



Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

The Venous System

Discussion paper prepared for

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WSIAT literature search reviewed by Dr. J. Duff in 2010, who is of the opinion that this paper still provides a balanced overview of the medical knowledge in this area.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

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THE VENOUS SYSTEM

The venous system consists of a network of vessels of varying size whose function is to return deoxygenated blood back to the heart and to act as a blood reservoir. Some 75% of the blood volume is contained within the venous system.

Veins differ from arteries in the following respects:

- They are thin walled. They have the same 3 layers as arteries but the muscle layer is much reduced. The major superficial veins of the extremities have thicker walls than the deep veins.
- The venous system is a low-pressure system and flow is much less dependent on the pumping action of the heart.
- Veins are normally only partially filled with blood. They have 3X the cross-sectional area of corresponding arteries.
- Veins of the extremities have valves. These are thin delicate bicuspid structures constructed of fibrous and elastic tissue lined with endothelium. At the site of each valve the vein is dilated creating a sinus space around the valve which facilitates the opening and closing of the valve. The purpose of valves is to break up the column of blood in the vein and ensure one-way flow.

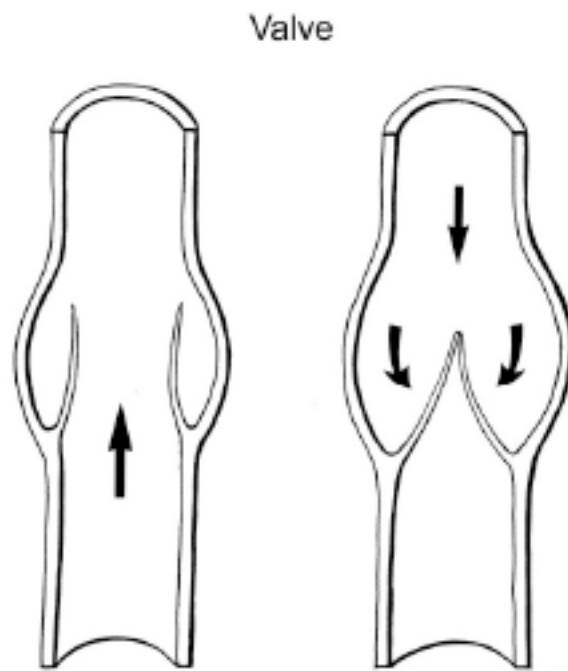


Diagram 1

Diagram 1 - Valve

Anatomy

The venous system in the leg is divided into superficial veins, deep veins, perforating veins, and intramuscular venous sinuses (Diagram #2).

