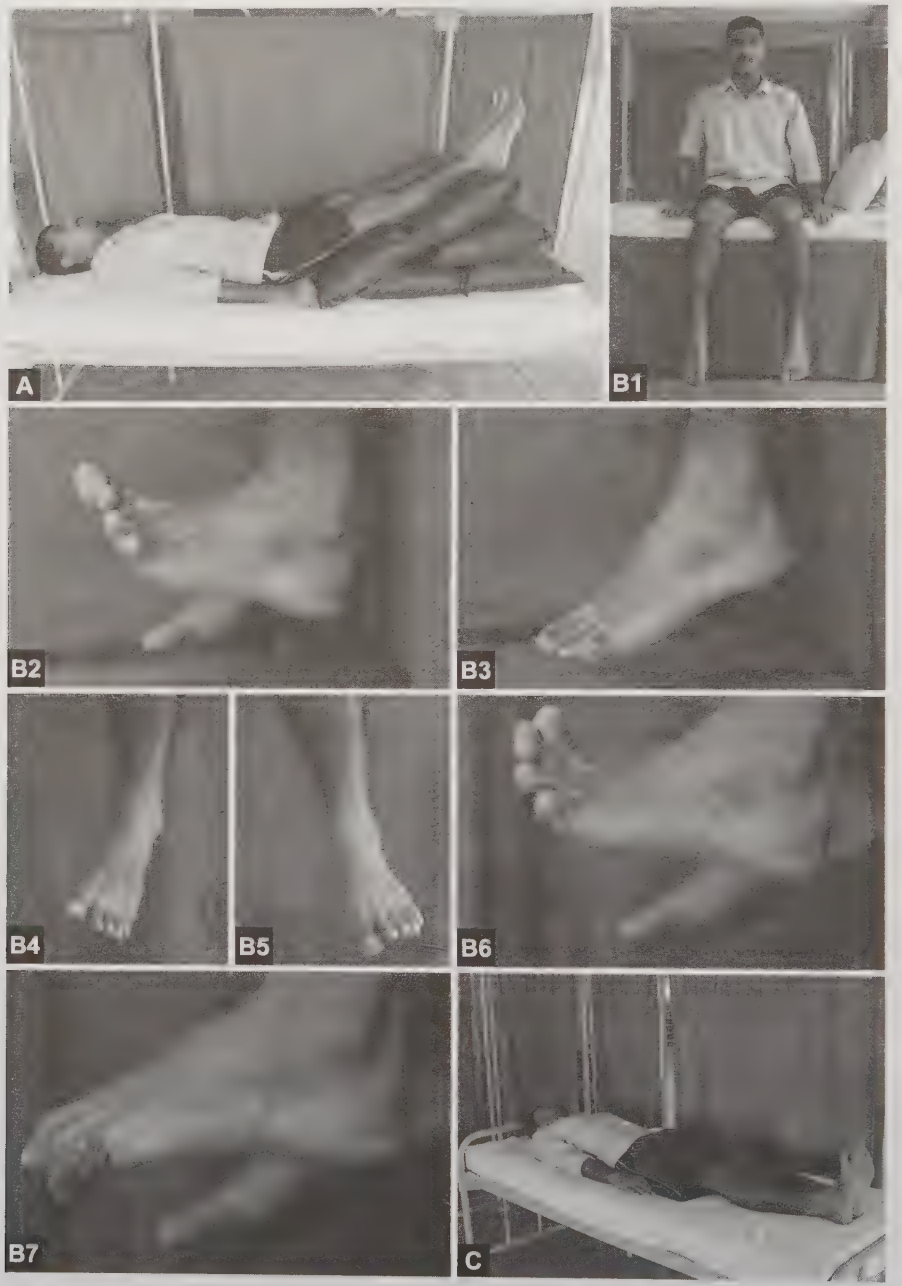
- Decreased endurance and increased frequency of muscular fatigue with functional activities such as walking
- Pain with exercise or at rest
- Skin breakdown and ulcerations
- Limitation of passive and active range of motion
- Weakness and disuse atrophy.

GOALS

- To improve the exercises tolerance for the activities of daily living (ADL) and to decrease the incidence of the intermittent claudication
- Promote vasodilatation/increase circulation
- Relieve pain at rest
- Prevent or minimize joint contracture and muscle atrophy particularly if the patient is confined to bed
- Prevent skin ulceration
- Promote healing of skin ulceration that is developed.

PHYSIOTHERAPY TECHNIQUES

- *Traditional Buerger's exercises and modified Buerger's exercise*
  - *Principle*: This exercise uses the principle of gravity
  - *Rationale*: These exercises improve the collateral circulation.
  - *Procedure*: It consists of three phases **(Figs 9.2.1A to C)**:
    i. Leg in elevation of 45° to horizontal until skin blanches for 2 min. The purpose of this phase is to assist venous circulation. As the leg is elevated gravity pull back the blood towards the heart, which empties the veins.



Figs 9.2.1A to C Buerger's exercise. (A) Patient is lying in supine position, legs 45° elevated; (B1) Patient in high sitting position: Performing; (B2) Ankle dorsiflexion; (B3) Ankle plantar flexion; (B4) Inversion; (B5) Eversion; (B6) Dorsiflexion of ankle and dosiflexion of toes; (B7) Plantar flexion of ankle with plantar flexion of toes; (C) Patient is lying in supine position *(For color version, see Plate 4)*

ii. In traditional Buerger's exercises patient sits with leg dependent position until skin color is bright red for 3 minutes for arterial circulation. In modified Buerger's exercises, leg movement such as dorsiflexion, plantar flexion, and dorsiflexion with inversion of the ankle or dorsiflexion of ankle with dorsiflexion of toes and plantar flexion of ankle with eversion or plantar flexion of ankle with plantar flexion of toes are performed as cycle of sets in high-sitting position with legs dependent. The purpose is to increase arterial circulation. As the legs are dependent, gravity pulls the blood towards the feet, which favors arterial circulation.

iii. Patient lies with leg horizontal until color returns to normal for 5 min. The purpose is to enhance the both arterial and venous circulation. As the position is gravity eliminated position it favors both arterial and venous circulation.

- *Frequency*: Exercises are performed five times for three sets in a day.
- *Prognosis sign*: Improvement is determined by decrease in the time required for change in the skin color changes.

- *Contrast bath*: It is a superficial heating modality where the limb is dipped alternatively between hot water and cold water; three minutes dipped in hot bath and one minute in cold bath. The rationale of the technique is to accelerate the rate of blood flow in peripheral vessels.
- *Iontophoresis*: It is the introduction of various ions into the skin by means of electricity. In order to drive the ions into the tissues, a direct (Galvanic) current needs to be employed. The electrical part of the iontophoresis uses a direct current (DC) that is positively or negatively charged active electrode and an oppositely charged dispersive electrode. The medication is then placed on the active electrode so that the active electrode has the same charge as the medication. When the electrodes are placed on the skin and activated, the charge from the electrode will drive the medication away from the electrode and into the area being treated. This electrical charge will actually push the medication through the skin and into the tissue to be treated. This treatment helps in decreasing the pain, improving circulation to the affected area and enhances healing of the wound.
- *Reflex heating of* contralateral limb increases vasodilation in the ipsilateral limb.
- *Rest pain can be minimized* by sleep with the legs in a dependent, but supported position over the edge of the bed or with the head of the bed slightly elevated.
- *Repetitive, active range of motion (AROM) against low-loads* can be given to minimize or prevent muscle atrophy.
- *Gentle stretching exercises or proper positioning* in bed to maintain joint and muscle extensibility.
- *Connective tissue massage*: It should be started from the sacral and lumbar region of the spine followed by cervical region and extremities. The rationale of this technique is that it reduces the tension in the back and patient feels warmth.

- *Vasotrain vacuum compression therapy device*: This provides alternating cycle of positive and negative pressure in which affected extremity rest inside an air tight cylinder. Negative pressure increases the blood flow whereas positive pressure increases the venous return. It also helps in wound healing.
- *Skin care*: Skin ulcerations can be prevented by proper care and protection of the skin particularly the feet or hands, proper nail care, proper shoe selection and fit. Avoid use of support hose and restrictive clothing, avoid exposure to extremes of temperature, i.e. both hot and cold.
- *Wound management* procedures for treating ischemic ulcers, including electrical stimulation and oxygen therapy (Refer Chapter 14.7).
- *Nutritional counseling* for weight control.
- *Patient education*: Patient must be advised to stop smoking and alter their diet, including limitation and avoidance of salt, sucrose and alcohol to lower their blood pressure, triglyceride and cholesterol levels.
- *Intermittent compression therapy*: It delivers high pressure at quick burst that can empty venous system. This seems to trigger an arteriovenous (AV) reflux that results in greater arterial inflow.
- *Graded exercise program*: Regular, graded aerobic conditioning program of walking or bicycling. Improve exercise tolerance for ADL and decrease the incidence of intermittent claudication.
- *Ratschow's exercise*: These are the specific exercises intended to improve collateral circulation to the feet and legs. The lower extremities are elevated to a 45–90° angle and ankle joint rotation is exercised until the skin blanches (appears dead white). The feet and legs are then lowered below the level of the rest of the body until redness appears. A positive effect is achieved by repeating and carrying out this exercise several times a day.

9.3 EXERCISE PRESCRIPTION IN PERIPHERAL ARTERIAL DISORDERS

Regular, graded aerobic conditioning program of walking or bicycling improve exercise tolerance for activities of daily living (ADL) and decrease the incidence of intermittent claudication.

RATIONALE

During active contraction of the muscle, blood flow temporary decreases, but rapid increase in blood flow occurs immediately after muscle contraction is over. After cessation of exercise, there is a rapid decrease in blood flow during the first 3–4 minutes. This is followed by a slow decline to resting levels within 15 minutes. With the repeated moderate exercise, blood flow in the muscle can be increased beyond the resting values for blood flow.

EFFECTS OF AEROBIC EXERCISE IN PERIPHERAL ARTERIAL DISEASE

The physiological, metabolic and mechanical alterations that occur during the period of exercise presumably stimulate an adaptive response that ultimately reduces claudication symptoms. Following are the effects:

- Increased peripheral blood flow because of formation of collateral vessels (angiogenesis), redistribution of blood flow, increased nitric oxide activity and increase in muscle capillary density (enhanced surface area).
- Improvement in oxygen delivery because of decreased blood viscosity, which increases more extraction of oxygen.
- Improvement in muscle metabolism because of increased oxidative enzyme activity.
- Improvement in lipid profile, obesity, blood pressure, glucose metabolism enhances the endothelial function.
- Reduces the plasma concentration of carnitine, which are the main causes for claudication.
- Improvement in walking ability because of change in gait.
- Improvement in walking efficacy because of less energy requirement for a given work load.
- Increase in the time before the onset of exercise pain, i.e. pain threshold during walking (alteration in pain perception).
- Improvement in the efficiency of oxygen utilization in exercising muscles (enabling patients to tolerate exercise over longer periods of time).
- Lower oxygen consumption for a given work load.
- Increase in range of motion and muscle strength through exercises.
- Quantitative improvement of quality of life.

EFFECTS OF RESISTED EXERCISE IN PERIPHERAL ARTERIAL DISORDERS

Surveys have shown that adults with peripheral arterial disorders (PAD) have smaller calf muscle area and poorer leg strength than those without PAD. This result is in greater functional impairments among people with PAD. The benefits of resistive training in PAD are less well known than those of walking. Since in claudication the main deficit is ability to walk and given the principle of specificity of training, it is not surprising that walking results in greater benefit.

EXERCISE GUIDELINES

Exercise Testing

- Patients with PAD generally are classified as high-risk; thus, exercise testing should be conducted in the presence of a physician.
- Because of the high-risk of cardiovascular disease in this population, exercise testing with electrocardiogram (ECG) monitoring should be performed, so that ischemic symptoms, ST-T wave changes in ECG, or dysrhythmias may be identified.

- Use scales of subjective ratings of pain.
- Multistage discontinuous protocol may be necessary to achieve peak oxygen consumption.
- Graded treadmill protocol at 2 mph with modest increases in grade of 3.5% every 3 minutes or 2% every' 2 minutes or a gradual ramp protocols may be used.
- Record time or distance to the onset of claudication pain and the maximal walking time or distance.
- Arm ergometry or pharmacologic stress testing can be used in patients who cannot perform leg exercise to assess cardiovascular status.
- Questionnaires are useful adjunct to exercise testing to assess community based activity levels.

Exercise Prescription

- The recommended mode, frequency, duration and overload generally arc consistent with general prescription those for aerobic, resistance, and flexibility exercise stimuli.
- Initial enrollment in a medically supervised program with ECG, heart rate, and blood pressure monitoring is encouraged.
- Warm-up such as static stretching, isotonic pumping exercises of the ankle and toes, and cool down period of 5–10 minutes each should be included.
- Treadmill and track walking are the most effective approaches to reduce claudication and should be performed 3–5 days per day.
- Initial treadmill workload is set to elicit claudication symptoms within 3–5 minutes. Patients walk at this workload until they reach claudication of moderate severity. This is followed by a brief period of standing or sitting to allow symptoms to resolve.
- The exercise-rest-exercise pattern is repeated throughout the exercise session.
- The initial duration is a total of 35 minutes of intermittent walking and increased by 5 minutes each session until 50 minutes of intermittent walking can be completed. Ultimately, 35–50 minutes of continuous walking is desired.
- Cardiac signs and symptoms may appear as patients increase their exercise capacity and reach higher heart rates and blood pressure.
- Resistance training and/or upper body ergometry is complementary, but not substitute for walking.

  A sample components of exercise prescription is given in **Table 9.3.1**.

SPECIAL CONSIDERATIONS

The most common procedure for assessing the peripheral circulation is the ratio of ankle to arm systolic blood pressure ankle-brachial index (ABI). Ankle systolic blood pressure (BP) and ABI are further reduced after exercise because blood flow is shunted into the proximal leg musculature at the expense of the periphery and distal circulation in the leg. Although serial measurements of

Table 9.3.1 Summary of exercise programming

Components	*Aerobic exercise*	*Resisted exercise*	*Flexibility exercise*
Mode	Treadmill walking Track waking	8–10 exercises of major muscle group	Static stretch of all major muscle groups
Training intensity	*Initial* • Set by results of the treadmill • Starting work load brings on claudication pain *Subsequent* • Speed or grade is increased if patient walks more than 8–10 minutes • Grade increased first if speed is more than 2 mph • Speed increased first if less than 2 mph	*Volitional fatigue*: • 19–20 RPE *Stop 2–3 repetition before volitional fatigue*: • 16 RPE	Stretch to tightness at the end of the range of motion, but not to pain
Duration	*Initial*: 35 minutes (intermittent) *Subsequent*: Add 5 minutes every session until 50 minutes is possible	1 set of 3–20 Repetitions Progress no of sets	15–30 seconds 2–4 times each stretch
Frequency	3–5 times per week	2–3 days per week	*Minimal*: 2–3 days per week *Ideal*: 5–7 per week

ABI are used for assessing progression in disease severity, an increase in leg blood flow is not a common response to exercise training. Hence, ABI is not useful for assessing the efficacy of intervention. Time to onset of intermittent claudication on exercise test is valuable outcome measure.

Patients with peripheral arterial disorders are also at risk of cardiovascular risk. So to promote safety, all the patients with clinical evidence of comorbid coronary disease are telemetry monitored during exercise sessions to evaluate heart rate, rhythm, blood pressure. Improvement in exercise tolerance may unmask myocardial ischemia. Knee stiffness is exacerbated in patients with peripheral arterial disorders, who have existing musculoskeletal complications. Cold weather induces vasoconstriction requires long warm up before exercising in cold environment. For patients with skin lesions multiple brief walking periods throughout the day may help in healing through increased tissue oxygenation. Patient with peripheral vascular disease (PVD) also will be having neuropathy, so sensory evaluation, foot hygiene are crucial.

Beta blockers may decrease time to claudication. Pentoxifylline, dipyridamole, aspirin and warfarin may improve time to claudication.

PRECAUTIONS

- Avoid exercising outside during very cold weather.
- Wear shoes that fit properly, have sufficient padding and do not cause skin irritation.

- Inspect the feet carefully for evidence of skin irritation after each exercise session.
- Discontinue a walking program if leg pain increases rather than decreases overtime.

CONTRAINDICATIONS

- Presence of skin irritation, ulceration, a wound or a fungal infection of the feet.
- Leg pain at rest because of advanced vascular disease.

9.4 CARE OF ANESTHETIC FOOT AND HAND

To achieve a healthy, clean, well-moisturized and intact skin, skin care is most important in the management of vascular diseases. Assess the skin and identify any problems, so that patients, can be advised regarding management of their skin. Good skin care regime promotes skin integrity and reduces the risk of infection.

SKIN ASSESSMENT

Inspection and Observation

- Inspect your feet daily
- Look for blisters, corn, redness, cuts, breaks or accidental trauma
- Swelling, pain and drainage
- Look for discoloration.

Two skin assessment tools, which are not currently validated, have been proposed for use within lymphedema management to assist healthcare professionals in their assessment of a patient's skin. Linnett (2000) proposed a simple grading system to assist in measuring the outcome of skincare according to the physical condition of the skin established on visual examination.

An earlier scoring system was proposed by Badger and Jeffs (1995) to consider the condition of the skin and subcutaneous tissues in relation to the site of the swelling, the shape and size of the limb. In this assessment tool a score is calculated from the assessment in order to evaluate the effect of lymphedema management.

While grading or scoring tools can be useful in assessing the condition of the skin and evaluating the outcome of skincare, subjective assessment by the patient and healthcare professional remains vital and should include the wider impact of the skin's condition upon the patient. A poor skin condition with associated problems can cause discomfort and influence quality of life. The healthcare professional should work with the patient to improve the condition of the skin where necessary, by providing appropriate information to promote

and maintain skin integrity and considering the patient's individual needs. The treatment of any underlying skin conditions should be a priority in lymphedema management as these may influence the outcome of care. The disease status of patients with a cancer diagnosis should always be established in order to assess, whether skin problems may be related to disease progression.

Palpation

Palpation of the skin should always accompany a visual examination and will reveal, whether the skin is hot a symptom that may imply infection or chronic inflammation. Palpation will also indicate the condition of the subcutaneous tissues.

Skin Cleansing

Gentle, daily cleansing of the skin should aim to remove pathogenic organisms, dust and dirt, but not to be so vigorous that natural oils, which protect the skin are also removed. A mild, unperfumed soap or soap substitute such as aqueous cream is preferred and abrasive soaps should be avoided.

Care should be taken when drying the skin. Rubbing will disturb the skin barrier function and should be replaced by patting of the skin during drying, taking care to dry the skin thoroughly. Particular attention should be paid to skin creases or folds and the spaces between the digits or toes where residual moisture in a warm environment can lead to fungal infections.

Skin Moisturizing

The daily moisturizing of skin in an area where edema is present or possible will encourage moisture to be retained by the skin and skin integrity to be promoted. Any dryness, flaking or cracking of the skin indicates that the skin is no longer intact and the increased colonization of potentially harmful bacteria in dry skin may compound the reduced immunity of the limb and increase the risk of infection developing. Moisturizing of the skin is therefore important for all patients, to prevent dryness occurring.

Emollient agents provide a surface lipid film to the epidermis that prevents water loss from the skin and reduces the risk of dryness. Emollients can be divided into three categories:

1. *Bath oils:* These help to restore the integrity of the skin and prevent it from drying out.
2. *Soap substitutes:* These can be mixed with water to provide a liquid soap with an oil and water content and should be used in conjunction with an emollient cream to rehydrate the skin if it is very dry.
3. *Moisturizers:* These may be in the form of lotions, creams or ointments.

The choice of emollient depends upon the condition of the skin:

- Well-hydrated skin requires a bland moisturizing emollient in the form of a lotion or cream that is easily applied and absorbed.
- Dry, flaky skin requires an emollient ointment that is greasy and forms an impermeable layer over the skin.

- Scaly/hyperkeratotic skin requires a soap substitute and moisturizing ointment to soften the areas involved, so that they can be removed.

Emollients should usually be applied once a day, but when the skin is scaly and hyperkeratotic, a twice-daily application is preferred. Applying the emollient liberally to the skin at night will ensure that the skin benefits from its application before any compression garments are fitted the next morning. The moisturizer should be applied to the whole of the limb and adjacent part of the trunk, with the last movement carried out downwards in the direction of the hair growth. This ensures that the moisturizer does not accumulate in the hair follicles that can lead to a condition called folliculitis in where there is inflammation of the hair follicles and sometimes infection.

PREVENTION

- Wash gently every day using lukewarm water and mild soap
- Dry thoroughly specially between toes
- Use powder or corn starch between toes.

EDUCATION

- Do not walk bare foot.
- Do not sit with your legs crossed.
- Do no wear stocking with elastic tops.
- Do not smoke.
- Do not expose to cold weather.
- Do not use heating pad.
- Do not get sunburn.
- Do not dig into corners of toes.
- Wash the skin daily with soap and dry carefully to promote skin hygiene.
- Keep the skin moist and well-hydrated by using a moisturizing cream daily can prevent skin dryness and chafing and promote skin integrity.
- Treat any cuts or breaks in the skin antiseptically to minimize the risk of infection.
- Use a high-factor sun cream, if the skin is exposed to the sun or cover the affected area because sunburn causes an inflammatory response and increased lymphatic load as the body tries to correct the damage to the skin.
- Use an insect repellent, if there is a likelihood of mosquito bites because the piercing of the skin and the vector of the mosquito are risk factors for infection.
- If the arm is at risk, wear protective gloves, when using the oven, using household cleaning agents and gardening. This can minimize the risk of accidental trauma to the skin that can lead to infection.
- Use an electric razor rather than a blade razor or chemical hair remover when removing unwanted body hair. This will avoid trauma and damage to the skin.
- Avoid needle sticks, blood tests, injections and intravenous infusions in the limb that is at risk because lymphedema may be triggered by an inflammatory response to the skin puncture or the development of infection entering through the skin.

- Pay attention to care of the nails on the hands or feet; do not cut or tear cuticles; use a cuticle stick covered with cotton wool. This will avoid trauma to the skin that may result in infection.
- Wear well-fitting, comfortable shoes, if the feet are affected. This will prevent trauma to the skin and the formation of blisters that may cause infection.

9.5 PREVENTION OF VASCULAR DISEASES

DIET

Elevation of plasma concentration primarily triglycerides and cholesterol contributes the formation of atherosclerosis. So patients with peripheral vascular disease (PVD) will benefit from reducing diet, in which fat intake is limited.

SMOKING

Vasoconstriction in terminal vessel occurs during smoking. So encourage patient to quit smoking.

HYGIENE

The patient will need to be advised to be extremely careful with his/her personal care particularly frequent washing and inspection of skin of feet and legs, wearing clean socks that should be well-fitting as should the shoes. He/she should understand, why it is important to keep the feet and hands clean and drying all weather conditions, especially if the sensation is poor and the skin is showing signs of trophic changes. The patient should care attention to nails, callosities and to take professional advice in treatment of blisters and minor skin lesions. There should be particularly careful of hot objects, e.g. hot water bottles, hot radiators, cookers and hot food spillage.

ACTIVITY

The patient's exercise tolerance should be carefully assessed and he/she should be encouraged to exercise frequently within limits of claudication or breathlessness. The patient should be advised not to lie in bed, sit or standstill for long period of time as this will contribute to venous stasis and further devitalize tissues.

DRUGS

If vasodilators are given orally their effect is general over the whole body with the normal vessels dilating more easily than those which are diseased and diverting blood from the areas, where it is most needed. If the patient is on

anticoagulants then even minor surgery such as dental extractions and trauma such as cuts and bruises could become serious, when the clotting time is lengthened.

9.6 PHYSIOTHERAPY TREATMENT IN ACUTE VENOUS DISORDERS

Immediate medical management is essential in this life-threatening disorder. During the initial stages of treatment, the patient will be on complete bed rest and systemic anticoagulant therapy and the involved extremity will be elevated. Movement of the extremity will cause pain and will increase congestion in the venous channels in the early inflammatory period.

GOALS

- To relieve pain during inflammatory period
- To regain functional mobility when the symptom subside
- To prevent recurrence of the acute disorder.

PHYSIOTHERAPY TREATMENT

- *Bed rest*: In severe cases the patient may be confined to bed for a short period until the local signs subside. This helps the thrombus to adhere to the venous wall.
- *Elevation*: In mild to moderate severity, legs are kept in elevated to maintain venous circulation.
- *Foot and leg exercises*: The patient should be encouraged to carry out foot exercises in the pain free range with the leg elevated.
- *Compression*: Firm elastic bandaging, stockings or crepe bandage are applied from the toes to beyond the upper limit of the affected area.
- *Pain relief*: Application of moist heat such as hot packs to entire length of the involved extremity can be given to relieve pain. Keeping the knees slightly flexed can also decrease pain.
- *Ambulation*: Graded ambulation with legs wrapped in elastic bandages or when pressure gradient support stockings are worn can regain functional mobility. The patient should avoid sitting or standing still for any length of time and either resting with legs elevated or walking is encouraged to prevent recurrence of the acute disorder.

