Section B: Background

Glossary

General Terms	
Clinical Practice Guideline (or clinical guideline):	A systematically developed statement, based on the best available evidence, to assist health practitioners making decisions. [1]
Duplex Ultrasound (duplex US):	Sonographic imaging utilises greyscale (B-mode) to visualise the vessel and surrounding structures, combined with colour Doppler or spectral Doppler to assess blood flow in veins or arteries. Both displays appear simultaneously on the same screen (duplex), providing overlapping images for easier interpretation.
Reporting clinician:	For the purpose of this guideline, the reporting clinician is a medical specialist (or a sonographer who is recognised to have a reporting role) who provides a clinical ultrasound report based on the findings of a duplex ultrasound examination of the lower limb veins. The report is typically issued to the medical practitioner(s) who requested the examination.
Sonographer:	An allied health professional who performs ultrasound examinations using ultrasound imaging devices to diagnose, monitor and guide treatments for health conditions. In the context of vascular sonography, this term may also be used by vascular technicians/vascular scientists. In this clinical guideline, a sonographer refers to any health professional, including medical sonographer, vascular surgeon, radiologist or phlebologist, who performs venous insufficiency ultrasound studies.
Vascular care provider:	In this clinical guideline, it refers to a medical or health care worker who may refer patients for a duplex ultrasound examination to investigate chronic venous disease and/or manage patients in relation to their vascular health under relevant legislative requirements. Vascular care providers may include general practitioners, vascular specialists, vascular and endovascular surgeons, phlebologists, interventional radiologists, dermatologists, podiatrists, clinical nurse specialists.
Definition of Venous Terms	
Chronic Venous Disease (CVD):	Any morphological and functional abnormalities of the venous system of long duration manifested either by symptoms and/or signs indicating the need for investigation and/or care. [2]
Chronic Venous Disorder:	Refers to the full spectrum of chronic morphological and functional abnormalities of the venous system. [2]
Chronic Venous Insufficiency (CVI):	A term reserved for advanced chronic venous disease, which is applied to functional abnormalities of the venous system resulting in oedema, skin changes, or venous ulcers. [2]
Pelvic Venous Disorder (PVeD):	The spectrum of symptoms and signs arising from the veins of the pelvis (e.g., gonadal veins, internal iliac veins and their tributaries, and the venous plexuses of the pelvis) and their primary drainage pathways (e.g., left renal vein, iliac veins, and pelvic escape points). [3]
Classification and Assessment Tools	
Aberdeen Varicose Vein Questionnaire (AVVQ):	A questionnaire of 13 questions addressing all elements of chronic venous disease including signs, symptoms, social issues, the effect of varicose veins on daily activities, and the effect of varicose veins from a cosmetic standpoint. [4]
Clinical-Eetiology-Anatomy-Pathophysiology (CEAP) classification:	An internationally accepted standard used clinically for describing patients with chronic venous disorder and to report clinical research findings.[5]
Venous Clinical Severity Score (VCSS):	A score, based on the clinical elements of CEAP (see directly above), provides a progressive ranking of the severity of chronic venous disease, allowing for longitudinal and objective follow-up of a patients' clinical condition. Clinical items include scoring of pain, varicose veins, venous oedema, skin pigmentation, inflammation, induration, and different items of leg ulcers. [4]
Venous Pathology Terms	
Agenesis:	Vascular anomaly in which a blood vessel fails to develop during embryogenesis, resulting in complete absence of a vein or of a segment of a vein. [6]
Aplasia:	The lack of full development of a vein or vein segment. Aplastic vein is not seen on ultrasound imaging. [6]
Atrophy:	A decrease in size or wasting away of a normally developed vein or segment of a vein, following a degenerative process. [6]
Axial reflux:	Uninterrupted retrograde venous flow between the groin and calf. May be confined to the superficial system (superficial axial reflux), to the deep system (deep axial reflux) or a combination of the superficial, deep and perforating systems. [2,7]
Deep vein thrombosis (DVT):	Refers to blood clot (thrombus) formation in one or more of the deep veins in the body.
Developmental venous abnormalities:	Include agenesis, aplasia, hypoplasia, dysplasia, venous aneurysm, venomegalia. [6]
Dysplasia:	A complex developmental abnormality of a vein or of a group of veins that greatly differs from the normal conditions in size, structure, and connections. [6]