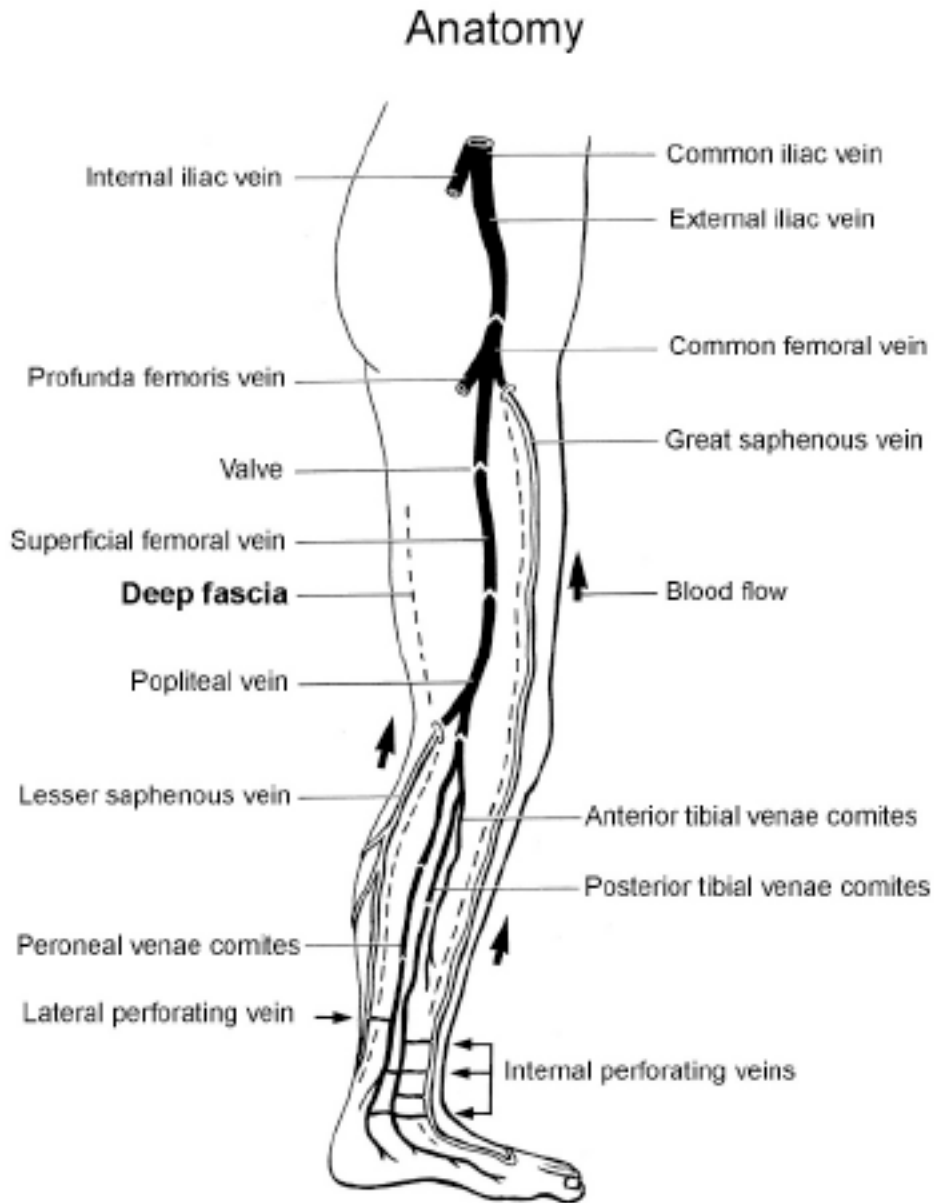


Diagram 2

Diagram 2 - Anatomy

The **superficial veins** lie in the subcutaneous fatty layer of the body just beneath the skin and superficial to the deep fascia enveloping the body musculature. The principal veins in the legs are the great and lesser saphenous veins and their tributaries; in the arms they are the basilic and cephalic veins and their tributaries. The **deep veins** accompany arteries and bear the same name as the arteries they parallel. It is common in the extremities for there to be two or more veins accompanying a small- to medium-sized artery. The **perforating veins** penetrate the deep fascia and connect the superficial veins to the deep veins. Those along the inner (medial) side of the lower leg play a major role in the pathogenesis of the “postphlebotic leg”. The **intra-muscular sinusoidal veins** are large, very thin walled, valveless veins within skeletal muscle. They connect directly with the deep veins.

Physiology

The venous system of vessels conducts blood back to the heart. Normal venous flow is dependent on four factors: the heart (dynamics/spontaneous flow), respiration (phasic flow), the venous pump and the valves.

Dynamic Flow: Flow in the arterial system is dependent on the pumping action of the heart and the elasticity and muscular activity of the arteries. In contrast, the veins, except for the major superficial veins, are thin walled with paucity of muscle; are designed for distention; and play an insignificant role in directly facilitating flow. Most of the force of the pulsatile flow produced by the pumping heart is lost as blood flows from the arteries through the vast network of capillaries (one cell layer micro-vessels where selective permeability allows the exchange of O₂ and nutrients with the tissues). What pulsatile flow filters through (dynamic flow) is of low pressure (15mmHg). Nevertheless, it is sufficient to produce a significant pressure gradient with the right side of the heart where the venous pressure is 0.

Phasic Flow: is the effect of respiration on normal venous flow. In the arms and neck, flow towards the heart increases during inspiration due to the negative intra-thoracic pressure produced. The opposite is true in the legs. With inspiration the diaphragm descends increasing intra-abdominal pressure slowing flow. Flow is somewhat increased with expiration with reduced intra-abdominal pressure.