PREVENTION OF VENOUS THROMBOSIS

Risks can be minimized by:

- The postoperative patients or inpatients confined to bed with a cradle under leg clothes, legs can be elevated periodically (15–22 cm).

- Thromboembolism-deterrent (TED) (antiembolic stockings) should be worn by all patients who are confined to bed postoperatively.
- Active pumping exercises (dorsiflexion and circumduction of the ankle) performed regularly throughout the day, while the patient is in bed.
- Active or mild resistive range of motion to both lower extremities if the postoperative condition permits.
- Daily passive range of motion if active exercise is not possible because of paralysis and medical conditions.
- Early ambulation will enhance the venous circulation and prevent of stasis of venous blood.
- Postoperatively patients should not sit with legs dependent.
- General breathing exercises will enhance the venous return.
- Any risk factor should be minimized, e.g. stop contraceptive pill before planned surgery.
- Anticoagulant therapy, e.g. low dose heparin may be given postoperatively in high-risk cases or dextran during surgery.

9.7 PHYSIOTHERAPY MANAGEMENT OF CHRONIC VENOUS DISORDERS (CHRONIC VENOUS INSUFFICIENCY AND VARICOSE VEIN)

PROBLEM LIST

- Edema
- Increased risk of skin ulcerations and infections
- Aching of involved limb
- Decreased functional mobility, strength, and endurance.

GOALS

- Teach the patient how to prevent or minimize impairments
- Prevent lymphedema; minimize venous stasis
- Increase venous return and reduce lymphedema if already present
- Prevent skin abrasions, ulcerations and wound infections.

MANAGEMENT TECHNIQUES

- *Elastic support*: This increases the efficiency of calf muscle as a pump. This may be elastic stockings or elastic bandages. Use of individually tailored pressure-gradient support stockings donned before getting out of bed in the morning and worn every day. Support garment worn during exercise and ambulation.

- *Ambulation*: Light active exercise, such as walking on a regular basis.
- *Elevation*: Elevation of lower legs for 10 minutes 3 times a day and sleep with end of bed raised. Also elevate the lower extremities after graded ambulation until the heart rate returns to normal.
- *Massage*: Manual massage to drain edema. Stroking has to be done in a distal-to-proximal direction. Clear the proximal nodes and areas of lymphedema first, then the middle, and finally the distal areas.
- *Range of motion*: Active ROM (pumping exercises) of the distal muscles, while involved limb is elevated.
- *Relaxation*: Elevate involved limb(s) above the level of the heart (about 30–45°) when resting or sleeping.
- *Mechanical compression pump*: Use intermittent mechanical compression pump and sleeve with involved limb elevated for several hours a day.
- *Education*: Patient education and self-management skills for skincare, self-massage for lymphedema, advised on how to dependent edema and a home exercise program.

PRECAUTIONS

- Avoid prolonged periods of standingstill and sitting with legs dependent.
- Do not cross your legs when sitting.
- Exercise regularly. Walking is a good choice. It improves leg and vein strength.
- Keep your weight down.
- Avoid standing for prolonged periods of time. If your job or hobby requires you to stand, shift your weight from one leg to the other every few minutes.
- Wear elastic support stockings.
- Do not wear clothing or undergarments that are tight or constrict your waist, groin or legs.
- Eat high-fiber foods such as bran cereals, whole grain breads, and fresh fruits, as well as vegetables to promote regularity (As constipation contributes to varicose veins).
- To prevent swelling, cut your salt intake.
- Exercise your legs. This can be undertaken even if you have a sedentary desk job (From a sitting position, rotate your feet at the ankles, turning them first clockwise, then counterclockwise, using a circular motion. Next, extend your legs forward and point your toes to the ceiling, then to the floor. Then, lift your feet off the floor and gently bend your legs back and forth at the knees).
- Elevate your legs when resting.
- Get up and move about every 35–45 minutes when traveling by air or even when sitting in an all day conference (Opt for an aisle seat in such situations).
- Stop and take short walks at least every 45 minutes, when taking long car rides or limited exercise of your legs as advised previously.
- Avoid tight clothing garments that constrict the legs, groin or waist.

9.8 VASCULAR REHABILITATION

DEFINITION

Vascular rehabilitation is a noninvasive exercise program that improves maximal walking distance in patients with claudication, pain in the legs with activity that subsides with rest. The patient should also be introduced this program for suspected arterial insufficiency. The components of vascular rehabilitation include patient education addressing risk factor modification and stress education techniques together with the exercise and walking programs.

Benefits of this program includes:

- Relief of pain
- Promote a greater sense of well-being
- Decrease the risk of further complications of arterial insufficiency
- Decreased discomfort with activity
- Increases walking distance and endurance
- Teaches about risk factors for vascular disease
- Assists in developing healthy eating habits
- Introduces stress awareness and management
- Offers ongoing support and encouragement
- Reduces potential need for surgery.

TEAM MEMBERS

- Vascular surgeon
- Physiotherapist
- Nurse
- Exercise physiologist
- Dietician.

INITIAL ASSESSMENT

The vascular rehabilitation program is customized for each individual based on age, ability, medical condition and prognosis. Before entering the program, a person undergoes initial vascular studies and a treadmill-walking test, ordered by their physician.

OUTPATIENT VASCULAR REHABILITATION

A participant follows the program, under the direction of a vascular surgeon.

There are two 12-week outpatient sessions:

1. Acute vascular rehabilitation
2. Maintenance vascular rehabilitation.

Participants gradually build-up their exercise tolerance (via treadmill walking) and learn lifestyle modification. Vascular rehabilitation may increase walking tolerance by 400–500%. At discharge, baseline testing is repeated and

Table 9.8.1 Vascular rehabilitation program

Weeks	*Exercise*	*Intensity*	*Ambulation*
1–3	• *Isometrics*: Quadriceps, Hamstring • *Active range of motion exercise*: Ankle pump, heel slide	Daily 3 sets 15 repetition 2–3 times daily Monitor daily	Half miles or just prior to the point of claudication
4–6	*Above and resisted exercises*: • Sanding on toes • Straight leg raise (SLR) • Wall squat • Hip, knee, ankle exercise with 2–3 lb of weight	Daily 3 sets 20 repetition 2–3 times daily	Half to 1 mile or to the point of claudication
7–10	Continues resisted exercise, resistance as much as tolerable	Daily 3 sets 20 repetition 3 times daily	1 mile distance as tolerated

Table 9.8.2 Comparison between hospital vs home based program

Hospital based programs	*Home exercised program*
Supervised	Not supervised
Treadmill based protocols	Primarily walk on level ground
More of structured with exercise goal of targeting heart rate or metabolic equivalents	More of unstructured with physical activity, e.g. walk for half an hour or 1 hour
Higher workload	Low workload
Programs with structured support and encouragement from hospital staff	No support and encouragement is given
These are costly Availability is limited Patients are taken out of their family and working environment	Poor compliance, fear of pain, inadequate knowledge, poor general condition contribute to the difficult of starting

reported to the referring physician with a recommendation to continue in a maintenance program. Exercise training is a lifelong commitment, if it is to be an alternative to surgery. **Table 9.8.1** shows a sample vascular rehabilitation program.

The rehabilitation program can be structured vs unstructured, supervised vs unsupervised, hospital based vs home based or community based program **(Table 9.8.2)**.

UPPER LIMB TRAINING VS LOWER LIMB TRAINING

Upper limb aerobic exercise training (arm cranking exercise) avoids the ischemic training experienced during lower limb exertion. It evokes similar improvements in cardiovascular fitness and walking performance in claudicants as that induced by lower limb cycle ergometry exercise. Patients can exercise in cycles of 2 minutes of exercise at a crank rate of 50 rpm, followed by 2 minutes of rest for a total exercise time of 20 minutes in a 40 minute session. Upper limb training could be used in the early stages of exercise rehabilitation program until patients feels more comfortable and confident to engage in lower limb exercise.

9.9 PHYSIOTHERAPY MANAGEMENT OF LYMPHEDEMA

To increase lymphatic drainage, the hydrostatic pressure of tissues must be increased. This is accomplished by external compression of the skin. Lymphatic and venous return can also be increased by elevation of the limb. Lymphedema caused by lymphatic disorders, such as lymphangitis and cellulitis, does not diminish as readily with elevation as compared to edema secondary to venous disorders.

PROBLEM LIST

- Presence of edema
- Stiffness of fingers
- Decreased range of motion (ROM) of affected extremities
- Ulceration
- Skin infections.

GOALS

- To reduce lymphedema
- To prevent further edema
- To achieve mechanical reduction and maintenance of limb size
- To alleviate the symptoms arising from lymphedema
- Education about preventing infections and cellulitis.

To Reduce Lymphedema

- Intermittent mechanical compression with a pneumatic pump and sleeve or bag for several hours daily.
- Elevation of the extremity above the level of the heart (about 30–45°), while sleeping and as often as possible during the day.
- Manual massage from distal to proximal along the length of the extremity.
- Isometric and isotonic pumping exercises of the distal muscles.
- Faradism under pressure.

To Prevent Further Edema

- Elastic support stocking or sleeve, individually measured and fitted to the patient.
- Regular elevation of the extremity.
- *Avoidance of sources of increased load on the lymphatics such as*:
  - Static, dependent positioning of the limb.
  - Prolonged use of muscles for even light tasks.
  - Hot environments.

To Prevent Infections and Cellulitis

- Care of skin abrasions, small burns, and insect bites.
- Avoidance of harsh chemicals and detergents.
- Frequent application of moisturizers to the skin.
- Use of antibiotics.

TECHNIQUES

- *Elevation*: The involved limb is elevated during use of a sequential compression pump, while sleeping or resting, or even during sedentary activities. The compressive bandages or garment are worn during periods of elevation.
- *Compression*: It can be achieved by either bandaging, garments or achieved with the help of pneumatic compression pump. Descriptions about these techniques are mentioned in succeeding chapters.
- *Massage*: Manual massage from distal to proximal along the length of the extremity.
- *Faradism under pressure*: Electrical stimulation of muscles by using the faradic current that generally acts as the muscle pump. The compression and elevation of the limb is to increase venous and lymphatic return so relieves edema. Pumping action of lymphatic and venous vessels by alternate contraction and relaxation of muscle and joint increases venous and lymphatic drainage.
- *Decongestive exercises or pumping exercises*: It is a sequence of lymphatic drainage exercises in the form of isometric and isotonic exercises for the drainage of distal and proximal muscles. Refer Chapter 9.10 for more details.
- *Heat therapy*: It has produced some benefits, which can be achieved by hot water immersion, microwave and electromagnetic irradiation. Microwave heat therapy has been combined with compression therapy, hot water immersion, and benzopyrones to reduce leg volume and improve skin tonometry. Histologically, the skin after heat treatment for lymphedema shows a near resolution of perivascular cellular infiltration, disappearance of the so-called lymph lakes and dilatation of blood capillaries. This decrease in the dermal inflammatory process associated with alteration of extracellular matrix may explain the reduction of lymphedema seen after heat treatment. Also heat by means of electromagnetic radiation produced its effect by increasing the venous return rather than by improving lymphatic flow.
- *Other exercises and techniques*: Deep breathing exercises, stretching, and low-intensity resistance exercises are integrated in management of lymphedema. Exercises are performed, while wearing a compressive garment or bandages and in a specific sequence, often with the edematous limb(s) elevated. A low-intensity cardiovascular/pulmonary endurance activity, such as bicycling, often follows ROM and strengthening exercises.

Combined Physical Decongestive Therapy

Management of lymphedema is referred to by a variety of terms, including complex lymphedema therapy, complete or complex decongestive physical therapy, or decongestive lymphatic therapy.

All of these regimens combine manual lymphatic drainage through light, superficial massage and compressive bandaging with active ROM, low-intensity resistance exercises, cardiopulmonary conditioning exercises, and good skin hygiene. Refer Chapter 9.10 for more details.

9.10 COMBINED PHYSICAL DECONGESTIVE THERAPY

Management of lymphedema is referred to by a variety of terms, including *complex lymphedema therapy, complete or complex decongestive physical therapy, or decongestive lymphatic therapy*. The components are detailed in the **Table 9.10.1.**

MANUAL LYMPHATIC DRAINAGE

Manual lymphatic drainage involves slow, very light repetitive stroking and circular massage movements done in a specific sequence with the involved extremity elevated whenever possible. Proximal congestion in the trunk, groin, buttock or axilla is cleared first to make room for fluid from the more distal areas. The direction of the massage is toward specific lymph nodes and usually involves *distal to proximal* stroking. Fluid in the involved extremity then is cleared, first in the proximal portion and then in the distal portion of the limb. Because manual lymphatic drainage is extremely labor and time intensive,

Table 9.10.1 Components of combined physical decongestive therapy

Components	*Subcomponents*
Elevation	
Lymphatic drainage	• Manual (by therapist) • Self (by care taker)
Compression	• Compressive bandage • Compressive garments • Pneumatic compression pump
Exercises	• Isotonic or pumping, or decongestive exercises • Isometric exercises • Deep breathing exercises • Relaxation exercises • Mild-resisted exercises • Flexibility exercises • Cardiovascular conditioning exercises
Skin hygiene	• Skin assessment • Skin care • Prevention of infections

methods of self-massage are taught to the patient as soon as possible in a treatment program.

ELEVATION

The involved limb is elevated during use of a sequential compression pump, while sleeping or resting, or even during sedentary activities. The compressive bandages or garment are worn during periods of elevation.

COMPRESSIVE BANDAGES, GARMENTS, OR PUMPS

Nonstretch, nonelastic bandages or low-stretch elastic bandages or garments are recommended because they provide relatively low compressive forces on the edematous extremity at rest. In addition, they provide a higher working pressure with active muscular contractions because of their less yielding nature than high-stretch bandages. Compression with custom-made elastic stocking (minimum pressure, 40 mm Hg) is an effective method, particularly in secondary lymphedema. Multilayer bandaging is another form of compression and has been shown to be effective in both upper and lower limb lymphedema. This form of compression consists of an inner layer of tubular stockinette followed by foam and padding to protect the joint flexures, and to even out the contours of the limb, so that the pressure is evenly distributed. Compression is provided by an outer layer of at least two short-stretch extensible bandages. Daily use of a sequential, pneumatic compression pump also may be advisable during the early stages treatment of substantial lymphedema. These pumps allow the development of high pressure up to 150 mm Hg. These pumps can reduce the limb girth measurements by 37–68.6%. Following treatment however, patients should continue to wear a compression stocking because there is a high risk of recurrence.

SKIN CARE AND HYGIENE

Lymphedema predisposes the patient to skin breakdown, infection and delayed wound healing. Meticulous attention to skin care and protection of the edematous limb are essential elements of self-management of lymphedema.

EXERCISE

The exercises employed in lymph drainage regimens cover a wide spectrum of therapeutic exercise interventions, specifically deep breathing, relaxation, flexibility, strengthening, cardiovascular conditioning exercises and decongestive exercises or pumping exercises (a sequence of lymphatic drainage exercises) as well.

Deep Breathing Exercises

Rationale

It assists in the movement of lymphatic fluid as the diaphragm descends during a deep inspiration and the abdominals contract during a controlled,

maximum expiration. Changes in intra-abdominal and intrathoracic pressures create a gentle, continual pumping action that moves fluids in the central lymphatic vessels, which run superiorly in the chest cavity and drain into the venous system in the neck.

Procedure

In deep breathing, subjects were asked to breathe in deeply and slowly through the nose and sigh out through the mouth.

Total Body Relaxation Exercises

Rationale

- Exercises are designed to help the patient relax
- Helps to clear the central channels and nodes
- Decreases muscle tension, which may be contributing to restricted mobility and lymph congestion.

Procedure

- Have the patient assume a comfortable supine position and begin deep breathing. Then, isometrically contract and relax the muscles of the lower trunk (abdominals and erector spinae) followed by the hips, lower legs, feet, and toes.
- Then contract and relax the muscles of the upper back, shoulders, upper arms, forearms, wrist, and fingers.
- Finally, contract and relax the muscles of the neck and face.
- Relax the whole body for at least a minute.
- Perform diaphragmatic breathing throughout the entire sequence. Avoid breath holding and the Valsalva maneuver.

Flexibility Exercises

Gentle, self-stretching exercises are used to minimize soft tissue and joint hypomobility, particularly in proximal areas of the body that may contribute to static postures and lymph congestion.

Strengthening and Muscular Endurance Exercises

Both isometric and dynamic exercises using self-resistance, elastic resistance, and weights or weight machines are appropriate if done against light resistance (initially, 1–2 lb) and by progressing resistance and repetitions gradually. Regardless of whether lymphedema has developed, it is important to monitor the circumferential size and the skin texture of the involved limb closely to determine whether an appropriate intensity of exercise has been established. Emphasis is placed on improving endurance and strength of central and peripheral muscle groups that enhance an erect posture and minimize fatigue in muscles that contribute to the efficiency of the lymphatic pump mechanism.

Cardiovascular Conditioning Exercises

Activities such as upper extremity ergometry, swimming, cycling, and walking increase circulation and stimulate lymphatic flow. About 30 minutes of aerobic endurance exercises complement lymph drainage exercises. Conditioning exercises are done at low intensity (at 40–50% of the target heart rate) when lymphedema is present and at higher intensities (up to an 80% level) when the lymphedema has been reduced and exercise is otherwise safe.

Lymphatic Drainage Exercises or Decongestive Exercises or Pumping Exercises

Introduction

Lymphatic drainage exercises move fluids through lymphatic channels. Active, repetitive ROM exercises are performed throughout each session. Static, dependent postures are avoided. Self-massage also is interspersed throughout the exercise sequence to further enhance drainage. These exercises also maintain mobility of the involved limbs.

Principles

The exercises follow a specific sequence to move lymph away from congested areas. It is similar to the sequence of massage applied during manual lymph drainage. In general, the exercises first focus on proximal areas of the body to clear central collecting vessels and then involve distal muscle groups to begin to move peripheral edema in a centripetal direction to the central lymph vessels. The affected upper or lower extremity or extremities are held in an elevated position during many of the exercises.

Rationale

- Contraction of muscle pumps fluid by direct compression of the collecting lymphatic vessels.
- Exercise reduces soft tissue and joint hypomobility that can contribute to static positioning and lead to lymphostasis.
- Exercise strengthens and prevents atrophy of muscles of the limbs, which improves the efficiency of the lymphatic pump.
- Exercise increases heart rate and arterial pulsations, which in turn contribute to lymph flow.
- Exercise should be sequenced to clear the central lymphatic reservoirs before the peripheral areas.
- Wearing compression bandages during exercises enhances lymph flow and protein reabsorption more efficiently than exercising without bandages.

Technique

Preparation for lymphatic drainage exercises

- Set aside approximately 20–30 minutes for each exercise session

- Perform exercises twice daily every day
- Keep needed equipment in hand, such as a foam roll, wedge or exercise wand.

During lymphatic drainage exercises

- Wear compression bandages or a customized compression garment
- Precede lymphatic drainage exercises with total body relaxation activities
- Follow a specified order of exercises
- Perform active, repetitive movements slowly, about 1–2 seconds per repetition
- Elevate the involved limb above the heart during distal pumping exercises
- Do not elevate the shoulder above heart level for proximal exercise as it may reduce venous return
- Combine deep breathing exercises with active movements of the head, neck, trunk and limbs
- Initially, perform a low number of repetitions. Increase repetitions gradually to avoid excessive fatigue
- Do not exercise to the point where the edematous limb aches
- Incorporate self-massage into the exercise sequence to further enhance lymph drainage
- Maintain good posture during exercises
- When strengthening exercises are added to the lymph drainage sequence, use light resistance and avoid excessive muscle fatigue.

After lymphatic drainage exercises

- If possible, rest with the involved extremity elevated for 30 minutes
- Set aside time several times per week for low-intensity aerobic exercise activities, such as walking or bicycling for 30 minutes
- Carefully check for signs of redness or increased swelling in the edematous limb, either of which could indicate that the level of exercise was excessive.

Exercises which promote lymphatic drainage for upper limb lymphedema

If the patient wears a compression garment, this should be worn as the exercises are completed. The exercises can be performed daily, but if the arm begins to ache during the exercise, it is important to reduce the activity. Arm elevation should not exceed shoulder height as this can obstruct venous return from the arm. The arm should be in a comfortable position, resting on a pillow that has been placed on the arm of the chair or the patient's knee **(Box 9.10.1)**.

- Slowly make a tight fist and then spread the fingers out wide. Repeat 10 times.
- With the palm of the hand facing down, slowly flex the wrist upwards and then back down again to point the fingers to the floor (repeat 10 times).

Box 9.10.1 Decongestive exercises for upper limb lymphedema
Finger bend and stretch
Wrist bending forwards and backwards
Wrist circling
Elbow bend fingers to shoulder stretch
Sitting arm place behind neck and behind waist

Box 9.10.2 Decongestive exercises for lower limb lymphedema
Toe movements
Ankle: Dorsiflexion and plantar flexion
Foot circling
Combined hip and knee—bending and straightening

- Wrist circling (repeat 10 times).
- With the arm straight and the palm facing upwards towards the ceiling, slowly bend the forearm up to touch the shoulder and then slowly straighten again (repeat 10 times).
- With the hand behind the body, reach up the back as far as possible without causing discomfort.
- With the head held still, lift the arm to place the hand behind the head.

Exercises which promote lymphatic drainage for lower limb lymphedema
The ideal position for leg elevation is with the body horizontal. The legs can be supported on a pillow, but it is important to ensure that they are not raised higher than the head, so that venous drainage from the head and neck is not reduced. Legs can be supported on a stool when sitting. If the patient wears a compression garment, this should be worn as the exercises are completed. Exercises should be combined with regular walking, swimming or cycling within the patient's capabilities. If the patient wears a compression garment, this should be worn during all exercises, except those in water. The exercises can be completed twice daily and should be **(Box 9.10.2)**.

- Bend and stretch the toes up and down for a slow count to 10.
- Flex the foot up and down at the ankle for a slow count to 10.
- Make slow, rhythmical circles with the foot for a slow count to 10.
- Bend and stretch the knee in a controlled, slow movement.

9.11 COMPRESSION THERAPY

INTRODUCTION

The terms *containment and compression* are often used incorrectly as synonyms, but in reality they indicate different concepts:

Containment: Passive action (static) of a rigid compression system (nonelastic or with hardly any elasticity), which is more or less inextensible and opposes systolic muscle dilatation, developing a raised working pressure (reinforcement effect on the venous pump); *the leg is contained at rest, but not compressed.*

Compression: Active action exercised at rest on a limb by the more or less elastic characteristics of the system with the development of high-resting pressures; *the leg is compressed even at rest.*

DEFINITION

Pressure exerted on a limb by materials of varying elasticity in order to prevent and treat disease of the venolymphatic system. The leg is compressed even at rest and during walking.

PROPERTIES OF THE BANDAGE

- *Extensibility*: It is the capacity of the bandage to stretch when pulled.
- *Elasticity*: It is the capacity of the bandage to return to its original state after being stretched.
- *Elastic power*: Determined by the strength required to obtain a specific extension. Power determines the amount of pressure a bandage will produce at a fixed extension.
- *Tension*: Initially produced by the force used to stretch the bandage; once the bandage is applied, tension maintain ability depends on the elastomeric properties (hysteresis—curve of extension and retraction) of the material used, which in turn depends on the type of fiber and fabrication.
- *Lock-out*: Defines the point when the physical structure of the bandage prevents further lengthening after a fixed extension has been reached.
- *Hysteresis*: Indicates the capacity of the elastic material to return to its original state after being stretched and is correlated to its extensibility, the viscoelastic properties of the fabric and the friction between the various spires.
- *Interface pressure*: The pressure exerted by the bandage that is measured in the interface between bandage and skin. The pressure exerted by the bandage depends on the fabric, the elastomeric characteristics of the bandage, on the tension applied, the number of overlapped layers and the anatomical characteristics (size and shape) of the limb being bandaged.
- *Laplace law*: The applied pressure will be directly proportional to the tension (T) of the elastic material and the number (n) of turns applied, while it will be inversely proportional to the radius (r) of the compressed surface and the width (h) of the bandage. According to the Laplace law, applying a bandage with the same tension, the pressure will decrease when the radius of the limb increases. Therefore, without changing the applied tension, we have a decreased pressure from the bottom towards the top due to the reverse conical conformation of the leg. Based on the radius of the anatomical structure we must be aware that the pressure exerted by the bandage is extremely strong on the Achilles tendon and on the tibial crest (very small radius), while it will be less at the back of the calves (wide radius) and negative at retromalleolar space. Therefore, it is advisable to increase the pressure in all those areas with a small radius and a high risk of too strong pressure, by protecting the acute angle with padding material (cotton, viscose or foam).