The “**Muscle Pump**”: the muscle pump mechanism is most highly developed in the calf muscles. Large venous sinusoids located in these muscles act as a bellows and the contracting muscle the force emptying the bellows.

Contractions of the calf muscles can produce a pressure in excess of 200 mmHg. This is sufficient pressure to empty the blood out of the sinusoids into the deep veins. The deep veins in turn are subject to a similar compressing force because of the strong fascial investment about the muscle compartment in which they are contained. As a result blood is pumped towards the heart with each muscle contraction (Diagram #3).

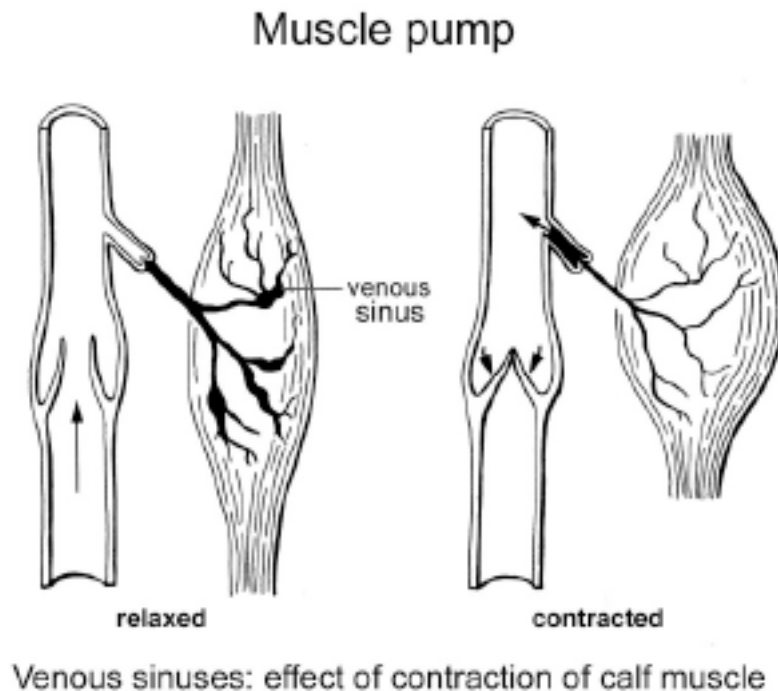


Diagram 3

Diagram 3 - Venous sinuses: effect of contraction of calf muscle

Valves: valves are structured so that flow is always towards the heart and flow from superficial to deep veins. Without valves there would be one continuous column of blood from heart to ankle when an individual stood. By preventing reflux the valves complement the muscle pump in returning blood to the heart.

Pathology

The most common pathological conditions in the venous system occur in the legs, and include valve incompetence and venous obstruction.

Venous obstruction is most commonly due to venous thrombosis, but may also result from vein compression (tumors, cervical rib, fractures, hematomas, arterial aneurysms etc). Factors producing venous thrombosis are vein injury, flow stasis (inactive muscle pump from lack of motion, bed confinement or paralysis), blood hypercoagulability (in post operative patient or an intrinsic coagulation disorder) and a combination of above as may occur in multiple trauma patients.

Valve incompetence may be due to congenital valve defect or develop as a complication of venous thrombosis and venous hypertension. Congenital valve incompetence occurs in the superficial veins and the perforators. Deep vein valve incompetence is considered a complication of previous deep vein thrombosis regardless that a history of deep vein thrombosis is obtained in less than 50% of patients.

Incompetence results in reflux with increased venous pressure in the segment of vein(s) below the incompetent valves when the individual stands (a condition referred to as “venous insufficiency”). Clinical manifestations depend on which venous system(s) (superficial, deep or perforators) are involved and the number of valves incompetent. When the valves of the deep veins are affected the ensuing venous hypertension produces backpressure in the capillaries causing leakage of fluid into the tissues with leg edema (swelling).

Venous Diseases

Varicose Veins

A varicose vein is dilated, elongated and tortuous. Most commonly affected are the saphenous veins in the legs (90% involve great saphenous system). The cause is venous hypertension resulting from valve incompetence. They may be primary or secondary.

Primary (saphenous) varicose veins

This is a congenital condition. Varices result from congenital weakness of the valve structure and possibly also a congenital weakness in the vein wall. There is a family history in 75% of patients. By themselves primary varicose veins produce few symptoms. Cosmetic appearance is the major complaint. A common symptom is heaviness of the legs towards the end of the day, particularly for those whose occupation requires considerable walking and standing. There should be little to no ankle or leg swelling.

Secondary Varicose Veins

Secondary varices are a sequela of either deep vein obstruction, incompetent deep vein valves or a combination of both. In each case the resulting venous hypertension renders the perforating veins incompetent allowing unrestricted back flow from deep to superficial veins. The superficial veins not being structured to withstand a high venous pressure become dilated and elongated, forming secondary varices.

Thrombophlebitis, Venous Thrombosis

When a thrombus (blood clot) obstructs a vein it sets up a sterile inflammatory reaction in the vein wall and the surrounding tissue. This condition is known as “**thrombophlebitis**”. When a loosely attached thrombus develops in a vein and is not obstructing, it produces no reaction in the vein wall. This condition is known as “**phlebothrombosis**.”

Superficial Thrombophlebitis:

Superficial thrombophlebitis of the great saphenous vein presents with a typical clinical picture. It begins with sudden development of pain and tenderness along the course of the section of vein involved. The skin over the vein is red and the adjacent tissue swollen. With resolution of the inflammation (2-3 weeks if untreated) the thrombosed vein can be felt as a cord-like structure beneath the skin.

The cause of the phlebitis and the reason that the great saphenous vein is usually targeted remains unknown. Local trauma and/or unusual activity may be a precipitating cause in some cases. It can be recurrent, developing in another section of the saphenous vein in the same leg. Superficial phlebitis is more of an annoyance than a serious condition. It resolves rapidly with reduced activity and anti-inflammatory drugs. However, in those cases where the thrombotic process extends to the level of the groin, there is a risk of involvement of the common femoral vein and the possibility of pulmonary embolism.

Superficial saphenous vein thrombophlebitis is distinct from thrombosis occurring in a cluster of varices. The latter is common in varicose vein disease. There is no risk of pulmonary embolism. Permanent discolouration of the overlying skin is the main complication. In the arms the common cause of superficial phlebitis is chemical damage to the lining of the vein from intravenous injection. Pulmonary embolism secondary to thrombophlebitis in the arms is rare.

Deep venous thrombosis (D.V.T)

D.V.T. by medical convention refers to thrombosis in the deep veins of the legs. It is a relatively common complication of major surgery, leg fractures and prolonged bed rest. Stasis because of muscle pump inactivity, a hypercoagulable state which is a biological reaction to injury and, local trauma to veins, all play a role.

The clinical presentation is related to site, extent and degree of obstruction produced by the thrombus. Thrombosis limited to the calf veins produces only mild calf soreness and tenderness and minimal, if any, ankle swelling. In contrast acute thrombosis obstructing the femoral and iliac veins results in a grossly swollen, painful, white leg (white because of the extensive edema under the skin). Rarely the extent of the thrombosis will include the entire venous network causing obstruction of arterial circulation. The result is massive leg swelling complicated by manifestations of vascular ischemia and possible gangrene (so called venous gangrene). When deep vein thrombosis does not cause obstruction (i.e. phlebothrombosis) pulmonary embolism may be the first and only clinical manifestation.

Acute D.V.T. is a medical emergency. Anticoagulation (blood thinning) is the prime treatment. It prevents extension of the thrombus. Clot lysing drugs (thrombolytics) are injected transvenously in selected cases. Thrombectomy (surgical removal of clot) has a limited role.

Recurrence of acute D.V.T. is common. Major complications include pulmonary embolism, chronic deep vein insufficiency and the postphlebotic leg.

Pulmonary Embolism

Pulmonary embolism occurs when a piece of clot (thrombus) breaks away from the vein wall, enters the venous flow and passes through the right side of the heart to lodge in the pulmonary artery or one of its branches. Clinical manifestations are determined by the size of the clot and the size of the vessel occluded. The spectrum includes sudden death (occlusion of main pulmonary artery), pleurisy-like symptoms from lung infarction (occlusion of segmental artery and local lung death); shortness of breath and pulmonary insufficiency (occlusion of multiple small vessels from showers of small emboli). Acute D.V.T. is the major source of pulmonary embolism.