- *Stiffness*: It is the ability of the bandage to oppose to muscle expansion when contracted; it depends on what type of material the bandage is made of. When inelastic, the bandage is less distensible and has greater stiffness. The capacity to oppose to muscular volume increase when standing and walking can generate peaks of high pressure (60–80 mm Hg) able to occlude the venous system intermittently and therefore, restore a sort of valvular function. This in turn, reduces the reflux and the ambulatory venous hypertension.

CLASSIFICATION

- *Based on their elasticity*:
  - *Elastic bandage*: Exerts its pressure when stretched. On one hand the elastic bandage tends to return to its original length when extended (squeezing effect) on the other hand, the bandage gives way to the muscle expansion. The bandage may exert a sustained pressure not well tolerated or unbearable especially at rest.
  - *Inelastic bandage*: Exerts its effect by resisting the increase of muscle volume during standing and walking. It produces a significant increase in the standing and working pressure and relatively low pressure at rest, therefore well tolerated, and high during muscular exercise. *The inelastic non-stretchable bandage* (zinc oxide or velcro bandages) is included in this group.
- *Based on their extensibility*:
  - Inextensible (zinc oxide bandage)
  - Short stretch (extensibility <70%)
  - Medium stretch (extensibility >70% and <140%)
  - Long stretch (extensibility >140%).

  Inextensible or short-stretch bandages produce high '*working pressures*' during ambulation because of the containment effect on the contraction of the muscles of the leg, whereas the '*resting pressure*' is reduced compared with the working pressure. In contrast, elastic bandages classified as medium or long stretch compared with their initial dimensions are characterized by the fact that there is a gap between the resting and working pressures that they exert, which is inversely proportional to their elasticity.
- *Based on stiffness*:
  - High stiffness if the SSI is more than 10.
  - Low stiffness if the SSI is less than 10.

  Above terminology should be used only for bandages with a single component when elasticity can be determined in the laboratory.
- *Based on their function*:
  - Conforming stretch bandage (elastocrepe bandages)
  - Light support bandages (restricting limb movement and exerting an intermittent pressure)
  - Compression bandages that exert a mild (up to 20 mm Hg), medium (up to 30 mm Hg), Strong (up to 40 mm Hg) and very strong pressure (up to 60 mm Hg).

The pressure exerted by the bandage is calculated at rest on a fixed ankle circumference (23 cm) with a bandage overlapping by 50%.

- *Based on characteristics: Place classification*:
  More recently a new classification has been proposed based on the four main characteristics of compression bandages: *Pressure, layers, components and elasticity*.
  - *Based on pressure exerted at rest at the B1 region in the supine position*: Mild (<20 mm Hg), medium (20–40 mm Hg), strong (40–60 mm Hg) and very strong (>60 mm Hg).
  - *Based on layers*: Multi-layer or single layer; it is important to consider that the only single layer compression system is the elastic stocking. All bandages are multi-layer because, even when overlapped by 50%, they will however be made up of two layers.
  - *Based on components*: Single-component when made up of one material, multi-component when composed of several components.
  - *Based on elasticity*: Inelastic if composed of nonelastic, nonextensible or slightly extensible material and *elastic* if composed of elastic material.

Multi-layer/multi-component: The term 'multi-layer' has been used in an improper way; all bandages are multi-layer because there is always some kind of superimposition of a bandage. The term multi-component better defines bandages made up of several materials.

MECHANISM OF ACTION

- *Action on the superficial and deep venous system*:
  - Reduction in caliber and diameter of the veins, which further increases the rate of venous and lymphatic flow.
  - Healthy valve cusps getter better adapted.
  - Reduction of pathological reflux (incompetent perforators) thereby reduces back flow.
- *Action on the blood volume*:
  - Reduces blood volume in the lower limb which increases right ventricular filling.
- *Action on the tissues*:
  - External pressure exerted by the bandage increases tissue pressure, promoting the reabsorption of fluids back into the veins in accordance with Starling's law, thus producing a reduction of edema, together with the mechanisms referred to above.
- *Action on the microvascular tissue compartment*:
  - Compression therapy with an elastic stocking produces a reduction of venocapillary ectasia, interstitial edema and reactive thickening of the arteriolar basal membrane in patients affected by venous insufficiency.
  - Compression promotes detachment of leukocytes from the endothelium and prevents them from adhering further
  - Capillary filtration is also reduced and reabsorption is promoted due to the greater tissue pressure.

- The therapeutic elastic stocking is also able to reduce oxidative stress in healthy subjects obliged to stand for prolonged periods at work.

- *Action on the venous thrombus*:
  - Compression causes a reduction of venous capacity, which will automatically diminish venous output and speed up venous return with an increase in the flow rate, which must be regarded as the main cause of the effects of compression therapy in the prophylaxis of VTE.
  - The compression bandage increases the adhesion of any thrombus to the vein wall provided this does not extend beyond the upper border of the bandage.
  - *Compression therapy has an effective action on various coagulation factors*: A reinforcement of fibrinolysis of the vein wall, produces an increase in capillary perfusion and tissue oxygenation, with an increase in the release of nitric oxide. The treatment acts also by reducing blood viscosity, with suppression of procoagulant activity.

USES

Low levels of compression 10–30 mm Hg applied by stockings are effective in the management of telangiectases after sclerotherapy, varicose veins in pregnancy, the prevention of edema and deep vein thrombosis (DVT). High levels of compression produced by bandaging and strong compression stockings (30–40 mm Hg) are effective at healing leg ulcers and preventing progression of post-thrombotic syndrome as well as in the management of lymphedema.

BANDAGING TECHNIQUES

Elastic compression bandaging can be carried out using:

- Nonelastic and short-stretch bandages
- Medium- and long-stretch bandages.

This is a fundamental distinction from the clinical aspect, as, depending on which group of bandages is used the bandage can be kept in place for several days (with bandages of the first group) or the bandage has to be removed in the evening and replaced the following morning (bandages of the second group) because they are not tolerated at rest.

As a general rule, the bandages of the first group are used in more elderly patients, in more severe venous insufficiency complicated by trophic disorders, and in forms associated with obliterative peripheral arterial disease (mild or moderate).

Conversely, the bandages of the second type are used for less severe venous disease, to reduce edema and for compression of the superficial venous circulation. As regards application of the bandage, this can be done using different techniques, each with different indications. The bandage should be unrolled keeping the hand close to the surface of the skin so as to avoid pulling it upwards or downwards and to avoid differences in tension, which can cause areas of non-uniform pressure within the same region.

The patient's position, seated or lying does not influence the placement of the bandage apart from the greater or less comfort of application. The heel can be kept covered or uncovered according to whether it is desired to allow the patient to walk correctly (maintenance of proprioceptive sensation on contact with the ground during a step), or it may be necessary to reduce edema involving the retromalleolar fossae. In the case of bandaging as far as the thigh, the knee joint must be kept free, except in special cases.

The most common techniques of which numerous 'personal' versions are possible, are:

- Bandaging with regular turns
- Figure of eight bandaging
- Figure of eight bandaging fixed at the ankle
- Spontaneously unrolled bandage
- Multilayer bandaging.

Bandaging with Regular Turns

All of the techniques share the rule of unrolling the bandage from inside to outside that is, in the medial to lateral direction. In practice, proceed anticlockwise for bandaging the right limb and clockwise for the left limb. Bandaging is started at the base of the toes and after two to three turns around the foot, it moves to the ankle and proceeds proximally ensuring that the turns overlap by 50%, i.e. the bandage is extended covering half of the turn underneath. When it has reached below the knee, the turn is finished and if there are a few leftover centimeters of bandage, these are extended in a distal direction without exerting much traction.

Figure of Eight Bandaging

It starts at the base of the toes in regular turns and proceeds proximally, crossing the turns of the bandage from the dorsum of the foot or from the ankle to below the knee in a figure of eight; the maximum pressure is obtained at the crossing points of the turns; this type of bandage is more compressive than the preceding one because it provides greater overlapping of the turns of the bandage, and it is more stable over time. When placing this bandage, it is important to ensure that the crossing points of the bandage do not correspond to the tibial crest as they could cause skin injury.

Figure of Eight Bandaging Fixed at the Ankle

In this case, starting at the ankle, one turn of the bandage is placed and it is continued distally on the foot, which is covered as far as the base of the toes. It is then continued proximally again, back to the ankle; at this point, a figure of eight turn is made and it is continued upwards with regular turns. It is indicated especially in the treatment of venous ulcers exerting strong pressure just above the ankle around the medial malleolus, where at least five to six turns of the bandage overlap in this case.

Spontaneously Unrolled Bandage

This bandage is indicated especially for diseases of the calf as it exerts its maximum compression posteriorly; starting at the base of the toes, a few turns are made and the bandage moves to the lower border of the belly of the calf muscle; at this point, it is rolled around the calf to below the knee, then making a so-called fixation turn that is, a complete turn of the bandage below the knee. It is then brought downwards again in regular turns so as to cover the leg completely to above the ankle.

Multilayer Bandaging

This is a system produced by Smith and Nephew, comprising a kit of four bandages, produced for different ankle circumferences (18–25 cm and over 25 cm), which are applied in a precise sequence, each one with a different technique:

1. The first layer consists of a bandage of synthetic wadding (orthopedic wool), which is applied in regular turns overlapping 50% and covering the heel also.
2. The second layer consists of a non-stretch cotton crepe bandage, which fixes the first layer, and is applied in regular turns with overlapping of 50%.
3. The third layer consists of a light long-stretch bandage, which is placed using the figure of eight technique.
4. The fourth and last layer consists of a cohesive bandage, which is applied with regular turns with extension of 50% and overlapping of 50%, which will produce final compression at the ankle of 40–50 mm Hg.

ELASTIC STOCKINGS

The *therapeutic or medical compression stocking* (MCS) is a stocking made with materials and methods according to the standards, which guarantees a defined and graduated pressure along the limb between certain parameters, specified according to compression classes and available in different models and sizes.

Elastic stockings must also be classified because there is no accepted international standard for them either. Elastic stocking can be divided into three categories:

1. Preventive stockings
2. Antithromboembolic stockings
3. Therapeutic stockings.

Preventive or Support Stockings (Up to 18 mm Hg)

Compression is restricted at the ankle area and diminishes rapidly with the increase in circumference of the leg. They are distinguished based not on exerted pressure but on their thickness measured in Denier [measuring unit of thickness of thread: 1 Denier (DEN) = weight in grams of 9 km of thread].

The compression at the ankle is variable depending on the DEN:

- *40 DEN*: <10 mm Hg
- *70 DEN*: 10–14 mm Hg
- *140 DEN*: 15–18 mm Hg

They are only available in standard sizes, usually inexpensive but unable to exert sufficient compression in venous insufficiency.

Antithromboembolic Stockings

18 ± 3 mm Hg. The antithromboembolic stocking is a therapeutic, elastic stocking made in such a way to be tolerable at rest, exerting a pressure of 18 +/- mm Hg at the ankle.

Therapeutic Stockings

A stocking that does not meet the standards, in whole or even in part, but which can guarantee pressure in mm Hg at the ankle and/or other points of the lower limb, maintaining a certain reduction in pressure from below to above is defined as an *elastic support stocking.*

CONTRAINDICATION TO COMPRESSION THERAPY

Absolute Contraindications

- Obstructive arterial disease
- Major neuropathy, since the absence or great reduction in skin sensation increases the risk of damage produced by the pressure exerted by the bandage
- Extrinsic compression on veins (Baker's cyst in the popliteal fossa, lymphadenopathy)
- Decompensated heart failure (depletion of the peripheral venous pool towards the heart)
- Rheumatic fibromyalgia (marked intolerance of any pressure on the skin surface).

Relative Contraindications

- Osteoarticular disorders
- Acrocyanotic syndromes
- Raynaud's phenomenon.

9.12 PNEUMATIC COMPRESSION TREATMENT

DEFINITION

Pneumatic compression is a therapeutic technique used in medical devices that include an air pump and inflatable auxiliary sleeves, gloves or boots in a system designed to improve venous circulation in the limbs of patients who suffer edema or the risk of deep vein thrombosis (DVT) or pulmonary embolism (PE).

INDICATIONS

- Lymphedema of the limbs (primary or secondary)
- Post-traumatic edema
- Also used to aid the reduction of flexion deformities and in spasticity.

PARTS

The apparatus consists of a pneumatic unit with a series of the following control knobs:

- Which regulate the sequence of inflation/deflation
- The amount of pressure applied
- The time ratio for inflation/deflation

The pressure garment, either arm or leg consists of a double layered, sealed polyurethane sheath.

TYPES

The two types of compression available are intermittent and sequential:

1. In intermittent compression, the sleeve will be a continuous single compartment.
2. In sequential compression, the sleeve will consist of a number of compartments, which can be inflated or deflated in sequence depending on the settings.

PRINCIPLES OF WORKING

In intermittent compression method, the sleeve inflates and compresses at the chosen pressure (mm Hg) and then deflates. The whole limb is therefore compressed at the same time. In sequential compression method, the cells inflate sequentially—as one set deflates so another set inflates allowing for a ripple or sequential effect upon the tissues.

PROCEDURE

Mode of Treatment

The mode of treatment can be in-patient, outpatient or as home settings. An explanation should be given to the patient about the purpose, how it feels.

Assessment

Edema has to be measured by either volumetric displacement method, girth measurement or by figure of eight method. Also therapist should note about type (pitting or nonpitting), mobility, pain, stretchiness of skin and muscle strength.

Preparation of the Patient

The limb to be treated should be completely bared. Any rings, bracelets, watch, splint or bandages must be removed. The limb is then placed in a cotton gauze sleeve before being put into the compressive sleeve (this is necessary because the limb will sweat). The sleeve is then connected by plastic/rubber tubing to the machine, which is then switched on.

Position for Treatment

Because the treatment time is lengthy, it is essential that the patient is positioned in a comfortable manner. The limb must be treated in some elevation supported by pillows or a foam wedge placed on a plinth, chair arm or table. It pays to take time to achieve maximum comfort.

Duration of Treatment

Duration of treatment will depend upon the type of lymphedema, whether the patient is an in-or out-patient. On an average the in-patient would receive three sessions of 3 hours each for 5–14 days. This would almost certainly be used only for severe cases. An outpatient would receive daily treatment of 1–1½ hours possibly combining it with additional home treatment. If daily treatment is not possible, then twice weekly treatment is given but this would certainly have to be combined with home treatment. Home treatment can be given for a period of minimum 2 hours.

Timing of Compression

If intermittent compression is being given, the ratio of inflation/deflation would be around 3:1, given that the time sequence is 60 seconds, this would mean 45 seconds compression and 15 seconds deflation. These are only average figures and the therapist must work out her own ratios for the individual patient. The pressure (in mm Hg) should start low and increase to about 60 mm Hg. Again this will be individually tailored. While it is seldom necessary to go above 60 mm Hg, there are occasions when considerably higher pressures may be used. The time ratio in sequential compression varies according to the number of cell compartments and again will need to be tailored to the individual patient.

CONTRAINDICATIONS

- Cellulites of the limb
- Thrombophlebitis
- Atrial congestion
- Abdominal or thoracic venous occlusion
- Ischemia of the extremities
- Dermatitis
- Carcinoma affecting the limb under treatment
- Coexisting renal failure
- Patients with any degree of cardiac failure should not have two limbs treated simultaneously
- In any cases of doubt treatment should not be started until the patient has been assessed by a doctor. If, during treatment, the patient complains of pain or discomfort, the machine should be switched off immediately and the treatment stopped.

9.13 MANUAL LYMPHATIC DRAINAGE

INTRODUCTION

A series of pumping and stretching hand movements in a range of sequences are used to move the skin in specific directions based on the underlying structure and physiology of the lymphatic system. The movements influence the lymph vessels, which transport lymph toward the lymph nodes.

OBJECTIVE

To improve the functioning of the lymphatic system. Where lymphedema is present, manual lymphatic drainage (MLD) can be used as an aspect of treatment in combination with other approaches to management of swelling. Its aim is to increase activity within the normal lymphatics, so that lymph drains more effectively within them, bypassing the damaged or obstructed lymph channels and transferring lymph across 'watersheds' into adjacent areas of the body where it can drain more easily.

AIMS OF MANUAL LYMPHATIC DRAINAGE

- To increase activity in normal lymphatics and improve their function
- To open the flow of lymph fluid across watersheds
- To encourage lymph fluid to flow along alternative drainage routes
- To aid lymph drainage from congested areas.

PRINCIPLES OF MANUAL LYMPHATIC DRAINAGE

In order to move fluid from a swollen area toward functioning lymphatics, the movements start at the neck and unaffected lymphatics first, then move toward the area of the trunk closest to the affected area before finally being completed on the swollen area itself. Lymphatic drainage movements are based on the principle of motion. The initial lymphatics are opened up by a straight motion performed with light pressure on the skin in order to stretch it gently, followed by a lateral motion to stimulate the initial lymphatics to drain. The initial lymphatics then need to close to ensure that the lymph drains, so the pressure is released and the stretched skin springs back to its normal position.

TECHNIQUES

There are four different techniques used in MLD:

1. Stationary circles.
2. Rotary technique.
3. Pump technique.
4. Scoop technique.

Detailed explanations of the above techniques are beyond the scope of this book. The reader can refer the book named 'Lymphedema care by Mary Woods' (Clinical Nurse Specialist), Blackwell Publishers. The hand movements required for these different techniques are used according to the area of the body being treated and involve one or both hands in a slow rhythmical, repetitive manner.

For MLD to be completed correctly, a trained therapist will ensure that:

- Correct pressure is used on the skin. If the pressure used is too deep, the initial lymphatics may become damaged or collapsed so that drainage does not occur. If the pressure is too light, the fingers will slide over the skin.
- The movements are completed slowly, rhythmically and repetitively. Lymph can be compared with honey, which takes time to move. If the movements are completed too quickly, there is insufficient time for the initial lymphatics to open, drain and close again.
- An appropriate sequence and number of movements are used to direct the lymph fluid toward the appropriate lymph nodes. By clearing the way ahead, a path is created for the lymph fluid to flow along, so MLD movements are always started close to the node to encourage the lymph fluid to flow there.

CONTRAINDICATIONS

- *Acute infection or inflammation*: MLD may cause pain or discomfort if infection or inflammation is present in the area to be treated. In addition, toxic substances may be moved into lymph channels and spread throughout the body rather than being eliminated by the action of the local lymph nodes.
- *Active, untreated malignant disease*: Concern exists that MLD may encourage the transport of cancer cells in untreated active disease.
- *Recent thrombosis:* Until anticoagulation therapy is well established, there is a risk of clot dislodgment, if MLD is carried out in an area where a thrombosis is known to have developed.
- *Cardiac edema*: If the heart is not fully functioning, there is a risk of cardiac overload as a result of MLD treatment.

9.14 SIMPLE LYMPHATIC DRAINAGE

INTRODUCTION

Simple lymphatic drainage (SLD) is based on the principles of MLD. The hand movements are simplified and the technique modified to enable patients to incorporate an easier technique of lymphatic drainage into the daily management of their lymphedema.

AIM OF SIMPLE LYMPHATIC DRAINAGE

The main aim of SLD is to provide a modified technique of MLD that the patient can complete independently of a therapist.

Aim is to:

- To stimulate normal draining lymphatics
- To encourage lymph fluid to move away from congested areas to areas where it can drain away more freely
- To improve superficial lymph drainage.

PRINCIPLES OF SIMPLE LYMPHATIC DRAINAGE

Simple lymphatic drainage is completed in areas where lymph drainage is unaffected by any treatment intervention or altered lymph drainage pattern. This means that areas that are swollen are not included in the technique, because lymphatic massage in these areas requires the skill of a trained MLD therapist to ensure that lymph fluid is moved in the correct direction. By working on areas where lymph drainage is believed to be normal, SLD can encourage lymph fluid to move from the swollen area to areas where it can drain away more easily by clearing the route ahead. The technique can be likened to road works on a busy road. In order to bypass the road works, the traffic has to find alternative routes around the obstruction. These alternative, minor routes, already working hard to keep local traffic moving, can quickly become congested unless the traffic is allowed to flow freely. So, by ensuring that there are no obstacles in the way, the routes are able to cope with the increase in traffic and the congestion is eased.

TECHNIQUE

The hand movements used in SLD are simplified versions of those used in MLD, which follow a set sequence to encourage lymph fluid to drain.

TREATMENT WITH SIMPLE LYMPHATIC DRAINAGE

Simple lymphatic drainage is a patient-led aspect of lymphedema management taught by the therapist for the patient to use independently. It can be used once or twice daily depending upon the patient's wishes and completion of the full technique should take about 20 minutes. There are some important points that need to be considered prior to completion of the technique:

- The patient should choose a quiet time with no disturbances. Some patients find it useful to set aside some time as they go to bed to perform the technique as it can be relaxing. Others find it better to perform the technique before they get up in the morning. Whatever the preference, the technique should be completed fully each time.
- The pressure on the skin must be very light. It is important to move only the skin and not the tissues below. If the pressure is too heavy, the skin becomes reddened as blood flows to the surface and if the pressure is too light, the hand slides over the skin rather than remaining in contact with it.

- The technique must be completed in a slow, rhythmical manner. Lymph, like honey, takes time to move and need to be encouraged.
- Relaxed hands and flat fingers are necessary throughout the technique to ensure that the fingers do not dig into the skin and redden it.
- The use of creams, oil or powder should be avoided in areas where the technique is being carried out to ensure that good contact between the hands and the skin can be maintained.

CONTRAINDICATIONS

Simple lymphatic drainage is not advisable in the following circumstances which also apply to MLD:

- *Acute infection or inflammation*: SLD may cause pain or discomfort if infection or inflammation is present in the area to be treated. In addition, toxic substances may be moved into lymph channels and spread throughout the body rather than being eliminated by the action of the local lymph nodes.
- *Active, untreated malignant disease*: Concern exists that SLD may encourage the transport of cancer cells in untreated active disease.
- *Recent thrombosis*: Until anticoagulation therapy is well established, it is advisable to avoid SLD in an area where a thrombosis is known to have developed.
- *Cardiac edema*: If the heart is not fully functioning, there may be a risk of cardiac overload if SLD is regularly carried out.
- In addition, SLD should not be carried out if the patient has impaired upper limb function or is poorly motivated to complete the technique.

9.15 PREVENTION OF LYMPHEDEMA

PREVENTION OF FACTORS

- Avoid static, dependent positioning of the lower extremities, such as prolonged sitting or standing.
- Avoid sitting with legs crossed.
- When traveling long distances by car, stop periodically and walk around or support an involved upper extremity on the car's window ledge or seat back.
- Elevate involved limb(s) and perform repetitive pumping exercises frequently during the day.
- Avoid *vigorous,* repetitive activities with the involved limb.
- Avoid carrying heavy loads, such as a suitcase, a heavy backpack or shoulder bag.
- Avoid use of heavy weights when exercising.
- Wear compressive garments, while exercising.

- Avoid wearing clothing that restricts circulation, such as sleeves or socks with tight elastic bands.
- Do not wear tight jewelry such as rings or watches.
- Monitor diet to maintain an ideal weight and minimize sodium intake.
- Avoid hot environments.
- If possible, avoid having blood pressure taken on an involved upper extremity or injections in either an involved upper or lower extremity.

SKIN CARE

- Keep the skin clean and supple.
- Use moisturizers, but avoid perfumed lotions.
- Avoid infections; pay immediate attention to a skin abrasion or cut, an insect bite, a blister or a burn.
- Protect hands and feet; wear socks or hose, properly fitting shoes, rubber gloves, oven mitts, etc.
- Avoid contact with harsh detergents and chemicals.
- Use caution when cutting nails.
- Women need to use an electric razor when shaving legs or underarm area.
- Avoid hot baths, whirlpools and saunas that elevate the body's core temperature.

10 Surgeries in Vascular Diseases

10.1 ARTERIAL SURGERIES

INDICATIONS

- Arteriosclerosis
- Atherosclerosis
- Thrombosis
- Aneurysm
- Congenital abnormalities
- Trauma to the arteries
- Raynaud's disease
- Thromboangiitis obliterans (TAO)

TYPES OF SURGERIES

- Sympathectomy
- Direct suture
- Embolectomy
- Endarterectomy
- Arterial grafts
- Angioplasty
- When arterial surgery has failed or it is not possible to revascularize a limb, amputation is the only treatment.

Sympathectomy

Introduction

Sympathectomy involves removal of the sympathetic nerve supply to a part of the body causing selective vasodilatation (increased activity of sympathetic system causes vasoconstriction). A sympathectomy produces a local increase

in blood supply to skin, but its effect is not permanent (vasodilator drugs have a general effect.) The sympathetic system does not affect muscle arterial circulation and it is of no benefit in the treatment of intermittent claudication.

Types

- Cervical sympathectomy
- Lumbar sympathectomy
- Chemical sympathectomy.