Lung scans and pulmonary angiograms confirm the diagnosis. Immediate and full anticoagulation (blood thinning) is basic treatment. Where obstruction of the main pulmonary or major branch is diagnosed thrombolytic agents are

injected directly into the clot by intravenous catheter. Surgical removal of the blood clots is warranted in selected cases. When the use of anticoagulants, is contraindicated a filter is inserted into the inferior vena cava to prevent further clots from reaching the lung.

Chronic Venous Insufficiency and Postphlebotic Leg

Chronic venous insufficiency is an overarching term which includes the various clinical and pathological entities resulting from impaired venous flow and venous hypertension. Chronic venous insufficiency in the deep veins is attributable to acute D.V.T. and its sequelae. The degree of insufficiency initially depends on the fate of the obstructing thrombus. The thrombus may be partially or completely dissolved by lytic enzymes. More commonly it is organized and replaced by fibrous tissue with varying degrees of recanalization (the development of small, valveless channels traversing the length of the organized thrombus). Recanalization produces little improvement in venous flow. Compensatory dilatation of collateral veins (superficial and deep secondary veins which bypass the obstructed area) is more effective in re-establishing the venous flow. The dilated superficial veins which may result are referred to as secondary varicose veins.

D.V.T. has a disastrous effect on the valves of the deep and perforating veins partially due to injury to the valves by the process of thrombosis, and more particularly due to venous hypertension, which produces abnormal dilatation of both the deep and perforating veins rendering their valves incompetent. The result is the inability of the venous pump to provide relief from venous hypertension when the individual stands and walks. As a consequence, the capillaries in the distal part of the legs are exposed to a high pressure (Diagram #4).

The result is increased capillary permeability with leakage of fluids into the tissues producing leg edema (swelling). The concentration of perforators around the ankle and the higher hydrostatic pressure with standing makes this area (the so-called "gaiter area") particularly vulnerable. (Diagram #5)

Pathology of postphlebitic leg

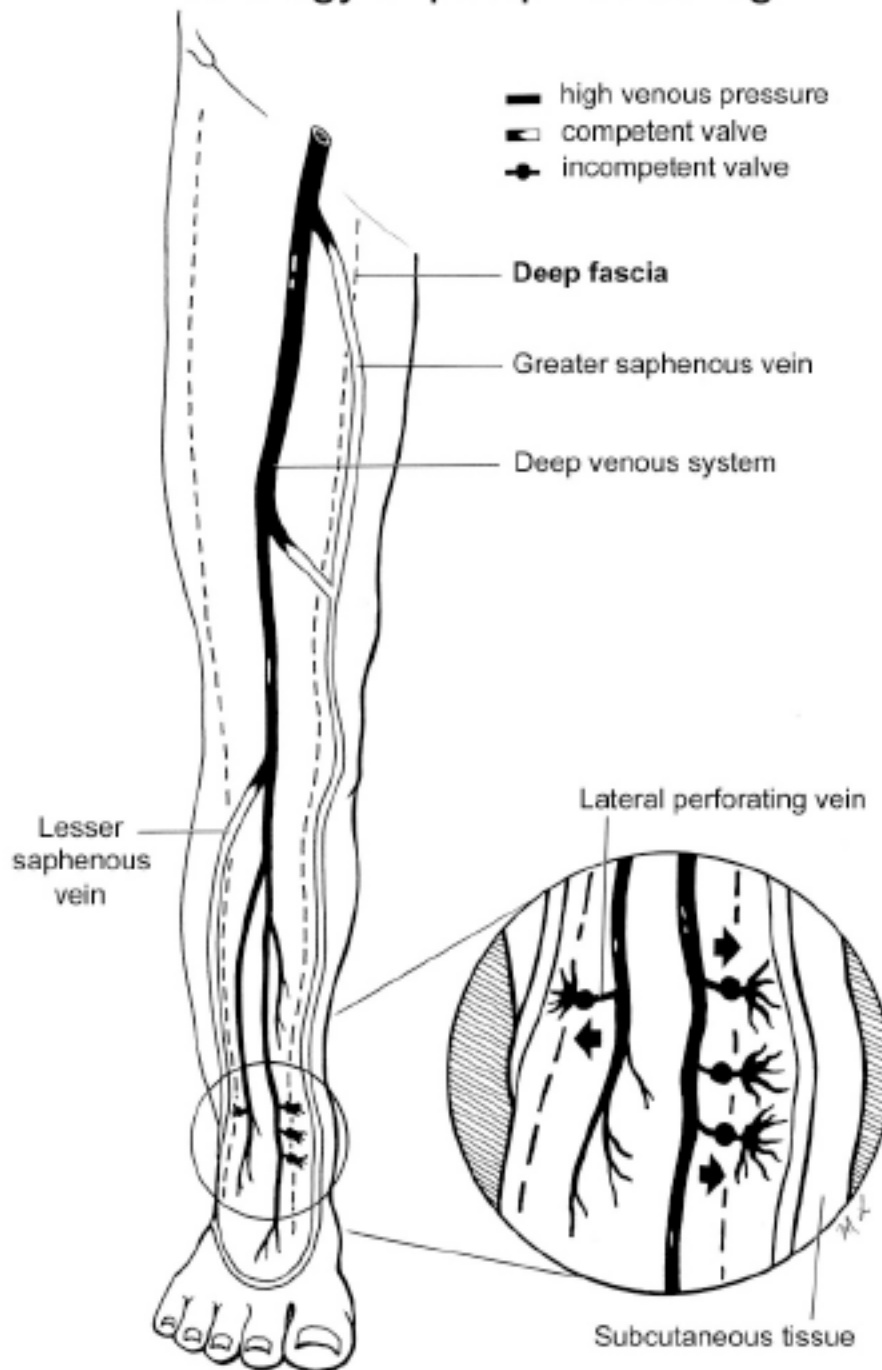


Diagram 4

Diagram 4 - Pathology of postphlebitic leg

Gaiter area

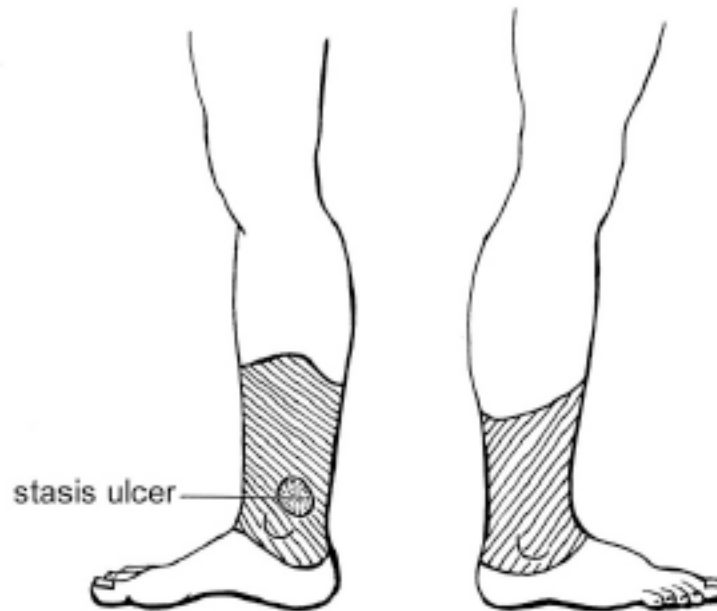


Diagram 5

Diagram 5 - Gaiter area

The combination of incompetent valves and persistent venous hypertension over time causes damage to cutaneous and subcutaneous capillaries, allowing protein rich fluid and red blood cells to escape into the subcutaneous tissue around the ankle. The effect is that the subcutaneous tissue becomes fibrotic, and skin pigmented from the brown discolouration by the iron deposited in the subcutaneous tissue after disintegration of red blood cells. The transmission of oxygen and nutrients to the tissue is subsequently impaired, making the area vulnerable to infection and even minor trauma. The result is the “**postphlebotic leg**”.