Cervical sympathectomy

- *Purpose*: To improve circulation in hand.
- *Indication*: Raynaud's disease, hyperhidrosis, complex regional pain syndrome.
- *Contraindication*: Atherosclerosis.
- *Incision*: Above the clavicle or in the axilla.
- *Complications*:
  - Damage to cervical ganglion produces Horner's syndrome (ptosis and loss of sweating and papillary constriction)
  - Damage to pleura results in pneumothorax
  - Damage to brachial plexus.

Lumbar sympathectomy

- *Purpose*: To improve circulation in foot.
- *Indication*: Hyperhidrosis, Buerger's disease.
- *Contraindication*: Atherosclerosis.
- *Incision*: Through the back at the level of L1 to L5 to remove lumbar ganglion.
- *Complications*:
  - Postsympathectomy causalgia and neuralgia
  - Sexual dysfunction such as retrograde injection.

Chemical sympathectomy

Injection of chemical to cervical or lumbar sympathetic ganglion:

- *Purpose*: To improve temporary circulation in the hand or foot.
- *Chemical used*: Phenol or alcohol.
- Indications are same as that of cervical or lumbar sympathectomy.
- *Site*: Chemical is injected through a needle into a paravertebral space in the neck or lumbar region.
- *Contraindications*: No absolute contraindications. Relative contraindications are coagulopathy, thrombocytopenia, anticoagulant therapy, contralateral pneumothorax, contralateral vocal cord paralysis.
- *Complications*:
  - Phenol may spread into neighboring structures such as nerve to produce nerve injuries
  - Failure
  - Bleeding
  - Infection
  - Neuralgia.

Angioplasty

Angioplasty is a nonsurgical procedure that can widen a narrowed or blocked artery. A thin tube (catheter) is inserted into an artery in the groin or arm, and advanced to the area of narrowing. A tiny balloon on the tip of the catheter is then inflated to enlarge the narrowing in the artery. Sometimes the catheter technique is used to insert a stent (a cylindrical wire mesh tube) into the affected area of the artery to keep the artery open. In other cases, thrombolytic medications (medications that dissolve blood clots) may be delivered to the blocked area via a catheter. Angioplasty does not require general anesthesia and may be performed by an interventional radiologist, cardiologist or vascular surgeon. Usually, a local anesthetic at the area of catheter insertion and a mild sedative are given. Major complications of angioplasty are rare, but can occur. These include damage to the artery or blood clot formation, excessive bleeding from the catheter insertion site, and abrupt vessel closure (blockage of the treated area occurring within 24 hours of the procedure).

Angioplasty is indicated when a patient has claudication that limits his/her activities and does not respond to exercise, medications and lifestyle measures. Most doctors also recommend angioplasty when disease is very severe and there is a focal, localized narrowing that is accessible via catheter. If a patient is too ill to have surgery and has severe ischemia (decreased oxygen) that threatens loss of a limb, angioplasty may also be attempted. Some cases of peripheral artery disease may be more difficult to treat by angioplasty. For example, blockages in multiple small arteries of the legs or blockages in extremely small vessels may not be treatable by this method. Cryoplasty is a newer form of angioplasty in which freezing is used to open a narrowed artery. In this procedure, the balloon on the catheter is filled with liquid nitrous oxide, which freezes and destroys plaques within the artery.

Bypass Surgeries

Indications for surgical treatment of peripheral artery disease include lesions that, for anatomical reasons, may be difficult to treat by angioplasty. Examples include lesions covering long segments of a vessel, vessels with multiple narrowed areas or long areas of narrowing. Bypass surgery involves using a vein from his/her own body or a portion of synthetic vessel (known as grafts) to create a detour around the blockage. One end of the graft is sewn to the damaged artery above the blockage and the other end is sewn below the blocked area. Blood flow is then able to bypass the area of narrowing or blockage. Bypass surgery is a major surgical procedure requiring general anesthesia and a hospital stay. Surgeries are performed by a vascular surgeon.

Arterial Grafts

A graft may be used to replace an aneurysm or obstructed segment of an artery in larger arteries. A graft may also be used to construct a bypass round an obstructed artery. A bypass will be successful by provided if there is no significant arterial disease proximal or distal to the bypass.

For a general artery bypass the next suitable material is a saphenous vein, which has a thick muscular wall and can withstand arterial pressure. As the vein has values to aid venous blood flow the vein used for grafting must be reversed, so that arterial blood flow is not obstructed. The cephalic vein in the arm can also be used. A number of synthetic materials have been tried for grafting, but the most commonly used is Dacron grafts remain patent in aortic or iliac regions, and it is the material of choice because saphenous veins are not wide enough. It does, however, show a tendency to thrombose when used in the femoral or political arteries and is used only when a saphenous vein graft is not possible or has failed. An arterial bypass graft is described by the proximal and distal anastomoses. For example:

- A femoropopliteal graft is from the femoral artery to the popliteal artery.
- An aorta-bi femoral graft is from the aorta to both femoral arteries (a trouser graft).
- A femoro-femoral graft is from one femoral artery to the other (cross-over graft) is used when one iliac artery is healthy and the other diseased.
- An axillofemoral graft is from the axillary artery to the femoral artery and is used to revascularize the lower limb when the aorta is blocked.

Other Least Performed Surgeries

- *Direct suture*: This is the rejoining of an artery after a part is removed.
- *Embolectomy*: This is direct removal of an embolus through an opening in the artery. A Fogarty catheter with a balloon at the end is passed beyond the embolus. The balloon is then inflated and the catheter is withdrawn, removing the embolus and clearing the vessel.
- *Endarterectomy*: This is the removal of an atheromatous occlusion by stripping it out together with the tunica interna and part of the media.

10.2 VENOUS SURGERIES

SCLEROTHERAPY

Procedure (Fig. 10.2.1)

The surgeon finds the point along the leg where the varicose veins penetrate through the sheath of fibrous tissue that surrounds the leg muscles and drain into the deep veins. These penetrating, or communicating veins are called perforators. To find the sites of the perforating veins, the surgeon will make the patient stand up and then mark with a pen the general distribution of the varicose veins on the leg. Then patient will lie down on the examination couch and leg will be raised to empty the veins. The surgeon will then carefully feel along the leg to find the

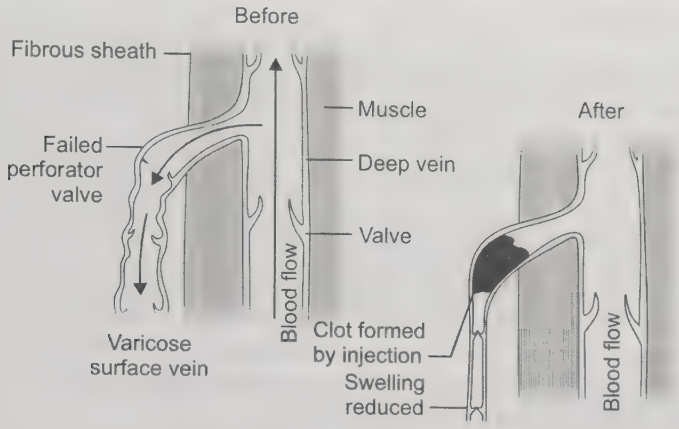


Fig. 10.2.1 Effect of injection treatment

little defects that mark the spots where the surface veins penetrate through the fibrous sheath of the leg into the deep veins. These points often correspond to particularly large bulges in the varicose veins themselves.

An injection of an irritant solution will be given into these sites, which will set up an intense, but controlled inflammation. The defence and healing mechanism will immediately start to close off the blow-out by the creation of scar tissue, thus stopping the high-pressure back-flow of blood through the varicose vein and so reduce the swelling. To make sure that the reaction does not spread to nearby healthy veins, the injection will be given into the vein, which is emptied of blood by raising your leg during the injection. The leg will then be firmly bandaged from the toes up to above the site of the injection and the bandage should be kept on and renewed for several weeks. To encourage the flow of blood from the leg and to prevent the slight risk of any extensive clotting of stagnant blood within the veins of the leg, patients will be instructed to walk briskly at least 3 miles (5 kilometer) every day, starting with the day of injection. It is also wise to keep leg raised in bed at night on a couple of pillows.

Indications

- Obliterating telangiectases
- Intracutaneous telangiectases
- Venous segments with reflux
- Correction of CVI
- Spider veins (< 1 mm)
- Venous lakes
- Varicose veins of 1–4 mm in diameter
- Bleeding varicosities
- Transfascial perforating veins
- Small cavernous hemangiomas (vascular or venous malformation).

Sclerosing Agents

- Hypertonic solution of sodium chloride (23.4%)
- Detergents such as sodium tetradecyl sulfate, polidocanol and sodium morrhuate
- Sodium iodide and chromated glycerin.

Action of Sclerosing Agents

It probably combines with the protein of the cells lining the wall of the vein, damaging them and causing a brisk local inflammation. It is so highly effective that only a small amount (0.5–1 mm) is necessary at any point of injection. In general, for smaller diameter veins, the sclerosing agent need to be diluted to avoid tissue inflammation and tissue necrosis.

Contraindications

- Veins that extend up to the thigh or groin
- Sites which cannot be compressed by bandaging after injection
- Very obese patients (very difficult to access perforator vein and difficult to bandage).

Complications

- Hyperpigmentation of the surrounding skin from hemosiderin degradation
- Thrombosis
- Pain
- Inflammation
- Recurrence.

SURGICAL CORRECTION OF VARICOSE VEINS

Surgeries are performed under general anesthesia or local anesthesia. Three procedures:

Stripping of Veins

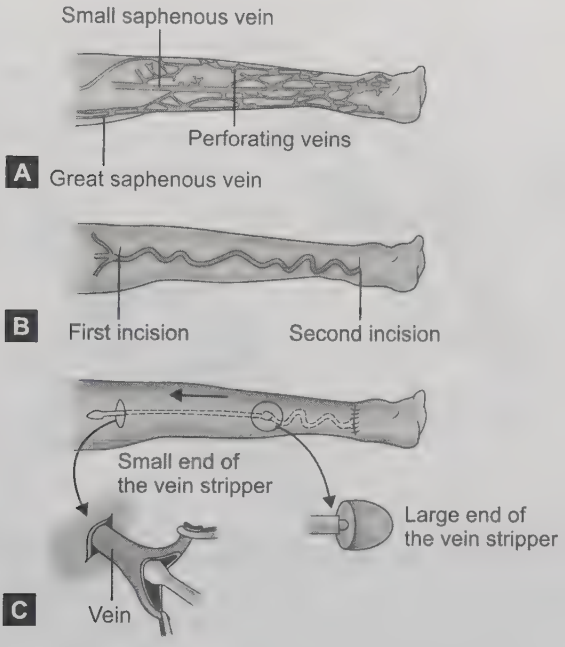
Here, the surgeon passes a wire or a length of plastic along the length of the varicose surface vein and uses this to pull out a whole segment of vein with the help of stripper. The **Table 10.2.1** compares sclerotherapy with stripping of veins **(Figs 10.2.2A to C)**, etc.

Phlebectomy

The varicose vein is mapped and marked on the skin while the patient is standing. The patient is then placed in a supine position, and a series of perpendicular stab incisions are made over the vein several centimeters apart. The saphenous vein is identified in the groin, brought to the surface via a small incision and ligated. The vein is hooked and brought to the surface at the next incision site.

Table 10.2.1 Comparison between sclerotherapy and stripping

	Sclerotherapy	*Stripping*
Application	For small vein	For large vein
Advantages	• No period of hospitalization • No general anesthesia required	• Short period of bandaging after injection • High success rate
Disadvantages	• Long period of bandaging after injection • Low success rate	• Short period of hospitalization • No general anesthesia required



Figs 10.2.2A to C Stripping procedure. (A) Superficial and perforator veins in the leg; (B) An incision is made at both ends of the section of vein through skin being removed; (C) A flexible wire is inserted through one end and extended to the other. The wire is then withdrawn, pulling the vein out with it

It is then pulled and dissected proximally and distally at each incision site to release it from the surrounding tissues and to sever any connections to tributary or deeper perforating veins. This process is repeated distally.

Separation and Tie off Varicose Vein

This operation involves the separation and tying off of the varicose vein. The surgeon makes a short incision over the region of the junction between the long saphenous vein and the femoral vein. The little branches of the saphenous are cut and ties them off with fine ligatures. Then divides and ties off the main

trunk of the saphenous vein. This effectively seals off this incompetent junction between the surface and deep vein systems, thus stopping the flow of blood into the varicose vein below the junction and reducing the swelling of the vein. Then, surgeon will insert the stripper (usually a length of plastic or metal) along the length of the vein until it passes below the level of the knee joint. Another small incision is placed at this site and the lower end of the stripper is brought out through the skin. The top end of the vein is then tied to the stripper and a small head may be attached to it to ensure that the vein does not come off the stripper. The stripper is then pulled through completely removing the dilated vein between the two incisions **(Figs 10.2.3 and 10.2.4)**.

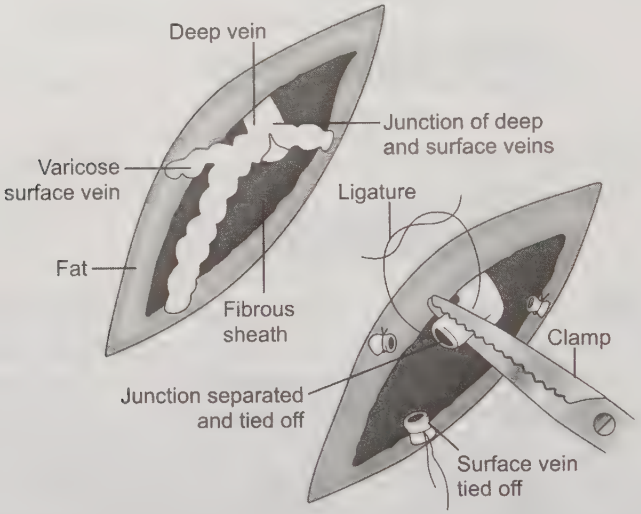


Fig. 10.2.3 Tie off operation

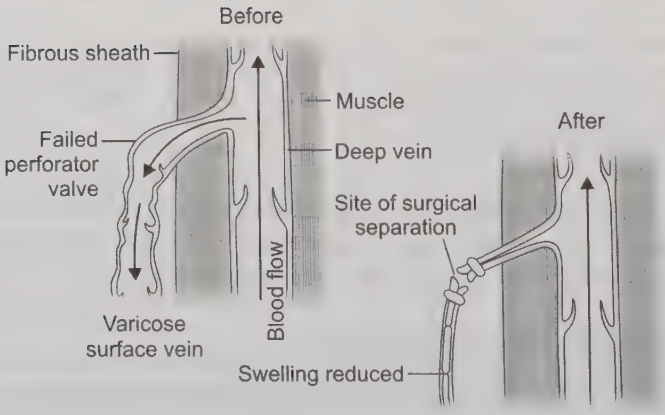


Fig. 10.2.4 Effect of tie off operation

Indications

- Recurrence after sclerotherapy
- Failure of sclerotherapy
- Patients who does not respond to sclerotherapy
- Patient who does not like injections
- Patients whose varicose veins are too extensive for injections to be effective
- Patients with large varicosities extend up to the thigh, often to the groin
- Obese patients with varicosities.

Complications

- Pain
- Recurrence, if vein is not removed
- Bruising beneath the skin
- Hematoma, if patients who are overweight
- Wound infection
- Thrombosis
- *Damage to superficial nerves*: Saphenous nerve in case of great saphenous vein, sural nerve in case of short saphenous vein. This causes causalgia to the patient.

10.3 LYMPHATIC SURGERIES

INTRODUCTION

Aim of the lymphatic surgeries is to produce a functional limb with the optimum cosmetic result that can be achieved. Any improvement gained from surgery will require long-term maintenance with compression hosiery.

INDICATIONS

- To reduce the weight of the bulky part
- To improve the shape of the limb, making it possible for patients with large limbs to wear normal clothes again
- To reduce the incidence of repeated infections
- To improve the texture of the skin.

Surgical procedures used in lymphedema management can be divided into three areas:

1. Reduction procedures
2. Lymphatic bypass procedures
3. Liposuction.

Reduction Procedures

These procedures remove excess, edematous, subcutaneous tissue.

- *Homan's reducing operation*: Edematous skin and subcutaneous tissues are excised and residual flaps are sutured together.
- Sistrunk's reducing operation
- *Charles' reducing operation*: Defects are covered with split thickness graft
- Thompson's buried dermis flap operation.
- Kontoleon's operation.

Indications

- Most frequently used to treat lower limb lymphedema
- The procedures have also been adapted to treat swelling in the eyelids and genitalia.

LYMPHATIC BYPASS PROCEDURES

The restoration of lymphatic function has been attempted using a variety of procedures. Obstructed lymphatic vessels have been anastomosed to vessels in the venous system in an attempt to drain the swollen limb and normal healthy lymph vessels have been transplanted into areas where lymphedema exists in order to connect poorly functioning lymph vessels with normal ones. Microsurgical techniques are required for these procedures and results appear to be more successful if patients are carefully selected and in the younger age group.

LIPOSUCTION

The removal of subcutaneous fat from a large limb by suction has been used as a method of reducing limb size when standard lymphedema management fails. The procedure involves several incisions along the length of the limb through which a cannula is inserted. The subcutaneous fat is sucked out through the cannula under vacuum. Liposuction does not correct lymph drainage and results are only maintained through the continued use of high compression garments.

COMPLICATIONS

- Reduction procedures causes destruction to the still functioning lymph vessels in the limb
- Scarring.

10.4 COMPLICATIONS OF SURGERIES

These can be general as in any major surgery or local around the site of operation.

GENERAL COMPLICATIONS

Circulatory complications are more likely to arise in arterial surgery than in other forms of major surgery because of the nature of the disease, the site of surgery and the age of patient:

- *Coronary thrombosis or cerebrovasular accident*: If a thrombosis dislodges it may block one of the blood vessels supplying the heart or the brain.
- *Deep vein thrombosis*: A thrombosis may from in the deep veins particularly of the calf due to sluggish venous circulation and increased release of thromboplastin at the operation site, which may be near veins.
- *Respiratory complications*: Secretions may accumulate because the patients are often smokers. The operation may take a number of hours and the patients may have limited respiratory function, which could result in complications.
- *Pulmonary embolism*: A pulmonary embolus may result from deep vein thrombosis.

LOCAL COMPLICATIONS

- *Infection*: This is more likely to arise with a Dacron graft because it has no natural antibodies. Infection leads to breakdown of the anastomosis and leakage of blood into the neighboring tissues. Signs of graft breakdown:
  - Excessive loss of blood from redivac drain.
  - Swelling at operation site.
- *Hemorrhage*: This results from immediate leakage at the suture line.
- *Graft obstruction*: This may arise due to thrombosis formation from slowing of the blood flow or irritation of the arterial wall.
  Signs of graft obstruction: The following signs may be present distal to the operation site:
  - Diminished or lost pulses
  - Limb feels and/or appears cold
  - Pain and numbness
  - Color becomes mottled, pale
- *Peripheral neuropathy*: Peripheral nerves may be damaged resulting in weakness of the muscles supplied by the damaged nerve.

11 Physiotherapy Management for Arterial Surgeries

PREOPERATIVE PHYSIOTHERAPY

All patients except those for acute emergency surgery, bleeding aneurysms or sudden total blockage of a main artery will be admitted two-three days before surgery. Investigations are carried out on the arterial and respiratory systems. Drug therapy is reviewed, e.g. antibiotics and anticoagulants (heparin or warfarin).

Aims

- Explanation to the patient
- To gain patient confidence
- To maintain adequate ventilation
- To teach postoperative circulatory exercises.

Respiratory Care

- The patient is strongly advised to give up or at least reduce smoking
- Expansion breathing exercises and breathing control technique are taught
- Effective coughing or huffing is practiced with the patient shown how to support the wound particularly if there is an abdominal incision
- More vigorous treatment may be required if lung infection is present.

Circulatory Care

The importance of foot exercise must be explained and the patient practices them except where there is evidence of gangrene in the foot. General deep breathing is also taught so that the diaphragmatic movement will aid venous return.

POSTOPERATIVE PHYSIOTHERAPY

The patient may spend 24 hours in the intensive care unit (ICU), particularly for aortic grafts. The patient wears antiembolic stockings and lies supine with a bed cradle to allow the feet to move freely. This also enables observation of skin color

and arterial pulses. The lower limbs (LLs) should be flat and not supported on a pillow. A redivac drain remains *in situ* until drainage is minimal.

Physiotherapy

- *Breathing exercises*: These are given when the patient recovers consciousness to the basal areas of the lungs combined with huffing to encourage expectoration with minimum effort by the patient. Thoracic incisions must be supported by the patient with the help of the therapist.
- *Foot exercises*: These are given immediately to prevent deep vein thrombosis (DVT). Active toe and ankle movements of both legs, particularly full-range dorsi- and plantar-flexion are encouraged with all levels of graft. The patient must do the exercises vigorously every hour. The therapist should note the skin temperature and color of the LLs for signs of postoperative complications. The temperature chart should be read daily, because a raised temperature is indicative of infection in the chest, urine or wound. Pain and swelling in the calf is indicative of DVT.

Bloodstained sputum together with chest pain should be reported in case a pulmonary embolus is developing. All arterial surgery has the same basic physiotherapy, but following an embolectomy or endarterectomy the patient can move all joints of the LLs and is discharged after a few days. Following arterial grafts no undue strain must be put on the graft and kinking must be avoided. The joints over which the graft passes must not be bent, e.g. avoid knee movements in femoral popliteal grafts and hip movements in iliofemoral grafts.

The patient begins walking in 2–3 days following a femoral popliteal graft and knee movements are gradually encouraged. With more proximal grafts the patient must be encouraged to stand straight. The walking pattern is corrected daily and the distance is gradually increased before discharge in 7–10 days. The patient should walk up and down stairs before discharge.

In surgery where an arm vein has been used for grafting all movement of the upper limb (UL) joints should be encouraged postoperatively. Patients do not normally require physiotherapy after discharge from hospital.

Advice to Patients

- Avoid restrictive clothing, which may interfere with the circulation, e.g. tight belts or bands
- Stop smoking or reduce it as much as possible
- Avoid positions which cause pressure on the graft, e.g. knee flexion beyond 90°, sitting back on heels or crossing one leg over the other in femoral popliteal grafts
- Avoid prolonged standing (but if this is unavoidable then practice marking time). However, a daily walk should be encouraged
- Avoid exposure to excessive cold and take care with application of heat, e.g. hot-water bottles
- A gradual return to normal function and increasing the amount of physical activity is to be encouraged.

12 Physiotherapy after Venous Surgeries

PHYSIOTHERAPY AFTER SCLEROTHERAPY

Injection of sclerosant solution into the vein is followed by firm bandaging of the legs for 6 weeks. The sclerosant produces inflammation in the vein causing its lumen to be obliterated so that no blood can pass through.

Physiotherapy

Patient is encouraged to practice foot and ankle exercises in elevation and instructed to walk 1–2 miles a day, in support stockings if necessary to keep blood flowing through the deep veins. The correct pattern of walking must be emphasized.

PHYSIOTHERAPY AFTER STRIPPING OF VEINS

Postoperatively the legs are bandaged and elevated to promote blood flow in the deep veins. When resting the knee should be straight, but the patient should practice leg exercises hourly as soon as possible. For example, foot and ankle pumping exercises, hip and knee flexion and extension exercises, quadriceps and gluteal contractions.

First Postoperative Day

The patient is helped out of the bed and walking is commenced with the legs well bandaged. The patient is encouraged to move the ankle and knee joints together with the correct push off with the even timing and stride length patterns.

Second Postoperative Day

The distance walked is progressed (avoid standing still) and a flight of stairs attempted. The patient is discharged within 48 hours with clear instructions on the wearing of support stockings for several weeks and the continuation of the exercises at home. Physiotherapy may also be given for any venous ulcers or edema if present.

13 Wound Management

13.1 WOUND CLASSIFICATIONS AND ETIOLOGY

DEFINITION

Wound can be defined as break in any body tissue due to external action (including surgery). It may be closed (blunt trauma) or open (penetrating trauma). The wound is generally applied to superficial forms of tissue damage whereas injury is due to damage to deeper structures.

CLASSIFICATION

Acute Wound

Open Wounds

Open wounds can be classified according to the object that caused the wound.

The types of open wound are:

- Incisions or incised wounds caused by a clean, sharp-edged object such as a knife, razor or glass splinter.
- Lacerations are irregular tear-like wounds caused by some blunt trauma. Lacerations and incisions may appear linear (regular) or stellate (irregular).
- Abrasions (grazes) are superficial wounds in which the topmost layer of the skin (the epidermis) is scraped off. Abrasions are often caused by a sliding fall onto a rough surface.
- Puncture wounds are caused by an object puncturing the skin, such as a nail or needle.
- Penetration wounds are caused by an object such as a knife entering and coming out from the skin.
- Gunshot wounds caused by a bullet or similar projectile driving into or through the body. There may be two wounds, one at the site of entry and one at the site of exit, generally referred to as a through-and-through.