The clinical manifestations of the postphlebotic leg include varying degrees of leg swelling (foot is spared); calf firmness and enlargement (due to water in the tissues surrounding muscles); and, most apparent, dermatitis, brown pigmentation, induration and eventually ulceration in the gaiter area. Skin ulceration occurs in up to 50% of postphlebotic limbs after five to 10 years and typically occurs on the inner aspect of the lower leg just above and behind the medial malleolus. All patients suffer heaviness of the leg on standing for any length of time and with modest exercise. The postphlebotic leg presents a significant disability.

Injury to veins

Superficial vein injuries are managed by simple ligation without consequence.

Isolated deep vein injury is uncommon and is usually accompanied by injury to the accompanying artery. Repair of the artery takes priority. The injured vein may be ligated, or repaired, sometimes with graft. A repaired vein may thrombose (because of the low pressure in the venous system) and is a potential source of pulmonary emboli. Edema and the post phlebitic syndrome complicate ligation of major veins in the leg.

Subclavian Thrombosis

Acute thrombosis of the subclavian vein in the arm, so-called “**Effort Thrombosis**” - is a relatively uncommon condition occurring in the young and middle aged. It has a very characteristic clinical presentation, with edema of the arm developing immediately or a few hours after some severe or unusual physical effort in which the arm is fully abducted. Duplex (ultrasound) studies and/or venography (injection of dye into vein under x-ray) confirm the diagnosis. Following treatment (anticoagulation), edema of the arm generally subsides within a few weeks, although a few patients may be left with some arm swelling and aching particularly with activity.

Arterio-Venous Fistula

Traumatic Arterio-Venous fistulae may occur when there is disruption of an adjacent artery and vein, and arterial blood flows into the vein under arterial pressure. The biological effect of fistulae depends on their size and location. The extremely high venous pressure will over time cause incompetence of valves, tissue edema from the backpressure on capillaries and, secondary varices. Large fistulae result in overloading of the venous system and high output heart failure.

Investigation of Venous Disease

Duplex (ultrasound) screening is the prime investigational study. It provides excellent imaging of thrombi and evaluation of venous flow patterns. Venograms are still the gold standard. However, because they are invasive and not without complications including allergy to dye used, venography is indicated only in special circumstances. It is particularly valuable in the diagnosis of venous problems in the major veins of the pelvis and trunk.

Lymphatic System and Lymphedema in the Leg

The lymphatic system is the third vessel system in the body. Its main function is to remove plasma proteins and fluids which have filtered through the capillaries into the tissue spaces. This protein rich fluid is “lymph”.

The lymphatic system has three major components: the terminal lymphatic capillaries, which absorb the lymph from the tissues, the collecting vessels, which transport the lymph (valves ensure one way flow), and, the lymph nodes, which act as mechanical filter, and have an immunological role. Collecting lymphatic vessels closely follow the veins in the legs and eventually discharge their lymph into the venous circulation.

Lymphedema is the swelling of soft tissue, which results from the excessive build up of lymph, which in turn is caused by a malfunctioning of the lymphatic drainage.

Inadequate drainage may be a primary lymphatic defect (congenital malformation) or secondary to another disease (lymph node excision for cancer; blockage of lymph channels from infection; fluid overload that overwhelms the functional capacity of the system). Unlike the swelling in chronic venous insufficiency the swelling in lymphedema does not “pit” (finger indentation left with mild local pressure); it involves the foot and toes; it does not ulcerate; it is not relieved by elevation of the leg (when well developed); and it is painless. Duplex sonography (ultrasound) has proved an excellent tool in diagnosis. It clearly demonstrates dilated lymphatics in the soft tissues of the leg.

Glossary

Aneurysm: Localized dilatation of an artery.

Arteriovenous (A.-V.) Fistula: Connection between an artery and a vein

Blood hypercoagulability: Abnormal increase in blood clotting.

Capillaries (L- capillus - hair): Network of small vessels (8-20 u) joining small arteries (arterioles) and veins (venules) Wall consists of a single layer of cells. Semi permeable to allow exchange of oxygen, fluids, electrolytes, and nutrients with tissues.

Claudication: Muscle cramping with exercise.

Duplex study: Provides a combination of ultrasonic image of vessels plus Doppler measurement of velocity and assessment of characteristics of blood flow.