Closed Wounds

Closed wounds have fewer categories, but are just as dangerous as open wounds.

The types of closed wounds are:

- Contusions, more commonly known as bruises, caused by a blunt force trauma that damage tissue under the skin.
- Hematomas, also called blood tumor, caused by damage to a blood vessel that in turn causes blood to collect under the skin.
- Crush injury caused by a great or extreme amount of force applied over a long period of time.

Chronic Wound/Ulcer

Chronic ulcers are the wounds, which fail to heal. Ulcer is discussed in the Chapter 14.1–14.5.

The most useful classification of the wounds from a practical point of view is that of *Rank and Wakefield* into tidy and untidy wounds.

Tidy Wounds

Tidy wounds are influenced by sharp instruments and contain no devitalized tissue. Examples are surgical incisions, cuts from glass and knife wounds. Tendons, arteries and nerves will commonly be injured in tidy wounds. Fractures are uncommon in tidy wounds.

Untidy Wounds

Untidy wounds result from crushing, tearing, avulsion, vascular injury or burns, and contain devitalized tissue. Tendons, arteries and nerves may be exposed, injured and might be injured in continuity, but will usually not be divided. Fractures are common and may be multifragmentary.

ETIOLOGY

Acute wounds: It can be due to accidental or as a result of planned surgical intervention.

Chronic wounds: It can be due to the following reasons:

- *Arterial insufficiency*: Prolonged pressure on one part of the foot causes ischemic damage to the tissues and if the circulation is inadequate then the tissue cannot repair by themselves, which results in the ulcer formation.
- *Venous insufficiency*: Patients with varicose veins or nonfunctional venous valves after DVT develop ambulatory venous hypertension, i.e. distal venous pressure remains elevated despite ambulation. This constant venous hypertension seems to cause white cell and fibrin build-up, which impairs capillary blood flow or traps growth factors.

- *Lymphedema*: Although not typically a cause of ulceration, extremity ulcers may fail to heal, because of untreated lymphedema.
- *Neuropathy*: Sensory neuropathy involving the feet may lead to unrecognized episodes of trauma caused by ill-fitting shoes. This is compounded by motor neuropathy causing intrinsic muscle weakness and spaying of the foot on weight bearing. The result is a convex foot with a rocker-bottom appearance. Multiple fractures go unnoticed, until bone and joint deformities become marked. This is termed a Charcot foot (i.e. neuropathic osteoarthropathy) and is observed most commonly in people with diabetes mellitus (DM).
- *Pressure (decubitus) ulcers*: Pressure (decubitus) ulcers occur, because of prolonged ischemia-producing external pressure, usually to a soft tissue region overlying a bony prominence. Tissue ischemia results when external pressure exceeds capillary closing pressure (i.e. 25–32 mm Hg in healthy individuals), the minimum pressure that causes collapse of the capillary when applied to a capillary bed. Shearing forces, exposure to constant moisture and heat build-up are also major contributing factors.
- *Neoplasm*: Neoplasm strongly suggests malignancy in any chronic non-healing wound, particularly if the wound appears to have occurred spontaneously.
- *Radiation damage*: The adverse effects of prolonged or excessive electromagnetic radiation vary with the wavelength. Gamma radiation and X-ray exposure cause a zone of stasis in which local blood supply is impaired by coagulative necrosis due to thrombotic occlusion of smaller arteries. The long-term result is inhibition of regeneration of skin cells from dividing basal cells. This may cause recalcitrant painful skin ulcers. The surrounding skin is atrophic with atrophy of hair follicles and a paucity subcutaneous fat.

  Ultraviolet radiation exposure, particularly ultraviolet B, causes sunburn initially and subsequently conveys a continuing risk of skin malignancy [e.g. basal cell carcinoma (BCC), squamous cell carcinoma (SCC) and melanoma].

13.2 WOUND HEALING

DEFINITION

Wound healing is defined as an intricate process in which the skin repairs itself after injury. In normal skin, the epidermis (outermost layer) and dermis (inner or deeper layer) exists in steady-state equilibrium, forming a protective barrier against the external environment. Once the protective barrier is broken, the normal (physiologic) process of wound healing is immediately set in motion.

PHYSIOLOGY OF WOUND HEALING

The classic model of overlapping phases of wound healing describes a process that is continuous and its phases are not distinct. The phases are divided into the following:

- Early cellular phase
- Inflammatory phase
- Proliferative phase
- Maturation/remodeling phase.

Early Phase and Cellular Phase

- The early phase, which begins immediately following skin injury, involves cascading molecular and cellular events leading to hemostasis and formation of an early, makeshift extracellular matrix providing structural support for cellular attachment and subsequent cellular proliferation.
- The cellular phase follows the early phase and involves several types of cells working together to mount an inflammatory response, synthesize granulation tissue, and restore the epithelial layer.

Subdivisions of the cellular phase are:

- Macrophages and inflammatory components (within 1–2 days)
- *Epithelial-mesenchymal interaction*: Re-epithelialization (phenotype change within hours), migration begins on day 1 or 2.
- *Fibroblasts and myofibroblasts*: Progressive alignment, collagen production, and matrix contraction (between day 4 and day 14).
- Endothelial cells and angiogenesis (begins on day 4)
- *Dermal matrix*: Elements of fabrication (begins on day 4, lasting 2 weeks) and alteration/remodeling (begins after week 2, lasting weeks to months—depending on wound size).

Inflammatory Phase

A scab covering a healing wound just before the inflammatory phase is initiated, the clotting cascade takes place in order to obtain hemostasis or stop blood loss by way of a fibrin clot. Thereafter, various soluble factors (including chemokines and cytokines) are released to attract cells that phagocytosed debris, bacteria and damaged tissue, in addition to releasing signaling molecules that initiate the proliferative phase of wound healing.

Clotting Cascade

When tissue is first wounded, blood comes in contact with collagen, triggering blood platelets to begin secreting inflammatory factors. Platelets also express glycoproteins on their cell membranes that allow them to stick to one another and to aggregate, forming a mass. Fibrin and fibronectin cross-link together and form a plug that traps proteins and their particles, and prevents further blood loss. This fibrin-fibronectin plug is also the main structural support for the wound until

collagen is deposited. Migratory cells use this plug as a matrix to crawl across, and platelets adhere to it and secrete factors. The clot is eventually lyzed and replaced with granulation tissue, and then later with collagen.

Platelets, the cells present in the highest numbers shortly after a wound occurs, release a number of things into the blood, including proteins and cytokines, including growth factors. Growth factors stimulate cells to speed their rate of division. Platelets also release other proinflammatory factors such as serotonin, bradykinin, prostaglandins, prostacyclin, thromboxane, and histamine, which serve a number of purposes, including to increase cell proliferation and migration to the area and to cause blood vessels to become dilated and porous.

Vasoconstriction and Vasodilatation

Immediately after a blood vessel is breached, ruptured cell membranes release inflammatory factors such as thromboxane and prostaglandins that cause the vessel to spasm to prevent blood loss and to collect inflammatory cells, and factors in the area. This vasoconstriction lasts 5–10 minutes and is followed by vasodilation, a widening of blood vessels, which peaks at about 20 minutes postwounding. Vasodilation is the result of factors released by platelets and other cells. The main factor involved in causing vasodilation is histamine. Histamine also causes blood vessels to become porous, allowing the tissue to become edematous because proteins from the blood stream leak into the extravascular space, which increases its osmolar load and draws water into the area. Increased porosity of blood vessels also facilitates the entry of inflammatory cells such as leukocytes into the wound site from the bloodstream.

Decline of Inflammatory Phase

As inflammation dies down, fewer inflammatory factors are secreted, existing ones are broken down, and numbers of neutrophils and macrophages are reduced at the wound site. These changes indicate that the inflammatory phase is ending and the proliferative phase is underway. *In vitro* evidence, obtained using the dermal equivalent model, suggests that the presence of macrophages actually delays wound contraction and thus the disappearance of macrophages from the wound may be essential for subsequent phases to occur. Because inflammation plays roles in fighting infection, clearing debris and inducing the proliferation phase, it is a necessary part of healing. However, inflammation can lead to tissue damage if it lasts too long. Thus the reduction of inflammation is frequently a goal in therapeutic settings. Inflammation lasts as long as there is debris in the wound. Thus the presence of dirt or other objects can extend the inflammatory phase for too long, leading to a chronic wound.

Proliferative Phase

About 2 o. 3 days after the wound occurs, fibroblasts begin to enter the wound site, marking the onset of the proliferative phase even before the inflammatory phase has ended. As in the other phases of wound healing, steps in the proliferative phase do not occur in a series, but rather partially overlap in time.

Angiogenesis

Angiogenesis also called neovascularization, the process of angiogenesis occurs concurrently with fibroblast proliferation, when endothelial cells migrate to the area of the wound. Because the activity of fibroblasts and epithelial cells requires oxygen and nutrients, angiogenesis is imperative for other stages in wound healing, such as epidermal and fibroblast migration. The tissue in which angiogenesis has occurred, typically looks red (is erythematous) because of the presence of capillaries. Stem cells of endothelial cells, originating from parts of uninjured blood vessels, develop pseudopodia and push through the extracellular matrix (ECM), into the wound site to establish new blood vessels. Endothelial cells are attracted to the wound area by fibronectin found on the fibrin scab and chemotactically by angiogenic factors released by other cells, e.g. from macrophages and platelets when in a low-oxygen environment. Endothelial growth and proliferation is also directly stimulated by hypoxia, and presence of lactic acid in the wound. To migrate, endothelial cells need collagenase and plasminogen activator to degrade the clot and part of the ECM. Zinc-dependent metalloproteinase digest basement membrane and ECM to allow cell migration, proliferation and angiogenesis. When macrophages and other growth factor-producing cells are no longer in a hypoxic, lactic acid-filled environment, they stop producing angiogenic factors. Thus, when tissue is adequately perfused, migration and proliferation of endothelial cells is reduced. Eventually blood vessels that are no longer needed die by apoptosis.

Fibroplasia and Granulation Tissue Formation

Simultaneously with angiogenesis, fibroblasts begin accumulating in the wound site. In the first 2 or 3 days after injury, fibroblasts mainly migrate and proliferate, while later, they are the main cells that lay down the collagen matrix in the wound site. Initially fibroblasts utilize the fibrin cross-linking fibers (well-formed by the end of the inflammatory phase) to migrate across the wound, subsequently adhering to fibronectin. Fibroblasts then deposit ground substance into the wound bed, and later collagen, which they can adhere to for migration. Granulation tissue functions as rudimentary tissue, and begins to appear in the wound already during the inflammatory phase; 2–5 days post wounding, and continues growing until the wound bed is covered. Granulation tissue consists of new blood vessels, fibroblasts, inflammatory cells, endothelial cells, myofibroblasts, and the components of a new, provisional ECM. The provisional ECM is different in composition from the ECM in normal tissue and its components originate from fibroblasts. Such components include fibronectin, collagen, glycosaminoglycan, elastin, glycoproteins and proteoglycans. Its main components are fibronectin and hyaluronan, which create a much hydrated matrix and facilitate cell migration. Later this provisional matrix is replaced with an ECM that more closely resembles that found in non-injured tissue.

Collagen deposition: One of fibroblasts most important duties is the production of collagen. Collagen deposition is important because it increases the strength

of the wound; before it is laid down, the only thing holding the wound closed is the fibrin-fibronectin clot, which does not provide much resistance to traumatic injury. Also, cells involved in inflammation, angiogenesis, and connective tissue construction attach to, grow and differentiate on the collagen matrix laid down by fibroblasts. Type III collagen and fibronectin are generally beginning to be produced in appreciable amounts at somewhere between approximately 10 hours and 3 days depending mainly on wound size. Their deposition peaks at 1–3 weeks. They are the predominating tensile substances until the later phase of maturation, in which they are replaced by the stronger type I collagen. Even as fibroblasts are producing new collagen, collagenase and other factors degrade it. Shortly after wounding, synthesis exceeds degradation, so collagen levels in the wound rise, but later production and degradation become equal so there is no net collagen gain. This homeostasis signals the onset of the later maturation phase. Granulation gradually ceases and fibroblasts decrease in number in the wound once their work is done. At the end of the granulation phase, fibroblasts begin to commit apoptosis, converting granulation tissue from an environment rich in cells to one that consists mainly of collagen.

Epithelialization

The formation of granulation tissue into an open wound allows the re-epithelialization phase to take place, as epithelial cells migrate across the new tissue to form a barrier between the wound and the environment. Basal keratinocytes from the wound edges and dermal appendages such as hair follicles, sweat glands and sebaceous (oil) glands are the main cells responsible for the epithelialization phase of wound healing. They advance in a sheet across the wound site and proliferate at its edges, ceasing movement when they meet in the middle. Though in healing that results in a scar, sweat glands and hair follicles do not form. Keratinocytes migrate without first proliferating. Migration can begin as early as a few hours after wounding. However, epithelial cells require viable tissue to migrate across, so if the wound is deep it must first be filled with granulation tissue. Thus the time of onset of migration is variable and may occur about 1 day after wounding. Cells on the wound margins proliferate on the 2nd and 3rd day post-wounding in order to provide more cells for migration. Epithelial cells climb over one another in order to migrate. This growing sheet of epithelial cells is often called the epithelial tongue. The first cells to attach to the basement membrane form the stratum basal. These basal cells continue to migrate across the wound bed, and epithelial cells above them slide along as well. The more quickly this migration occurs, the less of a scar there will be. Fibrin, collagen, and fibronectin in the ECM may further signal cells to divide and migrate. Epithelial cells have the ability to phagocytize debris such as dead tissue and bacterial matter that would otherwise obstruct their path. Because they must dissolve any scab that forms, keratinocyte migration is best enhanced by a moist environment, since a dry one leads to formation of a bigger, tougher scab. To make their way along the tissue, keratinocytes must dissolve the clot, debris, and parts of the ECM in order to get through. They secrete plasminogen activator, which activates

plasminogen, turning it into plasmin to dissolve the scab. Cells can only migrate over living tissue, so they must excrete collagenases and proteases such as matrix metalloproteinase (MMPs) to dissolve damaged parts of the ECM in their way, particularly at the front of the migrating sheet.

Contraction

Contraction is a key phase of wound healing. If contraction continues for too long, it can lead to disfigurement and loss of function. Thus, there is a great interest in understanding the biology of wound contraction, which can be modeled in vitro using the collagen gel contraction assay or the dermal equivalent model. Contraction commences approximately a week after wounding, when fibroblasts have differentiated into myofibroblasts. In full thickness wounds, contraction peaks from 5 to 15 days post wounding. Contraction can last for several weeks and continues even after the wound is completely re-epithelialized. A large wound can become 40–80% smaller after contraction. Wounds can contract at a speed of up to 0.75 mm per day, depending on how loose the tissue in the wounded area is. Contraction usually does not occur symmetrically; rather most wounds have an 'axis of contraction,' which allows for greater organization and alignment of cells with collagen. At first, contraction occurs without myofibroblasts involvement. Later, fibroblasts, stimulated by growth factors, differentiate into myofibroblasts. Myofibroblasts, which are similar to smooth muscle cells, are responsible for contraction. Myofibroblasts contain the same kind of actin as that found in smooth muscle cells. As the actin in myofibroblasts contracts, the wound edges are pulled together. Fibroblasts lay down collagen to reinforce the wound as myofibroblasts contract. The contraction stage in proliferation ends when myofibroblasts stop contracting and commit apoptosis. The breakdown of the provisional matrix leads to a decrease in hyaluronic acid and an increase in chondroitin sulfate, which gradually triggers fibroblasts to stop migrating and proliferating. These events signal the onset of the maturation stage of wound healing.

Maturation and Remodeling Phase

- When the levels of collagen production and degradation equalize, the maturation phase of tissue repair is said to have begun. During maturation, type III collagen, which is prevalent during proliferation, is replaced by type I collagen. Originally disorganized collagen fibers are rearranged, cross-linked, and aligned along tension lines. The onset of the maturation phase may vary extensively, depending on the size of the wound and whether it was initially closed or left open, ranging from approximately 3 days to 3 weeks. The maturation phase can last for a year or longer, similarly depending on wound type.
- As the phase progresses, the tensile strength of the wound increases, with the strength approaching 50% that of normal tissue by 3 months after injury and ultimately becoming as much as 80% as strong as normal tissue. Since activity at the wound site is reduced, the scar loses its red appearance as blood vessels that are no longer needed are removed by apoptosis.

The phases of wound healing normally progress in a predictable, timely manner; if they do not, healing may progress inappropriately to either a chronic wound such as a venous ulcer or pathological scarring such as a keloid scar.

FACTORS AFFECTING WOUND HEALING

Many factors controlling the efficacy, speed, and manner of wound healing fall under three types: local, systemic and general factors.

Local Factors

- Mechanical factors
- Edema
- Ischemia and necrosis
- Foreign bodies
- Low oxygen tension
- Blood circulation
- Tissue state
- Tension in tissue planes
- Collections
- Irritation
- Lack of rest to the part
- Excessive use of topical steroids
- Hypoxia
- Infection.

Systemic Factors

- Inadequate perfusion
- Inflammation
- Immunosuppression
- Connective tissue disorders
- Smoking.

General Factors

- Arterial disease
- Ascorbic acid
- Venous hypertension
- Steroids
- Marfan's syndrome
- Vitamin A deficiency
- Irradiation
- Obesity
- Anticancer drugs
- Anticoagulants
- Uremia
- Diabetes
- Drugs (steroids)
- Nutritional status.

FACTORS INFLUENCING WOUND HEALING

Role of Oxygen in Wound Healing

- Oxygen reaches the wound bed through blood flow to the area. Most cells in the wound environment have an enzyme that converts oxygen to a form that allows the cell to support wound healing.
- Wound contraction, collagen deposition, angiogenesis and granulation are examples of wound healing steps supported by oxygen.
- Oxygen even has an antibiotic effect allowing tissues to resist pathogens. Wound tissue oxygenation is a sensitive indicator for the risk of postoperative infections. The presence of edema and necrotic tissue makes it more difficult for oxygen to reach the wound. Since compression can reduce edema and debridement can reduce the presence of necrotic tissue, these procedural interventions are important components of most wound care.
- Improvement of oxygen level in wound tissue alone may trigger wound healing. Adequate oxygen levels will enhance the effectiveness of growth factors and a host of other cells that require oxygenation to maintain their function.

Role of Moisture in Wound Healing

- Modern wound management is based on the concept of creating and maintaining a moist wound environment to facilitate wound healing.
- Maintaining a moist wound with an occlusive dressing will hold endogenous fluids on the wound, preserving the cells needed for healing and keeping them in contact with the wound bed. It also softens wound scab and eschar; under the right conditions the body's own enzymes will dissolve the eschar in a process called autolytic debridement. Occlusive dressings maintain appropriate wound surface temperature to prevent delays in healing, protect the wound surface from trauma and from bacteria and other contaminants.
- *Basic principles of moist wound healing* include covering the wound with a barrier (occlusive dressing) that preserves adequate wound hydration limiting fluid loss from the wound surface, while the dressing is in place; all owing gaseous exchange; maintaining periwound integrity; controlling heavy exudates and removing the dressing, when exudate begins to leak out from edges of dressing.

Role of Nutrition in Wound Healing

- It is well, established that nutritional status can have a significant impact on wound healing.
- Nutrients that must be present for a wound to heal include iron, vitamin B_{12} and folic acid (essential so that red blood cells can deliver oxygen to tissues), vitamin C and zinc (essential for tissue repair) vitamin A (essential to stimulate collagen cross-linking) and arginine (enhances healing and immune function). High protein intake provides the amino acids required to build new tissue.
- Protein and calorie needs will vary depending on the size of the wound and the medical condition of the patient.

13.3 MANAGEMENT OF WOUNDS

GOALS

- Self-care of wound
- Management of peripheral vascular disease (PVD)
- Provide a moist wound healing environment
- Promote angiogenesis
- Promote granulation tissue formation
- Decrease necrotic tissue on wound site
- Promote wound re-epithelialization
- Decrease pain.

MANAGEMENT

Assessing the patient's ability to heal, diagnosing and modifying treatable cause of tissue damage will promote a moist wound healing environment. The patient must be assessed to determine if the blood supply is adequate to support healing. If a regional pulse is felt it will favor a wound healing. Low serum albumin levels will delay healing. Hence nutritional status has to be screened to favor healing.

Pain can cause activation of the sympathetic branch of the autonomic nervous system, leading to tissue hypoxia and can also stimulate the hypothalamic-pituitary-adrenal system to release cortisol, which affects wound healing. The different causes of the pain are back ground pain (pain at rest), incident pain (pain during day to day activities such as friction, bandage slippage, etc.) and procedural pain (pain from procedures such as dressing removal, etc.). The management strategies includes treating the underlying etiology pathologies of wound, analgesics and dressing selection, which is the key management.

Cleaning

The wound cleansing method should be selected based on its ability to support or return a wound bed to homeostasis.

Whirlpool

Whirlpool is effective in the deodorization, skin and wound cleansing, mechanical non-selective debridement, wound decontamination and infection control. Contamination, formation of edema (since the extremities in dependent position), delayed wound healing are its side effects.

Pulsatile Lavage with Suction (PLWS) or Forceful Irrigation

Forceful irrigation is a combination of wound irrigation combined with suction. It removes the wound exudates and loose debris, also it speeds healing by rapid removal of contaminants. The risk includes overuse especially with clean,

granulating wound and risk of trauma to newly formed tissue. Treatment may be also painful to the patient.

Nonforceful Irrigation

Nonforceful irrigation technique includes poring a solution directly over the wound without producing any force on wound bed. Infective wounds can also be effectively cleaned with nonforceful irrigation.

Topical Skin Cleansers

Along with nonforceful irrigation, topical cleansers have antimicrobial effect such as antimitotic effects.

Debridement

Debridement is defined as the removal of foreign material and dead or damaged tissue. Effects of debridement are removal of dead or devitalized tissue, prevent bacterial growth, encourage normal cellular activity in wound bed and enhance the rate of tissue repair. The two types are selective and nonselective debridement. In selective debridement removes the necrotic tissue in a controlled method. In nonselective debridement removes all the tissue both necrotic and living. Types of nonselective debridement includes wet to dry dressings, surgical debridement, pulsatile lavage with suction and whirlpool. Sharp debridement, chemical (enzymatic) debridement, autolytic debridement, biosurgery, medical grade honey are included in selective debridement.

Topical Agents

Topical agents are mainly to prevent infection and accelerate healing:

- *Antiseptics*: Povidone iodine, sodium hypochlorite solution, acetic acid solution
- *Oxidizing agents*: Hydrogen peroxide solutions
- *Antibacterials*: Neosporin, sulfadiazine, gentamycin, etc.
- *Analgesics*: Lidocaine, prilocaine, etc.
- *Growth factors*: Autologel, becaplermin gel, etc.

Closure

If a person presents to a healthcare center within 6 hours of a laceration they are typically closed immediately after evaluating and cleaning the wound. After this point in time, however, there is a theoretical concern of increased risks of infection if closed immediately. Thus some healthcare providers may delay closure, while others may be willing to immediately close up to 24 hours after the injury. A single study has found that using clean nonsterile gloves is equivalent to using sterile gloves during wound closure.

If closure of a wound is decided upon a number of techniques can be used. These include bandages, a cyanoacrylate glue, staples, and sutures. Absorbable

sutures have the benefit over nonabsorbable sutures of not requiring removal. They are often preferred in children. Buffering the pH of lidocaine makes the freezing less painful.

Adhesive glue and sutures have comparable cosmetic outcomes for minor lacerations <5 cm in adults and children. The use of adhesive glue involves considerably less time for the doctor and less pain for the person with the cut. The wound opens at a slightly higher rate, but there is less redness. The risk for infections (1.1%) is the same for both. Adhesive glue should not be used in areas of high tension or repetitive movements, such as joints or the posterior trunk.

Dressings

The dressing that is applied directly to the wound is referred to as primary dressing. The dressing that is applied over the primary dressing is referred to as the secondary dressing. Types of dressing includes gauze/fiber dressing, impregnated gauze, transparent films, foam, hydrogels, hydrocolloids, alginates, hydrofiber dressing, etc. Descriptions of these dressings are beyond the scope of this book.

14 Ulcer and Its Management

14.1 ULCER

DEFINITION

Ulcer is defined as a break in skin or mucous membrane with loss of surface tissue, disintegration and necrosis of epithelial tissue, and often pus. Ulcer is a type of chronic wound, which occurs as a result of an underlying or internal etiology.