D.V. T. (Deep venous thrombosis): Clot formation in deep veins

Edema: Abnormal accumulation of fluid in tissue.

Gaiter Area: An area extending circumferentially from just below the ankles to almost halfway up the lower leg (see Diagram #5).

Gangrene: Tissue necrosis (death)

Hematoma: Blood clot in tissue

Incompetence: Valvular reflux.

Inflammation: Is the local response of living tissue to injury.

Intermittent Claudication: Cramping muscle pain brought on by walking a certain distance; relieved by stopping; recurs after walking a similar distance.

Ischemia: Inadequate blood supply to an area of tissue.

Interstitial fluid: The fluid that bathes the cells of the tissues of the body. Resembles blood plasma in its composition. Contains nutrients and waste products from cell metabolism.

Lymph: Protein rich fluid (excess interstitial fluid) containing some lymphocytes, cell debris and waste products from cell metabolism.

Lymphedema: Swelling of soft tissues caused by an accumulation of an excessive quantity of lymph.

Phlebothrombosis: Thrombi resulting from stases in an uninflamed vein, generally the veins of the calves of the legs.

Postphlebitic Leg: A clinical entity in which the skin and subcutaneous tissues around and above the ankle, the “gaiter area”, are pigmented (brown), atrophic and, tightly scarred. When contrasted to the swollen calf above, leads to the descriptive term “chicken leg”.

Pulmonary Embolus: A dislodged thrombus, which passes through the right side of the heart and obstructs the pulmonary artery or one of its branches.

P.V.D. (Peripheral Vascular Disease): Refers to diseases affecting any of the body’s three vessel systems namely arteries, veins or lymphatics.

PAD (Peripheral Arterial Disease): Refers to diseases affecting the only the arterial system.

Rest Pain: Chronic persistent pain in an area (usually a toe) aggravated by lying down and which represents severe ischemia of tissue.

Stasis Ulcer (Venous Ulcer): Ulcerations secondary to chronic deep vein insufficiency and occurring only in the gaiter area.

Stenosis(es): Narrowing of the lumen of a vessel usually artery.

Thrombosis: Is the formation of a solid mass in the vessel lumen by the constituents of streaming blood (aggregate of platelets and fibrin in which red and white cells are trapped).

Thrombophlebitis: Venous thrombosis in which the vein wall is inflamed. Most apparent when superficial veins are involved.

Varicose Veins: An elongated, dilated and often tortuous vein

Venous Insufficiency: Impaired return of venous blood resulting in venous hypertension.

Venous Claudication: Deep bursting-like pain in the leg during exercise. Only relieved with sitting or lying down.

Questions and Answers

1. *What types of injuries are likely to predispose/cause deep vein thrombosis?*

Answer:

Major soft tissue injury to lower limb including fractures. Any major body injury requiring bed rest for prolonged periods and major surgery.

2. *What types of injuries can predispose a worker to varicose veins? How likely is this?*

Answer:

Injuries play no role in the pathogenesis of **primary varicose veins**. They are congenital and in 75% of cases familial. **Secondary varicose veins** are a common sequela of deep vein thrombosis and chronic venous insufficiency. Injuries predisposing to D.V.T. will also predispose to secondary varicose veins.

3. How likely is a “postphlebotic leg” to follow deep vein thrombosis? What factors contribute to this?

Answer:

Most patients (exact percentage cannot be given) who suffer deep vein thrombosis will subsequently (within 5-10 years) develop a postphlebotic leg or manifestations thereof. Prolonged walking, standing or sitting are exacerbating factors.

4. How likely is it that varicose veins are caused or exacerbated by prolonged walking, standing or sitting?

Answer:

Primary varicose veins are **not** caused by prolonged walking, standing or sitting. Prolonged walking does exacerbate varicose veins in the absence of the use of support stockings. Prolonged sitting can precipitate thrombosis in a group of varices, again in the absence of the use of appropriate support stockings.

5. How likely is it that a pulmonary embolus may be related to inactivity following an injury?

Answer:

Very likely. Prolonged bed confinement because of injury puts the patient at risk of silent venous thrombosis and pulmonary embolism. No percentages can be given. Medical and nursing care is designed to reduce the risk as much as possible.