CLASSIFICATION

Clinical

- *Spreading*: When the surrounding skin of the ulcer is inflamed and the floor is covered with slough without any evidence of granulation tissue.
- *Healing*: When there is granulation tissue in the floor of an ulcer, the surrounding skin is not inflamed and the edge shows bluish outline of growing epithelium.
- *Callous*: When there is pale granulation tissue in the floor, there is considerable induration at the base, edge and surrounding skin. This ulcer shows no tendency towards healing.

Pathological

- *Specific*: Seen in tuberculosis, syphilis, Meleney's, actinomycosis, malignant and rodent.
- *Nonspecific*:
  - *Traumatic ulcer*:
    - Mechanical, e.g. dental ulcer of the tongue from jagged tooth.
    - *Physical*: From electrical or X-ray burn.
    - *Chemical*: From application of caustics.

- Arterial ulcer occurs in atherosclerosis, Buerger's disease and Raynaud's disease.
- Venous ulcer occurs in postphlebitis limb or in chronic venous insufficiency.
  Tropical ulcer occurs in leg and feet of the people in tropical countries.
- *Cryopathic ulcer*: Ulcer chilblains and cold injury.
- Martorell's ulcer (Hypertensive ulcer).
- Bazin's ulcer (Erythrocyanoid ulcer).
- Diabetic ulcer.

Ulcer that is related to peripheral vascular disease (vascular ulcers) is mentioned in detail in the following chapters. **Table 14.1.1** provides differences venous ulcer and arterial ulcer.

Table 14.1.1 Difference between venous and arterial ulcer

Features	*Venous ulcer*	*Arterial ulcer*
Age	Common in 50–70 years	Elderly
Gender	Women	Men
Risk factors	Risk associated with venous disorders such as DVT, varicose, CVI, etc.	Risk associated with arterial disorders such as atherosclerosis because of hypertension, smoking, diabetes, etc.
Site	Lower two-thirds of the lower leg (slightly higher on the anterior and medial aspects) and on parts of the foot not supported by the shoe	More commonly on toes, foot and heel, but may be found on lower leg—lateral malleolus, tibial area and more pressure points
Cause	Venous congestion	Inadequate arterial supply
• Ulcer characteristics • Type • Base	• Shallow or irregular • With granulation tissue • Bright red	• Punched out appearance • Pale red • Unhealthy appearance of wound bed, with necrotic tissue
Pain	Minimal	Moderate to severe
	Throbbing, aching and heavy feeling in the leg	Intermittent claudication
	Improves with elevation and rest	Can be worse at night and at rest Improves with dependency
Condition of the skin	• Hyperpigmented, hemosiderin staining • Dry skin, normally warmer	• Shiny and taut skin • Reduced or no hair • Cooler to touch
Condition of the leg	• Pedal pulses present • Normal capillary refill • Edema • Eczema, itchy skin • Ankle flare • Lipodermatosclerosis • Varicose vein	• Pedal pulses absent • Delayed capillary refill • Dependent rubor and palor on elevation • Gangrenous toes

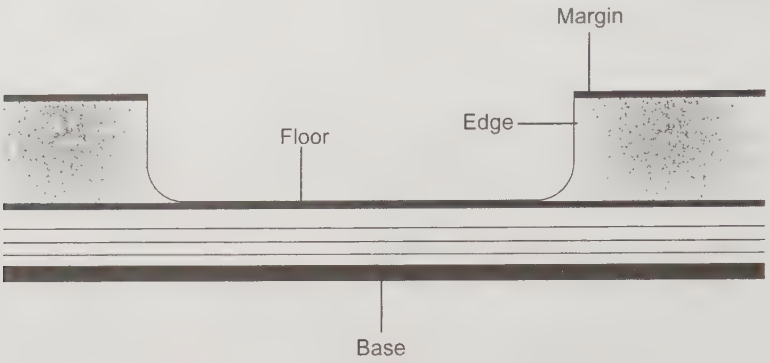


Fig. 14.1.1 Diagrammatic representation of various parts of an ulcer

Parts of an ulcer **(Fig. 14.1.1)**.

- *Margin*: It is the junction between normal epithelium and the ulcer; it is the boundary of the ulcer.
- *Edge*: It is the area between the margin and the floor of the ulcer.
- *Floor*: It is the exposed surface of the ulcer.
- *Base*: It is the part on which the ulcer rests.

14.2 VENOUS ULCERS

Synonyms: Venous insufficiency ulceration, stasis ulcers, stasis dermatitis, varicose ulcers or ulcus cruris.

DEFINITION

Venous ulcers are wounds that are thought to occur because of improper functioning of venous valves, usually of the legs (hence leg ulcers). Definition of chronic venous leg ulcer is defined as an open lesion between the knee and ankle joint that remains unhealed for at least 4 weeks and occurs in the presence of venous disease. Venous ulcers develop mostly along the medial distal leg and can be very painful.

ETIOLOGY

Chronic venous insufficiency is the most common cause of venous ulcer typically associated with earlier deep vein thrombosis, congenital weakness of repetitive stresses on venous valves or vasculitis, inflammation of the small vessels. It can occur in combination with diabetes, peripheral arterial diseases or infection.

Precipitating Factors

- Occupations demanding prolonged standing
- Poor personal hygiene and malnutrition
- Family history of chronic venous insufficiency
- Obesity preventing calf muscle contraction
- Local trauma (often very slight) breaks the weakened skin.

PATHOLOGY

Because of above mentioned etiologies, valve incompetence results. Damaged venous valves leak, so the lower leg bears more of the weight of the column of venous blood between heart and ankle. This increased pressure is called venous hypertension. This hypertension forces fluid out of vascular system into interstitial space, which results in lower-limb edema. This results in increased exudate and slowing down of the blood flow. Nutrition of the tissues is diminished and the skin is devitalized. It causes the itching, painful, inflamed skin called venous dermatitis. If edema is not corrected, it causes hyperpigmentation, lipodermatosclerosis, white atrophy and hyperkeratosis. The cells become necrosed and the skin break down ultimately results in venous ulcer. There is insufficient oxygen and nutrition to promote healing and the area remains open. Bacteria may invade the area or the dead cells may irritate the normal tissue, causing inflammation and the ulcer spreads.

CLINICAL FEATURES

Ulcer commonly located on the medial aspect of the lower leg. Lower leg edema is reduced by leg elevation. Increased lower leg ache or pain as edema increases during prolonged standing or sitting. A palpable normal temperature and palpable peripheral pulses in the feet rule out venous ulcer from arterial ulcer.

The floor of the ulcer may be pale and anemic with watery discharge; indolent (static, unhealing) ulcer, green or yellow discharge; infected ulcer, pink, bubbly with red spots; granulating ulcer. The edge of the ulcer may be well-defined, straight or undermined, red and shiny; ulcer spreading, hard, edematous, overhanging floor; chronic ulcer, shallow, sloping out from floor with bluish tinge; ulcer healing. The base of the ulcer may show gross induration (hardening), this is extent of which varies according to the severity and duration of the ulcer. There may be pigmentation because of breakdown of red blood cells. There may be coarse skin texture with heavy scaling or papery thin and eczematous tissue. There may be partial scar tissue. edema may be present at the base of ulcer and foot and ankle to shoeline. There may be considerable pain around the ulcer, especially if infected. Pain increases on walking. There will be limited movement of the foot and ankle. There may be muscle weakness and atrophy mainly of calf muscles with loss of pumping action. Patients adopt a walking pattern poor with no push off.

MANAGEMENT

Conservative Management

Management is same as that of outlines given in the wound management such as cleansing, debridement, topical agents and dressings, etc. stopping the use of nicotine and substances that restrict circulation. Improve nutrition to the patient. Avoid scratching or other trauma to the venous ulcer or surrounding skin.

Cleaning and Dressing the Ulcer

The ulcer is cleaned before the administration of physiotherapy. The cleaning and dressing should be carried out using a nontouch technique with sterile packs, instruments and lotions. Cotton wool balls soaked in saline (clean ulcer) or hydrogen peroxide or eusol (infected ulcer) are used for cleaning. If the ulcer is very painful it may be irrigated instead of cleaned with cotton wool. Sterile gauze swabs soaked in saline can be used for screening the ulcer for ultraviolet radiation. A great variety of ointments, solutions and preparations are available for dressing the ulcer and surrounding skin. A desloughing agent may be applied to a very infected ulcer and specific antibiotics creams may be necessary for specific infections. Paraffin gauze is useful to protect the floor of a granulating ulcer. A soothing cream, e.g. calamine, may be used if surrounding skin is irritable, painful and eczematous, but arachis oil is better for dry, scaly skin.

A gauze compress covering up to 2 cm of the ulcer surrounds may be used if the ulcer is shallow and granulating at the edges. For deep ulcers silastic foam (a silicon-based fluid, which sets to the shape of the ulcer) may be used during the granulation stage, but for infected ulcers ribbon gauze soaked in varidase packed into the cavity ensures that the lotion is in contact with the floor. Finally the ulcer is dressed in a nonabsorbent dressing, e.g. Melolin.

If the ulcer has a copious discharge, cotton wool padding will absorb the exudate. A sorbo-rubber pad or white felt 2 cm thick can be applied if the ulcer is over granulating, or there are persistent patches of local edema (induration).

An ulcer often responds to one solution or dressing for 1–2 weeks and then slows up. When this happens it is useful to change the solution or dressing.

Advice

When sitting, the patient must elevate the legs with support under the knees and avoid standing still for any length of time. Any increase in pain must be reported.

Surgical Management

The healing of large venous ulcers may be hastened by surgical intervention. This may include:

- Ligation of veins
- Debridement and skin grafting.

Ligation of veins: Is usually necessary to improve the venous return of the lower-limb, which is the predisposing cause of the ulcer.

Debridement and grafting: If an ulcer is infected it must be cleaned before applying a skin graft. This must be done by local application of antiseptic lotions and application of ultraviolet therapy. Various types of split skin grafts may be carried out. Mesh grafts are more successful particularly if the ulcer is large. The skin is normally taken from the thigh and passed through a mesher, which makes multiple slits enlarging the graft. The slits enable the circulation to move freely through the graft and therefore 'take' readily.

If the chronic venous congestion is reduced and the circulation, bringing oxygen and nutrition to the area, is improved together with the removal of any infection and the mobilizing of the soft tissues, the ulcer will heal with the formation of the scar tissue.

PREVENTION OF VENOUS ULCER RECURRENCE

Education about venous ulcer etiology, nutrition, infection signs, lower-leg exercise, elevation and compression.

14.3 ARTERIAL ULCER

Synonym: Ischemic ulcer.

DEFINITION

An ulcer caused by diminished blood flow through an artery, especially the one that nourishes a finger or toe. It is always caused by atherosclerosis, or fatty plaque build-up in the arteries, which narrow and harden, resulting in poor blood circulation. Even a small scratch may not heal properly because of the compromised blood supply, and can lead to the development of an ulcer.

INCIDENCE

Gender: Men are more affected than women

Age: Commonly affects the elderly.

ETIOLOGY

Arterial ulcer occurs in those parts of limbs, which are subjected to repeated pressure and trauma. This causes ischemic damage to the tissues and there may be lack of nutrition to the skin. If the circulation is inadequate because of disorders such as atherosclerosis, Raynaud's disease and Buerger's disease, tissues cannot repair by themselves the ulcer develops. Pure arterial ulcers are unusual.

Arterial insufficiency frequently contributes to poor healing in ulcers with another primary etiology such as diabetic neuropathy or venous insufficiency. Ulcers in patients with rest pain and gangrene may progress rapidly and delay in referral increases the risk of limb loss.

Site

On the dorsum of feet, lateral malleoli, on the heels, tips of toes, between the toes where the toes rub against one another or anywhere the bones may protrude and rub against bed sheets, socks or shoes. They also occur commonly in the nail bed, if the toenail cuts into the skin or if the patient has had recent aggressive toe nail trimming or an ingrown toenail removed.

CLINICAL FEATURES

History of intermittent claudication with the discoloration of one or more toes becomes the differentiating feature. Arterial ulcers are typically very painful, especially at night. The patient may instinctively dangle his/her foot over the side of the bed to get pain relief. If leg is elevated above the hearts level, the ulcer shows no sign of healing.

Characteristics

Arterial ulcer being small, round and with smooth, well-demarcated borders ulcers tend to be punched out and destroy the whole skin and the deep fascia, may expose the tendons in the floor of the ulcer.

Base

It has a yellow, brown, gray or black color and usually does not bleed and lacks granulation tissue. The floor of the ulcer is pale, anemic and liable to infection. The surrounding skin may be normal or ischemic.

Borders

The borders and surrounding skin usually appear punched out. If irritation or infection are present, there may or may not be swelling and redness around the ulcer base. There may also be redness on the entire foot when the leg is dangled; this redness often turns to a pale white/yellow color when the leg is elevated. Patches of dry gangrene are present along with the arterial ulcer.

The physical examination may reveal a decrease in peripheral pulses-dorsalis pedis and posterior tibial pulses, lack of hair over the distal leg, and cyanosis, pallor, and/or atrophy of the surrounding skin. Lifting the leg greater than 30° can induce pallor in the ischemic limb. When dropped to a dependent position, the limb may become very red. There may be delay in capillary filling response, venous filling time and rubor of dependency tests, Ankle Brachial Index results shows the value less than nine. Transcutaneous oxygen tension ($TcPO_2$) on the peri-wound skin <40 mm Hg. There may be Doppler arterial waveforms disparities and dampened pulse volume recordings.

MANAGEMENT

The ulcer will not heal unless the blood supply is improved and usually surgery is necessary. Depending on the patient's condition, the physician may recommend invasive testing, endovascular therapy or bypass surgery to restore circulation to the affected leg. Management is same as that of venous ulcer. The following points should be noted.

Management of arterial ischemic ulcers classically includes conservative debridement, pain control, use of occlusive dressings, and improvement of circulation. Treatment is also directed at the pathogenic causes of arterial disease. In arterial ulcer with dry gangrene or eschar, debridements should not be used until arterial in flow has been established. Devitalized tissue impairs the ability to fight infection and serves as rich environment for bacterial growth. Arterial ulcers should be considered for short course of antibiotics even if sign of infection are not present. Topical antimicrobial dressings may be beneficial. Avoid the condition such as dehydration, cold, stress and pain that reduces tissue perfusion as this could reduce oxygen delivery to the tissues. Smoking reduces tissue oxygen by peripheral vasoconstriction. After surgery local treatment as for a venous ulcer will reduce infection and promote healing.

Moist wound care accelerates the wound-healing process. A moist wound environment physiologically favors cell migration and matrix formation accelerating the healing of wounds. Dry dressings, except over intact skin, are considered injurious. They can cause desiccation of the wound, which reduces new granulation tissue formation, as well as removing granulation tissue during dressing changes. Dressing changes should be only once daily.

Spinal cord stimulation (SCS) seems to be promising as an adjuvant therapy in managing lower-limb ischemia and ulceration based on animal studies and case series. It is particularly useful in reducing pain. Topical negative pressure wound therapy appears to be promising for mixed ulcers. Intermittent pneumatic leg compression (IPC) increases blood flow and it may be beneficial in limbs with impaired distal perfusion, either before or after revascularization. Hyperbaric oxygen therapy (HBOT) should be considered as an adjuvant therapy.

The goals for arterial ulcer treatment include:

- Providing adequate protection of the surface of the skin
- Preventing new ulcers
- Removing contact irritation to the existing ulcer
- Monitoring signs and symptoms of infection that may involve the soft tissues or bone.

Prevention of Ulcer

In order to prevent ulcers from developing or getting worse the following risk factors have to controlled:

- Quit smoking
- Manage blood pressure

- Control blood cholesterol and triglyceride levels by making dietary changes and taking medications as prescribed
- Limit intake of sodium
- Manage diabetes and other health conditions, if applicable
- Encourage in physical activity or exercise
- Lose weight if you are overweight
- Aspirin therapy to prevent blood clots.

Surgical Management

In patients with arterial insufficiency ulcers, restoration of blood flow by revascularization is the intervention that will most likely lead to healing. Prior to revascularization, an anatomic road map should be obtained through angiogram and angiography.

Closing the arterial ulcer with an autologous skin graft (pinch graft, split-thickness graft, meshed graft, full-thickness graft) or an autologous flap can assist in healing the wound and aid in the preservation of lower limbs. Skin graft survival is dependent on appropriate wound bed preparation and perfusion.

14.4 DIABETIC ULCER

INTRODUCTION

Patient with diabetes develop a complications called diabetic foot. Diabetic foot can be of two distinct entities. They are neuropathic foot and neuroischemic foot. Diabetic ulcer is a major component of diabetic foot.

DEFINITION

A nonhealing or poorly healing full-thickness wound, through the dermis, below the ankle in an individual with diabetes, critical in the natural history of the diabetic foot.

ETIOLOGICAL TRIAD

- Neuropathy
- Ischemia
- Infection.

Neuropathies

In diabetic neuropathy, nerve damage is because of the metabolic abnormalities affects sensory, motor and vasomotor control of pedal circulation. Overall these results in loss of protective sensation, lack of awareness, altering the distribution

of forces during walking, causes thickening of skin (callus formation) at the sites of abnormal load.

Ischemia

Foot tissues can become ischemic because of atherosclerotic disease. The protective sweating is lost and skin becomes red, dry, thin with dystrophic nails and susceptible to pressure from shoes and nearby toe.

Infection

As blood glucose is a good medium for culture of bacteria, infections are more common in diabetic ulcers. Infections can be superficial, local, soft tissue, cellulitis and osteomyelitis. These infections result in localized digital gangrene.

PREDISPOSING FACTORS

- Poor vision
- Limited joint mobility
- Deformities of foot
- Consequences of cardiovascular or cerebrovascular disease
- *Accidental trauma*: Ill-fitting footwear.

EXAMINATION OF DIABETIC FOOT

GRADING OF DIABETIC ULCER

The three main diabetic foot classification system are discussed that are commonly used in clinical diagnosis of diabetic foot are Wagner-Meggitt classification, depth-ischemic classification, University of Texas classification. Most common and widely used classification system is the Wagner diabetic foot classification system. This system is basically anatomical with gradations of superficial ulcer, deep ulcer, abscess osteitis, gangrene of the forefoot and gangrene of the entire foot. Only grade 3 addresses the problem of infection. In this system foot lesions are divided into different grades starting from grade 0 to grade 5. Grade 0 includes high-risk foot, but no active lesion and grade 5 includes gangrene of entire foot. But this system does not mention about ischemia or neuropathy and that is the drawback of this system **(Table 14.4.1)**.

Table 14.4.1 Wagner-Meggitt grading

Grade	*Lesion*
0	No open lesion
1	Superficial ulcer
2	Deep ulcer to tendon or joint capsule
3	Deep ulcer with abscess, osteomyelitis or joint sepsis
4	Local gangrene—forefoot or heel
5	Gangrene of entire foot

Inspection

Sites to be checked are dorsum, plantar surface (sole) of foot (metatarsal heads and midfoot), toes (dorsal interphalangeal joints or distal tip), interdigital areas, medial and lateral borders of both feet, and back of heel over Achilles tendon. Corns and calluses develop at sites of high-pressure and friction.

Ingrowing toe nails: Because of incorrect nail cutting and poorly fitting shoes and fungal nail infections. Unilateral hot, red, swollen foot, deformities such as clawing of toes, high arch are the characteristic features of Charcot foot. Fibrofatty pad reduction over the head of the metatarsal bones is a common sign. This pad thinning reduces the ability of foot to absorb increased plantar pressure, which increases the risk of ulceration. In neuropathic ulcer skin will be dry because of decreased sweating and in ischemic ulcer the skin will be thin, dry and fragile. Web space maceration and skin blistering or fissuring indicates fungal infection.

Palpation

Palpation of femoral, popliteal, dorsalis pedis and posterior tibial pulse is equally important. If either dorsalis pedis or posterior tibial pulse can be felt, then ischemia is unlikely. A warm and palpable pulse is a characteristic feature of neuropathic ulcer where as cold and absent pulse is a characteristic feature of ischemic ulcer. Checking the warmth for both feet will rule out an infection, fracture or acute Charcot foot. Presence of edema and crepitus indicates a foot at high-risk for severe infection.

Neurological Assessment

- Signs of motor neuropathy are high medial arch, claw toes and metatarsal head prominence.
- Ankle reflexes are absent or diminished in neuropathic ulcer and present in ischemic ulcer.
- Signs of autonomic neuropathy are dry skin and fissuring.
- Signs of sensory neuropathy include ulcer formation and skin changes. Pressure has to be checked with a monofilament and vibration with a tuning fork.

Inspection of Footwear

Patient's shoes and socks should be assessed. Too narrow or small shoes increase the friction and ulceration. The toe box of the shoe must be wide and deep and sole of the shoe should be thick enough to provide protection from penetration of any sharp objects. Socks should be absorbent and must not be thick.

Investigations

Foot X-ray: This will rule out fracture, gas in soft tissue or a Charcot joint.

Vascular investigations: Ankle brachial index (ABI), toe systolic pressure, Doppler as mentioned before in Chapter 8.3. Transcutaneous oxygen tension measurement is used to check the degree of ischemia.

Neurological investigations: A neurothesiometer can quantify the degree of neuropathy present. A vibratory stimulus is applied to the foot and this increases as the voltage is raised. Any patient unable to feel a vibratory stimulus of 25 volts is at risk of ulceration.

Foot pressure assessment: This assesses the distribution of plantar pressure on the sole of the foot.

Skin temperature assessment: Comparison of similar areas on each foot is done with the help of digital skin thermometer.

MANAGEMENT

Eradication of infection can be achieved by the application of broad spectrum antibiotics. Removal of callus can be performed by a podiatrist.

Ulcer Management

- Ulcer bed preparation removes necrotic tissue, exudates, bacteria and callus
- *Application of antiseptics*: Iodine and silver
- *Dressings*: Hydrogel, hydrocolloids, films, foams and alginates.

Surgical procedures such as angioplasty or thrombolysis or bypass surgery can be performed to revascularize area associated with ischemia if possible.

Physiotherapy Management

Outline is already mentioned in Chapter 14.7. In addition to electrophysical agents, Buergers exercises are administered to facilitate circulation in lower extremities.

Offloading: Is the ability to reduce pressure forces over the wound site. Custom made orthotic devices and plaster or fiberglass casts are use to offload the wound, while allowing the patient to remain active. In case of offloading, prevent muscle wasting by active range of motion (ROM) for leg and foot muscles.

Diabetic footwear: The purpose of a footwear is that it relieves excessive plantar pressure, minimizes friction between foot and the footwear, stabilization of deformity and also helps in offloading and recurrence of ulcer.

Total contact cast is the appropriate way of resting the foot with diabetic ulcer. It distributes the weight along the entire plantar aspect of the foot. It produces shortened stride length and decreased walking velocity. Rocker soles can also relieve pressure from the different area of sole of the feet. The different types of rocker soles used are mid rocker soles, heel to toe rocker soles, toe only rocker sole, severe ankle rocker sole and negative heel rocker sole.

Modification of the gait pattern: Patients should emphasize a hip pull-off gait pattern. Patient should pull their legs forward from the hips and shorten their steps, but not slow their walking speed. This reduces their push-off at the ankle.

Walking aids: Use of walking aids such as crutches, walker, cane can significantly achieve offloading thereby reduces plantar pressures. Axillary crutches were more effective in reducing the pressure followed by walker and canes.

Functional mobility: Range of motion (ROM) and strengthening exercises, gait training with assistive devices. Soaking the feet for 20–30 minutes in cold water can be done in order to keep the foot supple and smooth especially in patients who have fissures and cracks.

Limited joint mobility: This can be corrected by teaching active exercise to toes and foot.

- Neuropathic pain can be minimized by the application of transcutaneous electrical nerve stimulation (TENS) or interferential therapy (IFT).
- *Regular foot care, skin care and nail care*: Already mentioned in chapter.

Prevention of Ulcer Recurrence

- Improve blood glucose control
- Reduction of cardiovascular risk factors
- Reduce further abnormal pressure loading such as cushioning, individually fitted footwear
- Education on foot care
- Regular examination of sole of the foot for blister, callus, corns and wound.

14.5 DECUBITUS ULCER

Synonyms: Pressure sore, bedsore.

DEFINITION

Pressure sore is a term used to describe an area of erythema to a deep-seated ulceration, damage to the underlying soft tissues and exposing the underlying bone that results from external pressure, friction or shear forces applied to the skin.

TYPES

There are two types of pressure sores—superficial and deep.

Superficial Type

This begins with breakdown of the skin surface resulting in destruction of the epidermis, dermis and possibly subcutaneous tissues. The resultant ulcerated area may become infected with a yellow or green exudate.

Deep Type

This begins in the subcutaneous tissues overlying bony prominences. It results in necrosis of the subcutaneous tissue, fascia and possibly muscle tissue.

The only sign may be a slight reddening of the skin surface. In severe cases the destruction may spread superficially through the dermis and epidermis until a deep cavity is exposed.

PREVALENCE

- *Age*: Can occur at any age, but is more commonly found in the elderly
- *Gender*: Sexes equally affected
- *Site*: Found in pressure areas, e.g. heels, buttocks, hips, elbows
- *Condition*: More commonly affects patients with neurological disorders, e.g. paraplegia and Parkinson's disease.

ETIOLOGY

This can be external factors (in the environment) or internal factors (in the body itself).

External Factors

Prolonged and constant pressure causes deficiency of blood supply. The tissue damage will depend on the amount and type of pressure—shear or friction. In shear pressure the skin remains stationary and the underlying tissues move forward, destroying the circulation, but in friction the skin surface moves over the bed surface, causing a superficial abrasion. Untrained home givers in turning or moving the patient can precipitate this.

The pressure may be caused by immobility of the patient due to:

- Postoperative pain
- Immobility in a plaster of Paris
- Unconsciousness
- Loss of sensation where the patient does not feel pain
- Prolonged bedrest.

Internal Factors

- Bony prominences, e.g. sacrum, greater trochanter, cause pressure to build-up internally.
- Increased muscle tone results in the patient remaining in a fixed position with increased pressure.
- Illness reducing the nutritional status, fluid, protein, calories, vitamin and minerals of the body.
- Incontinence results in skin breakdown due to moisture.
- Neural impairment, which results in weak or wasted muscle bulk causes poor protection for the underlying tissues.
- Diabetes may lead to trophic ulcers, infections.
- Previous ulcers.
- History of alcohol, nicotine or other drug use.

PATHOLOGY

- Direct external pressure exceeding the local skin capillary pressure can occlude local circulation.
- Friction such as that caused by dragging a patient across a bed or chair surface may stretch or damage local blood vessels
- Shear stresses arise when skin is held in contact with the supporting surface, but subcutaneous bone and tissue move, deforming and occluding local blood vessels.

In all three cases, local circulation is impaired, depriving cells of oxygen and nutrients, so they die. If the pressure is prolonged acute changes of inflammation take place with necrosis of tissue, suppuration and healing by second intention. Superficial pressure sores are three times as common as deep sores, but deep sores occur in seriously ill patients and are associated with a very high-mortality rate. In both types, the pressure compresses the tissues, occludes the blood supply and the nutrition is cut off.

CLINICAL FEATURES

There is an open area of varying size on a pressure site. The floor of the sore may be pink and vascular or filled with infected exudate. The cavity may be shallow or deep with loss of subcutaneous tissue and exposure of bone. Around the cavity, the skin is red or blue. The patient will complain of pain if sensory nerve endings are not destroyed.

ASSESSMENT

- *Location*: Identified by bony prominence using anatomical descriptions
- *Size*: Length, width, depth, undermining (maximum length × maximum width)
- Stage and grade
- *Surrounding skin*: Erythema, edema, damage, rashes
- *Ulcer edge*: Bevelled, vertical, rolled
- *Wound bed tissue type*: Necrotic, granulation tissue, epithelium
- *Exudate*: Serous, serosanguinous or purulent
- *Exudates amount*: None, scant, minimal, moderate, copious
- *Wound odor*: Presence or absence
- *Structures visible*: Tendon or bone
- *Pain*: Mild, moderate or severe.

PREVENTION OF PRESSURE SORES

Prevention of pressure sores is better than treatment of the sore. The aim of prophylactic treatment is to relieve pressure and prevent breakdown of skin:

- Turning the patient every 2 hours day and night and avoiding pressure on the sore.

  For example, for a trochanteric sore change from lying to side lying on the unaffected side.

- Use of a special mattress or bed designed to relieve pressure in lying:
  - *Water bed*: Which provides even pressure over all parts of the body
  - *Ripple mattress*: Which continually alters the pressure points
  - *Net bed*: An open mush net provides reduced pressure and is suspended between two wooden rollers allowing easy turning of the patient
  - *Air-fluidized bed*: Air is pumped through a sand medium giving complete flotation; the fluidization can be switched off giving a solid surface for ease of handling
  - *Low air loss bed*: Consists of waterproof sections filled with air to different pressures providing even pressure distribution.
  - Sorbo packs, which can be positioned to keep susceptible areas pressure free.
- *Sitting position*:
  - Avoid elevating the head of the bed more than 30°
  - Chair positioning with the back tilted slightly backward, legs supported on a rest with heels free
  - Uses special cushions.
- Sheepskins can help to keep skin dry and reduce friction, but are not suitable for incontinent patients. They can vary in size from a small square to one, which protects the whole body. Boots lined with sheepskin help to prevent pressure sores on the feet.
- *'Roho' cushion*: An air-filled cushion, which molds to any shape and spreads pressure evenly.
- Encourage patient to be mobile as soon as possible and encourage short walks.
- The patient is instructed to inspect pressure sites for signs of pressure and taught methods of self-pressure relief.
- Treatment of associated diseases will help to prevent skin breakdown. Incontinence must be treated, edema reduced and anemia corrected.
- A balanced diet to maintain patient's general health is essential.
- Good instructions in turning and lifting to the patient, the patient's relatives and carers is necessary if prophylactic measures are to be completely successful.
- Dermalex spray.
- *Physiotherapy includes the following exercises*:
  - Exercises for strengthening muscles to enable patient to lift himself/herself in bed or chair for pressure relief
  - Active exercises to encourage mobility in bed and walking assisted, if necessary as soon as possible
  - Ice massage over a reddened area for a few minutes several times a day will increase circulation and reduce edema, thereby preventing tissue breakdown
  - Relaxed passive movements to paralyzed limbs aid circulation and prevent contractures, which might produce pressure sores.

MEDICAL MANAGEMENT

The aims of treatment are to relieve pressure, reduce infection, control external moisture (urine and feces) improve circulation and promote healing:

- Turning and positioning the patient, together with the use of suitable beds and cushions as described above.
- Sterile cleaning to reduce infection using forceful or nonforceful irrigation or application of skin cleansers.
- Application of a dressing to promote healing. For example, nonstick perfron, semipermeable Op-site, silastic foam for deeper granulating sores, bactigras for infected sores or paraffin gauze for large granulating sores.
- A high-protein, high-calorie diet including all vitamins and iron improves the patient's general health and promotes healing.

PHYSIOTHERAPY

Tracings can be used to show decrease in the size, but not the depth of a sore. The treatment methods are outlined in Chapter 14.7. Since there is no edema of the surrounding tissues and no limited joint movement, compression and support bandaging are not used in the treatment of pressure sore. The response of the sore to the different modalities is very variable and the choice will depend on the state and progress of healing.

SURGICAL MANAGEMENT

Surgical Excision and Grafting

If the sore is infected debridement (excision of infected tissue) is necessary first. When a large sore is healing and unlikely to be subjected to further pressure a skin graft may be sufficient, but a rotation flap may be necessary if there is any danger of further breakdown. In a rotation flap there is rotation of muscle and skin flap to cover the defect and an additional skin graft to cover the area left by the flap. Following the flap, pressure must be relieved until the wound begins to heal.

14.6 WOUND ASSESSMENT

The characteristics of wounds may be defined as dry, wet or granulating. Wounds can also be defined by their etiology such as diabetic, vascular or traumatic. Wound characteristics describe the physical appearance of the wound but often provide the clues to the etiology, phase of healing and likelihood of closure. Wound characteristics can provide valuable information needed to make sound clinical judgments about the treatment.

The following are characteristics that should be tracked and documented throughout the phases of wound healing:

Location: Body diagrams can be used for pain to document wound location.

Shape: Wound shape is determined by evaluating the perimeter of the wound. As the wounds heal, they often change shape and may begin to assume a more regular, circular/oval shape. A butterfly shape or mirror image pressure ulcer on the sacrum has been associated with rapid evolution and mortality.

Size: Size is measured by linear method. Techniques are mentioned under wound measurement.

Depth: Depth is measured with the help of cotton tip applicator. Techniques are measured under wound measurement.

Edges: The following qualities are assessed in edges:

- *Distinctness*: The normal tissues blend into the wound bed in case of indistinct and diffuse edges.
- *Attachment*: If the edges are even with the skin surface and the wound base means that the wound is flat. If edges are not attached to the base of the wound or wound has sides, it implies that a wound with some depth of tissue involvement.
- *Thickness*: As the wound ages, the edges become rolled under and thickened to palpation. Wounds of long duration may continue to thicken with scar tissue and fibrosis developing in the wound edge, causing the edge to feel hard, rigid, and indurated.
- *Color*: The edge gets a unique color with time. The pigment turns a grayish hue in both dark- and light-skinned persons.

UNDERMINING/TUNNELING

Undermining and tunneling represent the loss of tissue underneath an intact skin surface. They are calculated by inserting a cotton tip applicator. Technique is mentioned under the heading wound measurement.

Necrosis

Necrosis is dead, devitalized tissue. Characteristics of necrotic tissue included in most wound healing measurement tools include presence, amount, color, consistency or moisture content, and adherence to the wound bed. The amount of necrotic tissue present in the wound is evaluated by measuring the length and width of the necrosis, and multiplying it to determine surface area of necrosis.

Exudate/Drainage

Evaluate the exudate or drainage type, amount and consistency.

Surrounding Skin Characteristics

The tissues surrounding the wound indicate further tissue damage. So look for color, including erythema, edema, induration, maceration, hemorrhage or hematoma.

Granulation Tissue

The presence of granulation tissue signals the proliferative phase of wound healing. It is present in full-thickness wounds only. Partial thickness wounds do not require granulation tissue formation for wound healing. Granulation tissue is healthy when it is bright, beefy red and shiny.

Epithelialization

It is the process of epidermal resurfacing and it appears as pink or red skin.

In partial-thickness wounds, the epithelial cells migrate from islands on the wound surface as well as from the wound edges. In full-thickness wounds, epidermal resurfacing occurs from the edges only.

Exposed Structures

Color and condition of bone, tendon, and ligament.

Pain

Although not a visible characteristic. It is measurable and significant to the intervention.

Quantity of Bacteria

Amount present in a wound. This is referred to as the bio-burden.

SUBJECTIVE EXAMINATION

- *History*
  - *Types of wounds*: Primary: Traumatic wound, surgical wound, burn/scald, fungating lesion. Secondary: Arterial ulcer, venous ulcer, diabetic ulcer, foot ulcer, pressure ulcer.
  - *Mechanism of injury or mode of onset*: Whether the ulcer developed due to trauma or spontaneously. Traumatic ulcer heals by itself. Spontaneous development of the ulcer can be due to varicosities or arterial insufficiency.
- *Location*: Vascular ulcers location will be different based on type of ulcer
- *Timing*
  - Date of onset
  - Progression of wound since onset
  - *Duration of wound*: An acute ulcer will be present for shorter duration whereas chronic ulcer will remain for longer period.
  - How often the patients get the wound

- *Settings*
  - Does the wound occur at certain place?
  - Does the wound occur under certain circumstances?
- *Pain history*: Pain: Ulcer resulting from nerve diseases is painless. Arterial ulcers are painful at rest and during limb elevation. Venous ulcers are painful in dependent position.
  - *Body diagram*: (Mark location with X and number each wound), e.g. front, back
  - *Feet diagram*: Right and left
  - Severity of pain can be assessed by visual analog scale
  - Position of the leg whether it increases or reduces pain.
- *Emotional distress on 0 to 10 scale*: How much the patient is distressed with the wound?
- Aggravating factors and relieving factors, e.g:
  - Wound due to arterial insufficiency, pain increases with elevation and reduces with legs dependent.
  - Wound due to venous insufficiency, pain reduces with elevation and increases with legs dependent.
- *Associated diseases*: Nervous disorders such as tabes dorsalis, syringomyelia, transverse myelitis, and peripheral neuritis may result an ulcer. Diabetes may lead to ulcer formation.

Past history: History of diabetes, hypertension, hypercholesterolemia, etc. provide clue about arterial diseases.

Previous medical history like chronic illness, hospitalization; and surgical history like amputations. Many medications interfere with wound healing or may interact with wound therapy.

Personal history: Alcohol, tobacco, and illicit substance use, in particular, presents significant problems for tissue perfusion and nutrition for wound healing. Exercise patterns influence healing of several wound types, such as venous disease ulcers.

Family history: History of diabetes mellitus, heart disease, and stroke, as these diseases can impair wound healing and are risk factors for further wounding.

Environmental history: The work environment, along with the occupational history, can identify job-related health risks and provide information on the ability of a patient to eliminate certain risk factors for impaired healing.

OBJECTIVE EXAMINATION

Observation

- *Type of lesion*: Primary [macules (without elevation), papules (elevation no fluid), vesicles (elevation and fluid)] or secondary (result of primary lesion called as ulcer)
- *Size and shape*: A bigger ulcer will definitely take longer time to heal. Varicose ulcers are oval in shape.
- *Number*: Varicose ulcers are more than one in number.

- *Position*: Venous ulcers are commonly located at medial malleolus. Trophic ulcers are located at the heel or on the ball of the foot. Arterial and diabetic ulcers are commonly located in the feet.
- *Edge*: Classification is given in the **Figure 14.6.1**.
- *Floor*: When the floor is covered with red granulation tissue, the ulcer seems to be healthy and healing. Pale and smooth granulation tissue indicates a slowly healing ulcer.
- *Type of drainage*: Serous, sanguineous, sero-sanguineous, sero-purulent
- *Amount and smell of discharge*: A healing ulcer shows a scanty serous discharge. A spreading ulcer shows purulent discharge.
- *Tissue type on wound bed*: Necrotic black, slough yellow, granulation red, epithelialization
- *Peri-wound skin (skin area adjacent to wound)*: Macerated, dry, edematous, erythema, excoriated, healthy, eczema wet/dry. If the surrounding area of an ulcer is glossy, red and edematous, the ulcer is acutely inflamed. If the surrounding skin is eczematous and pigmented, the ulcer is due to varicosities.
- *Presence of edema*: Pitting or nonpitting
- *Whole limb*: Inspection of whole limb is necessary. If there is presence of varicose vein and deep venous thrombosis and skin is pigmented, the ulcer can be varicose ulcer.
- *Stage of wound*: **Table 14.6.1**.

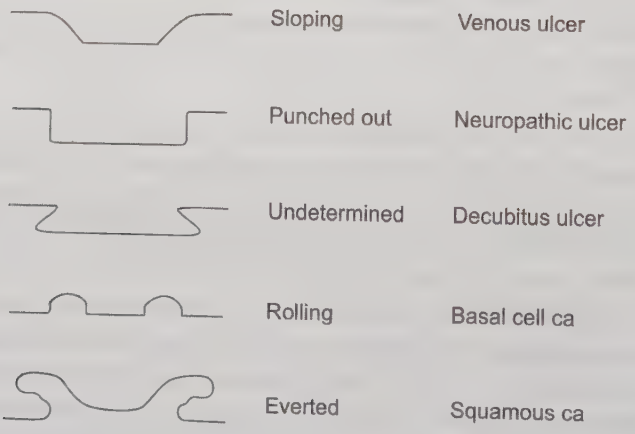


Fig. 14.6.1 Different edges of ulcer

Table 14.6.1 Stages of wound

Stage	*Description*	*Tissue involvement or loss*
I	Non blanchable, erythema epidermis, dermis intact	None
II	Complete loss of epidermis, partial description of dermis	Partial thickness
III	Complete loss of epidermis or dermis	Full thickness (superficial)
IV	Complete loss of epidermis or dermis with destruction of fascia, with muscle bone	Full thickness (deep movement)

Palpation

- *Tenderness*: An acutely inflamed ulcer will be always tender. A varicose ulcer may not be tender.
- *Edge and margin*: Marked induration (hardness) is characteristic feature of a carcinoma. Even the induration can be seen in chronic ulcer.
- *Base*: Base is better felt than seen compared to floor. So floor is not palpated.
- *Depth*: Can be measured in mm or mL. Mention in the wound assessment.
- *Bleeding*: Indicates malignancy
- *Relation with deeper structures*: Fixed or unfixed
- Palpation of the peripheral pulses is to be done rule out peripheral arterial diseases

WOUND MEASUREMENT

It includes:

- Linear method
- Tracing film method
- Wound photography.

Linear Method includes Measurement of Surface Area, Undermining, Depth and Volume

- Measurement of surface area (SA)
  - The greatest length and greatest width method refers to measurement across the diameter of the greatest length and greatest width of the wound. Multiplying the length by the width gives the estimated SA of the wound in centimeters squared.
  - The clock method is another way to measure the SA of wounds. In this method, you imagine the wound as the face of a clock. Select a 12:00 reference position on the wound and this position is usually toward the patient's head. Then measure from wound edge at 12:00 to wound edge at 6:00 position and measure from wound edge at 3:00 to wound edge at 9:00 position. Multiply these measurements to get the area.
- Measurements of undermining (erosion under the wound edge) and tunneling (a sinus) indicate the extent of wound damage into surrounding deep tissue. They are calculated by inserting a cotton tip applicator. Map undermining around the entire wound perimeter by inserting a moist, cotton-tipped applicator into the length of the undermined/tunneled space and continuing around the perimeter. Mark the place on the skin where the cotton tip causes a bulge, and withdraw the cotton-tipped applicator. Mark the points on the skin with a pen and connect them. Measure the length and width, and multiply these measurements to calculate the overall undermined estimate.
- Wound depth is defined as the distance from the visible skin surface edge to the wound bed. Insert a cotton-tipped applicator perpendicular to the wound edges (around the corners of length and breadth). Then place

applicator stick along a centimeter-ruled edge. Record for each of the four positions that is extreme corners of length and breadth.

- Measuring the wound volume
  - *Filling method*: Wound volume can be measured by filling the wound with a measured amount of normal saline from a syringe and making the fluid level with that of margin of a wound. Wounds can be positioned horizontally so the liquid does not spill out. Disadvantage is that the amount of fluid absorbed by wound tissue or left in the wound cannot be measured.
  - *Mold method*: Jeltrate is an alginate hydrocolloid which takes its own shape after pouring. By pouring this rapidly setting hydrocolloid into the wound, a mold of the wound is made.

Tracing Film Method and Graph Plotting

Tracing show changes in the shape but not in depth of an ulcer. A sterile glove (two layers of cellophane) is placed over the wound and a tracing is made on the top layer. The underneath layer is thrown away and the tracing is transferred to graph paper for easy comparison. The tracing should be taken at the first attendance and at regular intervals thereafter.

Photographic Documentation

Wounds will be photographed with a camera that can adjust variations, adjust in lighting, allow close up images.

Sensory Examination

To rule out nerve lesion.

Special test of PVD to be performed to differentiate arterial insufficiency from venous insufficiency (*See* Chapter 8.2).

Measurement of edema also has to be performed.

14.7 PHYSIOTHERAPY MANAGEMENT OF WOUND/ULCER

The management outline is common for all types of wound, and ulcer with here and there little differences!

GENERAL AIMS

- Relieve pain
- Relieve congestion and reduce edema

- Improve general circulation to lower limb
- Soften induration of lower leg especially around ankle area
- Mobilize joints of lower limb especially foot and ankle, and strengthen lower limb muscles especially calf
- Improve condition of skin of lower leg
- Teach home care and management
- To prevent ulcer recurrence.

LOCAL AIMS

- Increase circulation to ulcer to promote healing
- Clear any infection
- Reduce edema and induration around ulcer
- Free adherent ulcer from underlying tissues.

PHYSIOTHERAPY MANAGEMENT

Remove all bandages and dressings, clean wound and cover with gauze swabs during general techniques. The leg is elevated to an angle of 45° at the hip periodically throughout the day to aid venous drainage.

METHODS OF TREATMENT

Soft Tissue Techniques

Deep manipulations are given to the whole limb to reduce edema and congestion, beginning with the thigh and continuing down the limb. Slow, deep kneading (squeezing kneading, if necessary) followed by slow, deep strokes of effleurage. Then progress to picking up and wringing on the thigh. Special attention should be paid to the dorsum of the foot, the region of the tendocalcaneus and behind the malleoli. Thumb kneading over the anterior tibialis muscles, finger or thumb kneading over above areas and deep kneading to the foot followed by deep effleurage can be given.

The region of the ulcer is next treated with finger and thumb kneading to soften the induration, working inwards from the periphery to the edges of the ulcer itself. Care is necessary, if the skin is thin when the techniques must be stationary. Support one side at a time, if the ulcer is very painful.

The ulcer can also be moved from side-to-side, the physiotherapist placing her fingers on one side and her thumbs on the other, to free it from the underlying tissues and improve the circulation. This can be progressed to wringing as the mobility of the tissues improves. Local techniques are better avoided when the ulcer is infected.

Electrical Stimulation

The effects of using electrical stimulation to promote healing chronic wounds have been studied since the 1960s. ES affects various types of cells and their activities by supporting, altering or providing electrical currents to accelerate wound healing.

Electrical stimulation has following effects:

- It augments blood flow there by accelerating phases of wound healing such as decreased inflammation and promote granulation.
- *By galvanotaxis*: It is the mechanism by which an electrical field promotes wound healing by attracting of cells of repair towards anode or cathode. Neutrophils are attracted to the negative pole, if the wound is infected and to the positive pole if not infected. Lymphocytes, keratinocytes, fibroblasts, platelets are attracted towards cathode. Macrophages are attracted towards anode. Electric current triggers Ca channels in fibroblast cell membranes to open. There will be sudden influx of Ca, which induces insulin to bind receptors. This stimulates activity of fibroblast, which results in synthesis of collagen.
- *Effects at cellular level*: Stimulates DNA, collagen synthesis and protein production.
- Increases the tensile strength of the wound scar by activation of myofibroblasts.
- *Remodeling of a scar*: Mast cells regulate remodelling of the wound throughout the healing cycle. An excessive number of mast cells produce a hypertrophic scar or Keloid scar. Reduction of mast cells can be achieved under anode with electrical stimulation.
- *Bactericidal effect*: It increases the amount of leukocytes and phagocytes thereby eliminating bacterial load. Animal and human studies have shown that a direct current has an antibacterial effect. Rowley stated that a current of 1–140 mA inhibits the growth of *Escherichia coli B*, with the effect being attributed mainly to the negative electrode. Other studies also found that direct current (like other types of electrical stimulation) slows the growth of *Staphylococcus aureus, Escherichia coli* and *Pseudomonas aeruginosa*. These effects are commonly seen at the cathode. Also with stimulation there is enhanced perfusion, which leads to increased subcutaneous oxygen thereby inhibiting the growth of the bacteria.
- Reduces edema associated with wound.
- *Reduces wound-related pain*: A painful wound heals more slowly since the body produces adrenalin, which prevents the transportation of oxygen and nutrients to the tissue. When pain is reduced patient is more mobile thereby increases the circulation.

TENS

Two or four small electrodes are placed on both sides of the wound. It is important that the patient has normal sensory of touch. Either high TENS or low TENS is preferred. In high TENS the patient should feel a pleasant tingling sensation. In low TENS, should give light muscle vibrations in the hand without being painful. A common treatment time is 30 minutes, 2 times/day.

High Voltage Pulsed Galvanic Stimulator

High voltage pulsed galvanic stimulator (HVPGS) is the twin peak current with high peak intensity but lower average current and very short pulse duration

developed from interrupted direct current. HVPGS is given for 45 minutes to 1 hour, three to five times a week, till it heals. The active electrode (negative) made up of mettalline gauze can be secured directly over the wound, which is loosely packed with sterile gauze soaked in isotonic saline. The inactive electrode placed 20 cm proximal to wound. Settings are pulse duration are 100 microseconds, peak intensity 150 volt, and pulse frequency 100 Hz. Following treatment the wound has to be redressed again in the manner consistent with the condition of the wound.

Therapeutic Direct Current

Direct current (also known as galvanism) is an electrical current that flows in one direction and in wound care; a low intensity (20–1,000 μA) direct current is used to avoid damaging healthy tissue. It appears that higher healing rates will be achieved if a negative electrode is placed directly onto the wound for the first 3 days or until the wound has been debrided. It can be applied by direct method or indirect method. In direct method of application, saline-soaked gauze or a hydrogel dressing applied to the wound bed or cavity to enhance electrical conductivity under the treatment electrode. In the indirect method gel electrodes be on both sides of the wound and interface with the periwound skin.

Iontophoresis

Iontophoresis is the introduction of various ions into the skin by means of electricity. In order to drive the ions into the tissues, a direct (Galvanic) current needs to be employed. The electrical part of the iontophoresis uses a DC that is positively or negatively charged active electrode and an oppositely charged dispersive electrode. The medication is then placed on the active electrode, so that the active electrode has the same charge as the medication. When the electrodes are placed on the skin and activated, the charge from the electrode will drive the medication away from the electrode and into the area being treated. This electrical charge will actually push the medication through the skin and into the tissue to be treated. This treatment helps in decreasing the pain, improving circulation to the affected area and enhances healing of the wound. Iontophoresis is commonly used in the treatment of infected wound and for the destruction of granulation tissue. The common ions used are zinc ion and copper ion. The ions react with the tissue protein to form zinc albuminate, which forms a filling over the surface and adhere to underlying tissue. This coat prevents the entry of bacteria.

Microcurrent Electrical Neuromuscular Stimulator

Microcurrents are low voltage current in microampere range pulsating between 0.1 and 680 Hz. The current is too low or weak to produce muscle contraction or to block pain. MENS also accelerate wound healing (in all phases) by the effect is in cellular level.

Continuous Short Wave Diathermy and Pulsed Electromagnetic Energy

Short wave diathermies both continuous and pulsed have been used successfully to treat chronic open wounds, facilitating progress from one phase of wound healing to the next. Thermal and nonthermal effects are used in the management of wounds. Wound sites treated with diathermy have demonstrated increased fibroblast proliferation, collagen formation, tissue perfusion and metabolic rate. Pulsed electromagnetic energy (PEME) is the interrupted high frequency currents. Since, it is interrupted it produces more of non-thermal effect than thermal effect. With a non-thermal effect there will be increased organization of connective tissue and growth of epithelial tissue thus promoting healing. A pulse duration of 65 microseconds set at a frequency of 400 pulses per minute (ppm) given for up to 30 minutes daily would be suitable for treating wounds. Treatment is usually delivered without touching the skin. Wound with a cause of arterial insufficiency is not good candidate for this method as they will not be able to dissipate heat to avoid burns.

Ultrasound

Ultrasound (US) may have effects through both thermal and nonthermal properties; for example, effects on the remodeling phase is a thermal effect and changing cell membrane permeability is a nonthermal effect. The application of therapeutic ultrasound is not only to treat pain, but also to accelerate the healing process. It stimulates cell activity, accelerate process such as inflammation. In inflammatory phase, it helps in degranulation of mast cells, which releases histamine thereby attracting neutrophils. In the proliferative phase, it stimulates the fibroblasts to release collagen and myofibroblasts contract to pull the edges together. In remodeling phase, it increases the tensile strength of the scar. In venous ulcers, it softens the induration and increase the vascularity in the surrounding tissues. US can be applied to wound bed directly or to the periwound area. Standard procedure for the application of US to the wound bed is to cover the wound with a sheet of hydrogel or an application of amorphous hydrogel. US is then delivered with a hand-held applicator. To apply US to periwound area, transmission gel or coupling cream is applied to this area and treats from this region. The 3 MHZ head using a low dosage, e.g. 0.25–0.5 W/cm^2 is applied for 5–10 min. A pulsed beam is used if the area is large. The dosage can be increased if the area covered is small, but a continuous beam is used if the area is large. The dosage can be increased up to 1.0 W/cm^2 for chronic indurated areas in the lower leg. Ultrasound is contraindicated in the presence of superficial or deep venous thrombosis.

Ultraviolet Radiation

Ultraviolet radiation (UV) has cutaneous and bactericidal effects that include increase blood flow, enhanced granulation tissue formation, destruction of bacteria and thickening of the stratum corneum. If infection control is the

primary therapeutic goal, a UVC device should be used. UVC has been found effective in treatment of methicillin-resistant *Staphylococcus aureus* (MRSA). UVC or very low exposure to a UVA or B light source with the induction of a very minimal skin erythema [minimal erythemal dose (MED)] that appears in 4–6 hours, but resolves within 24 hours is used for wound healing. The treatment is typically delivered to a clean wound with dressings removed, using a UVB or UVC lamp. UV-resistant ointments such as petrolatum jelly, cellophane filters or UVIOL may also be used to protect the periwound area that is immediately adjacent to the wound bed. A treatment time of 30–60 seconds, but no more than 180 seconds administered once daily for 1 week. UV treatment is indicated in nonhealing wounds, necrotic wounds and purulent or infected wounds. The ultraviolet rays are given to stimulate the circulation in case of indolent ulcer. Absorption of the rays produces hyperemia in the congested area and produces an increased exudate. There should be distance of one inch from UV energy source and wound bed. An E4 dose is given for infected ulcer, E3 for indolent ulcer, E2 for deep ulcer and E1 for shallow ulcer can be administered. If the ulcer base becomes pink and vascular dosage can brought down to the next level. An E1 or E0 dose is given to the edge and surrounding area of the ulcer.

Cold Laser Therapy or Low-level Infrared Laser

Low energy laser treatment uses light in infrared spectrum is used for the management of the wound. Biological effects of laser include increased cellular proliferation and differentiation (muscle and fibroblast), increased mitochondrial production of ATP, increased collagen synthesis, increased activity of T and B lymphocytes including binding of pathogens, increased macrophage activity. Skin cells exposed to laser grow five times faster. It also increases the circulation and reduces pain by increasing the release of nitric oxide into microcirculation. It augments wound healing and reversing the symptoms of peripheral neuropathy in individuals with diabetes. Laser therapy can be given to both acute and chronic wounds, slow or nonhealing wounds and infected wounds. Laser therapy can be given to wound surface along with a transparent film and wound periphery with direct contact. The energy dosage should be 1 J/cm^2, which is less than the normal 2–4 J/cm^2 because there is no skin. This is achieved by applying the probe at 30 mW for 33s. A higher power for a shorter time is more effective. The probe is held at 90° to the wound just off the surface of the ulcer. Treatment should not be administered more than once per day.

Ionozone Therapy

This is the production of steam, which is ionized, by being passed over a mercury vapor arc, into a mixture of ionized water, ozone and oxygen. It is applied at approximately 35 cm from the ulcer and surrounding area for 10–20 min. This will reduce pain, overcome infection and promote healing. The steam is directed horizontally with the patient appropriately positioned. It is useful where the patient cannot be positioned satisfactorily for screening for

treatment with ultraviolet rays. All grease should be removed from the ulcer and surrounding areas. If the surrounding skin is thin, the area should be screened with a waterproof material or the distance should be increased up to 50 cm. The treatment is applied daily to infected ulcers and reduced to two or three times a week as healing occurs.

Electroionizing Radiation Therapy

This induces ionization of the air in a limited area. In association with the ionization there is a discharge current between the high voltage electrode and the part of the body being treated also accompanied by a magnetic field, which is induced in the surrounding areas. It has an analgesic effects, edema reduction, microcirculation improvement and angiogenesis stimulation.

Polarized Light Therapy

Healing of venous ulcer achieved with the help of equipment manufactured by Zepter-Bioptron. The features of light generated by this apparatus are polarised, incoherent, low level and polychromatic. Different wavelengths of light trigger specific biological and cellular reactions. Light is absorbed by living tissues increasing the level of ATP, which improves cell metabolism. This results in increased cellular energy, increased microcirculation and cell activity, increased production of collagen and elastin, reduced swelling and inflammation. All cells and body systems react to BIOPTRON light, which promotes cell activation, thus accelerating the healing process. Capillaries start to proliferate as buds, penetrate the wound, make anastomosis and create a rich circulatory network in granulation tissue.

Compression Therapy

Compression therapy in venous ulcer

The purpose of this therapy is to reduce venous hypertension. The pressure exerted by the bandage must be strong enough to equal or exceed the ambulatory venous hypertension during walking. The transmural pressure is reduced, which then reduces filtration and favors fluid reabsorption. The effects of compression in venous insufficiency are reduction of vein volume and increase of venous flow velocity, reduction/elimination of both superficial and deep venous reflux, improvement of the muscle pumping and an increase of ejection fraction, reduction of ambulatory venous hypertension, increase of lymphatic drainage, reduction of edema, shifting of blood volume into the central compartment and improvement of microcirculation. The inelastic bandages are preferred than elastic bandages as it produces high pressure during walking compared to resting pressure. As the effects of compression are much greater during movement, the patient must be encouraged to walk.

Compression therapy in mixed ulcers

Inelastic bandages are preferred in mixed ulcers, if arterial damage is minimal. Thicker padding layers are placed to protect projecting bones and tendons.

In the case of a significant arterial disease, the pressure exerted by the bandage must be reduced to prevent possible ischemic damage.

Compression therapy in arterial ulcers

Rationale of the therapy is to limit formation of edema and improve arterial flow which is induced by the reduction of venous pressure and the increase of the arteriovenous pressure gradient. If the ABPI is between 0.5 and 0.8 an inelastic bandage must be applied, with a low resting pressure. However, the pressure must never exceed the arterial pressure. When the ABPI is lower than 0.5, never apply a bandage and refer the patient to a vascular surgeon. If in the patient with severe arterial disease revascularization is not feasible or has not been successful, intermittent pneumatic compression can be indicated.

Compression therapy in lymphatic ulcers

Compression therapy helps in reduction of edema, remodeling of leg, prevention of skin lesions. An inelastic, multilayer and multicomponent bandage is indicated. The pressure range must be strong to very strong considering the fast pressure drop due to the edema reduction. The pressure level should be lowered in case of usual co-morbidities like, for instance, arterial disease. Special care must be taken in:

- Modeling of the shape of the leg by filling the skin folds and adding supplementary protection to prevent the bandage from slipping.
- Protection of the skin (often very fragile in these patients), also with use of emollients and supplementary protection at high friction points, for instance Achilles or pretibial tendon, and the sole of the foot.

Stockings

Stockings are mainly prescribed for venous ulcers. Compression stockings enhance the muscle pump and aids reduction of edema round the ulcer and in the limb. Pressure is applied such a way that it reduces from distal to proximal increases venous blood flow and prevents dilatation of the leg veins. A sorbo pad or gauze compress is applied over the dressing, of a size corresponding to the ulcer and the edematous area round it. Wool or felt padding is placed in the grooves behind the malleoli and round the lower leg and foot. It is kept in position by a gauze bandage. Over this an elastic bandage, tubigrip or elastic stocking is applied from the metatarsals heads to the tibial tubercle.

Exercises

Active exercises of the ankle, subtalar and midtarsal joints are essential to improve venous circulation and mobilize the joints. The exercises should be carried out with the elastic support removed to emphasize joint mobility and against the resistance of the support to increase circulation and muscle strength especially the calf muscles. Re-education of walking with emphasis on the 'push-off' must be given. Functional activities, which particularly work the ankle should be practised, for example:

- A treadle machine
- Cycling

- Foot power loom
- Walking a dog.

If the joints of the knee and foot do not regain full range with active exercises, mobilizations (passive oscillatory techniques), both physiological and accessory movements may be applied. The exercises must be carefully taught and explained to the patient who must practise them frequently throughout day.

Pneumatic Compression Therapy (In Case of Venous Ulcers)

A double-layered plastic sleeve, which may have zip or velcro fastenings is applied to the lower limb. It can cover dressings and provides intermittent or sequential compression where the ankle, knee and thigh are compressed in turn. This is followed by a rest period for approximately 1 minute. Pressure can be varied up to 100 mm Hg, but is normally between 35 and 55 mm Hg. Some machines blow cool air over the leg to make it more comfortable. The sleeves are worn up to 24 hours per day. The veins are compressed for relatively short periods of time, which greatly reduces venous stasis, improves venous circulation and promotes healing. Due to the mechanical compression and relaxation there is reduction in edema.

Negative Pressure Wound Therapy (NPWT) or Vacuum-assisted Closure (VAC) (In Case of Arterial Ulcers)

It is the process of applying negative pressure to the wound site via foam or gauze dressing, tubing and vacuum device. A foam dressing gauze is placed into the wound cavity. An airtight thin film drape is sealed over the foam/gauze, and a tubing pad is applied over a hole cut into the drape. Controlled negative pressure, typically 125 mm Hg below ambient pressure, is applied and distributed through the porous foam over the entire wound surface. Excessive fluid and debris are drained into the canister. This enhances granulation tissue formation, promote wound edge approximation, promote moist wound healing environment, increased blood flow, enhancement of epithelial migration, wound contraction, remove edema from wounds, clearance of bacteria and increased blood flow thereby improving oxygen levels in the wound. An open cell foam dressing is placed in the wound and a suction tube is connected from the foam to a portable pump. Initially negative pressure is applied continuously via portable pump system. Later pump is programed to apply pressure intermittently. NPWT should not be used in the presence of uncontrolled hemorrhage, blood dyscrasia, coagulopathy and untreated osteomyelitis.

15 Scar

15.1 DEFINITION, TYPES AND MANAGEMENT

DEFINITION

Scar is the area of fibrous tissues that replaces the normal skin after an injury. The longer the healing time, the more likely it is that a wound will form scar tissue.

CLASSIFICATION

- *According to the elevation and the extension*:
  - *Normal tropic scar*: It is the visible scar of same height as that of surrounding area.
  - *Hypertrophic scar*: It is a raised, red, rigid scar that does not extend beyond the wound boundaries and fade with time.
  - *Keloid scar*: It is a raised scar that extends beyond the original wound boundaries. They make three months or even years to develop. Do not regress with time following injury.

    Types of keloid:
    - *Acne keloid*: Folliculitis in presence of hair fragments
    - *Ornamental keloid*: Due to wearing of ornaments
    - *Butterfly keloid*: Due to varying tension exerted on the scars by breast

  Table 15.1.1 differentiates keloid scar from hypertrophic scar.
- *According to the type of trauma*:
  - *Surgical scar*: After a surgery
  - *Non-surgical scar*: Due to wound, boils, carbuncles, acne, etc.
- *According to the appearance of the scar*:
  - *Mature scar*: Flat/soft/supple scar, in which the color matches that of the surrounding skin.
  - *Immature scar*: Red raised and hard occasionally itchy, and painful and lack of suppleness.
  - *Unstable scar*: Over area of stress.

Table 15.1.1 Difference between hypertrophic scar and keloid scar

Hypertrophic scar	*Keloid scar*
Not extend beyond the wound boundaries	Extend beyond the wound boundaries
Red, rigid, raised	Pink to purple
Develops quickly	Years to develop
Fade with time	Do not regress with time
Not painful	Painful and itchy
Histopathology reveals that nodules pattern combination of cells and collagen	Hyalinized collagen bundles called as keloid collagen with mucinous ground substance
Collagen fibers arranged parallel to long axis of scars	Collagen fibers arranged in haphazardous pattern
More likely at the shoulder, upper arm, upper back, dorsal feet and buttocks	Most commonly appear somewhere between the ears and the waist or from the elbow to the shoulder

PATHOPHYSIOLOGY

Fibroblasts are the origin for scar tissue. Fibroblasts produce elastin and collagen. The ratio of elastic fibers to collagen is less in scar compared to normal skin. Elastic fibers provide elasticity and flexibility. Collagen fibers mainly provide strength, but also afford some flexibility within the normal dermis. Collagen in scars is produced at a much greater rate than in normal skin. In all healing wounds, collagen deposition strengthens the wound site, and collagen degradation remodels the wound. When wounds demonstrate an imbalance between the rate of collagen deposition and degradation such that the rate of collagen production exceeds the rate of degradation, a scar that is raised and thick—that is, either a hypertrophic or a keloid scar forms. As a scar actively forms, it lacks suppleness and also it is red and raised. During this 'phase' of scarring, the scar is commonly referred to as *immature scar*. As the scar matures in due course, the redness fades, the scar levels out to some degree and the scar tissue softens. It commonly takes 6–18 months for a scar to mature.

Factors, which increases scar formation:

- Depth of the wound
- Highly pigmented skin
- Increased skin tension
- Young people compared to elder people
- Genetic predisposition.

MEDICAL AND SURGICAL MANAGEMENT

- Vitamin E application
- *Steroid injection (hydrocortisone or triamcinolone)*: These are effective because of their capability to increase activity of collagenase in breaking down the scar.

- By chemical peeling
- Needling
- Derma-abrasion
- Collagen injections
- By ablative laser surgery and resurfacing technique—to reduce redness
- *Silicon gel application*: A silicone polymer gel is produced in sheets or pads that are applied directly over a maturing scar. The viscosity of silicone used for scar treatment. The pressure applied by the gel is used for controlling the hypertrophy.
- Tissue expanders, which are silicone balloons surgically implanted in the subcutaneous fat or under the muscle, are injected with saline, and are used to increase the surface area of normal skin adjacent to the scar. This expanded area of skin eventually can be transferred as a flap to cover an excised area of scar. Tissue expansion allows for better matches of skin color, thickness, and texture then do techniques such as grafting.
- *Surgical realignment*: Z-plasty, Y-V plasty and local advancement or rotational flaps are surgical techniques used to realign or replace scar and break-up tension lines.
- *Excision*: Larger scars may be excised with special equipment and a graft placed to cover the wound.
- *Serial excision or segmental scar reduction*: This is achieved by excising a central portion of the scar and primarily closing the wound. This procedure is then replicated over a period of several months until the entire scar has been removed.

COMPLICATIONS

- Cosmetic deformity results in social isolation, which reduces self-esteem and quality of life.
- *Functional deformity*: Reduced mobility, if the scar is formed over a joint. Contracture can result, if ligament or capsule is shortened.
- Sensory loss is common
- Sensitiveness to cold
- Itching.

15.2 ASSESSMENT AND MANAGEMENT OF SCAR

CONSEQUENCES

Scar tissue gives rise to:

- Pain which can be caused by
  - Nerve tissue becoming involved in the scar.
  - Venous congestion in deep scars.
  - Traction on the neighboring structures when the scar is adherent.

- Limitation of movement, which arises when the scar is over a joint line.
- Impaired blood supply when the scar constricts blood vessels.

ASSESSMENT

Site

- Palm of the hand following the release of Dupuytren's contractures
- Knee, elbow and wrist surgery
- Ankle after internally fixed fractures
- Hip surgeries
- Burn and skin graft.

Subjective Assessment

- *Body image*: Perception attitude (visual and tactile contribution)
- Restricted range of motion (ROM)
- Paresthesia or abnormal sensation distal to the scar.

Objective Assessment

Observation

Color: An immature is hypervascular and red in color. A mature scar returns to a normal color. Sometimes, a matured scar can become hypopigmented or hyperpigmented.

Contour: It can be raised, flat and depressed. The height of a scar provides information about the level of hypertrophy of the scar. A scar that is raised above the plane of the normal adjacent skin demonstrates hypertrophy. The scar will not necessarily flatten as it matures, without some interventions.

Texture: Normal, indurated, tropic, rough or uneven. The texture of the scar may also indicate hypertrophy. As scar tissue is being actively deposited, if it becomes hypertrophied to any degree, the texture of the scar will deviate from that of the normal surrounding skin.

Margin: It can be distinct or indistinct. The normal tissues blend into the wound bed in case of indistinct and diffuse edges.

Size/Location, number to be noted

Palpation

- Hard or soft
- *Tenderness*: Tender or nontender
- *Suppleness*: A scar can be supple, if it is flexible with minimal resistance.
- *Adherence*: Whether the scar is adhered to adjacent tissue.
- *Pliable*: To assess the pliability of a scar, simply pinch it. A scar that is not pliable will be difficult to pinch up between your fingers, because of the stiffness of the tissue. Also, a scar that is not pliable will typically move as a unit when manipulated. A mature scar is more pliable than immature scar.

Examination

- Length of the scar
- Range of motion (ROM) of nearby joints to find out whether the scar is restricting the ROM or not
- Area (length and breadth)
- ROM (active, passive)
- End feel
- Sensation
- *Functional assessment*: Vancouver scar scale.

Management

This varies according to the age of the scar.

Problem List

Recently healed scar: Up to 3 weeks

- Pain
- Loss of joint movement
- To prevent stiffness.

Aims

- Prevent contractures and loss of joint movement
- Mobilize the scar.

Adherent scar: Over 3 weeks

- Restricted ROM
- Adhesions
- Contractures.

Aims

- To mobilize the scar
- To stretch adhesions and contractures
- To regain normal function.

Management

- *Massage*: Massage should be useful in mobilizing superficial tissues by loosening the adhesions of scar to the tissue. Benefits of massage may include lubrication of the scar to prevent drying and cracking of the skin, a decrease in reported pruritus and the psychologic benefits of touch. Aggressive massage of early forming scar tissue should be avoided, because it may cause blisters or skin breakdown:
  - Friction massage (transverse friction massage) can be applied to lubricate the scar in case where tissue is tough and thick.
  - Stroking round and toward the scar with thumb.
  - Thumb kneading on one side of the scar while the other side is supported avoiding stretching, which could split the fibrin.
  - Picking up and wringing between thumb and index finger and skin rolling are useful for mobilizing the scar.

- *Ultrasound*: Applied over the scar, this increases tissue length and the mechanical movement of tissues make the scar tissue more pliable. This allows for more effective stretching of contracted scars. It also mobilizes the scar from the underlying tissues. The frequency used should be 3 MHz. An intensity of 1 W/cm^2 for 4 minutes increasing up to 10 minutes has been found to be effective. Pulsed mode is suitable for small scars and continuous for large scars.
- *Paraffin wax bath*: The skin becomes moist and pliable following wax application, which can therefore help to soften adhesions and scars in the skin prior to mobilization and stretching procedures. This improves the condition of the skin and makes it more supple. Also with heat collagen tissue is most effectively stretched.
- *Passive stretching*: Once the scar tissue has been mobilized and lengthened with massage and ultrasound passive stretching may be applied, if the scar is near a joint. A slow continuous stretch may be used or an oscillatory technique may be applied or (often) a combination of both is effective.
- *Passive range of motion*: After stretching, passive range of motion can be given to maintain the range of motion. Passive range of motion exercise is indicated in case of weakness or paralysis or when the patient is otherwise cognitively unable to participate in a prescribed active exercise program. Passive exercise may also be indicated when a wound is acute. During passive exercise, avoid overstretching the scar or exceeding a patient's pain tolerance. Aggressive stretching might lead to heterotrophic ossification. Passive exercise should progress to active assisted or active exercise as soon as possible.
- *Active exercise*: It is the preferred method of exercise in treating scar. This type of exercise allows a patient to control the extent and amount of stretch placed on a scar. Active exercise will also help to overcome any loss of strength or endurance associated with varying levels of muscle disuse sometimes associated with injuries that lead to scar formation. An active exercise program should be prescribed and monitored by a physical therapist. Active exercise should follow passive stretching and ROM, and the patient should continue with this at home. Active exercise can be given to move the joints through full range without stretching the scar transversely.
- *Active-assisted exercise*: Encourage patients to complete as much of the motion as they can by themselves; then apply additional stretch to maximize tissue elongation. Weights may be used to enhance a stretch. The patient may also provide assistance to active motion by using equipment such as reciprocal pulleys.
- *Directed functional exercise*: Development of scar results in reduced strength of a muscle in particular range of motion. This reduced strength and range of motion results in functional impairment. So, the exercises which are appropriate for a particular age improves motor skills and confidence in daily activities.
- *Splints*: Splints are indicated for the positioning of a scar to avoid deformation or to maintain or increase the stretch on a scar. A conforming splint is custom-fit to a patient and matches the patient's anatomic shape, preferred for controlling scar formation. Static splints are commonly used in the early

phases of scar formation. Dynamic splint can be used to continue a gentle force to scar, thus providing an extended period of stretching. Serial splints (or casts) might be used, if a scar is particularly difficult to stretch (e.g. a contracted scar).

- *Whirlpool baths* which soften scar tissue may be applied by immersing the affected part in a hot bath through which an air stream is passed to agitate the water molecules.
- *Positioning*: Positioning may be used to sustain tissue elongation to counter scar contraction. Pillows, splints or shoes can be used to position the extremities. Wrist is placed in a functional position, and ankle should be positioned in neutral.
- *Pressure garments*: Pressure therapy is typically recommended when a wound takes longer than 14 days to heal (as longer the wound heals more the chance of scar to develop). Early pressure can decrease edema formation and facilitate wound healing. This may also have some effect on eventual scarring. Pressure to a scar can be applied through the use of elastic wraps, self-adherent stretch wraps or elasticized cotton tubular bandages and customized pressure garments. 'Inserts' such as foam, thermoplastics and rubberized material can also be used to augment pressure provided by a well-fitting pressure garment in areas such as the webspaces of the hand.

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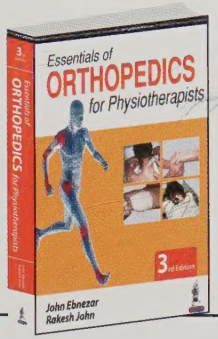

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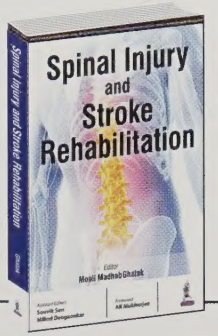

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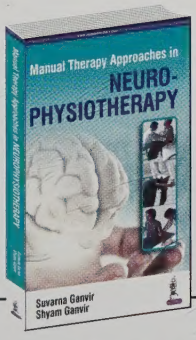

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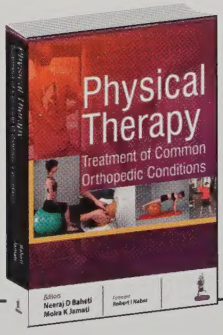

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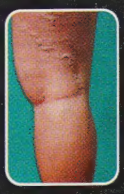

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