Hypoplasia:	The incomplete development of a vein or of a segment of a vein; a calibre 50% of normal values seen on ultrasound indicates hypoplasia. [6]
Klippel-Trenaunay syndrome (KTS):	A rare congenital condition in which blood and or lymph vessels fail to form properly. The three main features are 1) vascular malformations of the capillaries (characterised as port wine stain), venous and lymphatic vessels, 2) varicosities of unusual distribution, particularly lateral venous anomalies and 3) unilateral soft and skeletal tissue hypertrophy (mostly in the lower extremity).
May-Thurner syndrome (MTS):	A condition caused by the specific compression of left common iliac vein by the right common iliac artery and lumbar vertebrae. MTS produces congestive signs and symptoms with a risk of deep vein thrombosis. It is a subvariant of other mechanisms of iliac vein compression, including distended bladder, endometriosis, iliac vein aneurysm and tortuous iliac artery on the ipsilateral side. [2, 4]
Paradoxical Reflux:	Ascending reflux commonly, but not exclusively exhibited in the cranial extension of the small saphenous vein (or Giacomini vein) during muscular systole or distal compression secondary to venous obstruction in the femoropopliteal vein.
Pathologic perforating vein:	Incompetent perforating vein that is located underneath an active or healed ulcer, indicating the need for intervention. [8]
Perforating vein incompetence:	Reversed flow (deep to superficial direction) of abnormal duration in a perforating vein during muscular relaxation or release after distal compression. [9]
Phlebectasia:	Vein dilatation without tortuosity. [6]
Phlebitis:	Inflammation of a vein. Commonly caused by blood clot formation, known as thrombophlebitis, but can also results from infection, injury, or irritation of the vein. [6]
Phlebosclerosis:	An age dependent fibrotic degeneration of one or all three vein wall layers, predominantly the intima, and with or without calcification. It may impair the venous function and contribute to the development of thrombosis. [10,11]
Post-thrombotic syndrome (PTS):	Chronic venous symptoms and/or signs secondary to deep vein thrombosis and its sequelae. [2] Morphologic characteristics include lumen fluctuations of the veins, dilation of collateral veins, partial occlusions of the original lumen and irregular thickening of the venous wall and venous valves. [12]
PREVAIT:	Acronym meaning **PRE**sence of **V**arices (residual or recurrent) **A**fter **I**n**T**ervention. [2]
Residual varices:	Varicose veins which persist after treatment. [2]
Recurrent varices/recurrent veins after surgery (REVAS):	The reappearance of varicose veins in an area previously treated. [2]
Superficial Vein Thrombosis (SVT) or Superficial Thrombophlebitis (STP):	Represents blood clots formation and inflammation in the thrombosed superficial veins characterised by a painful, warm, erythematous, tender, palpable cord-like structure along the course of a superficial vein. [13]
Superficial Venous Insufficiency (SVI):	Is defined as retrograde flow in the superficial veins of abnormal duration.
Segmental reflux:	Localised retrograde venous flow, which may involve the deep or superficial in any combination in the calf or the thigh, but not in continuity from the groin to the calf. [2]
Truncal (aka truncular varicose veins) varicosities:	Varicosities distributed along the great or small saphenous veins and their tributaries. They may be associated with varicose veins of other venous territories such as pelvis and gastrocnemius and soleus muscles or due to isolated incompetent perforating veins. [14]
Varicocoele:	Refers to presence of scrotal varicose veins (varicose veins of the pampiniform plexus).
Varicose veins (synonyms; varix, varices, varicosities, venectasia):	Refers to superficial veins of the leg which are dilated >2 mm in diameter), tortuous, with demonstrated retrograde flow indicating venous incompetence. [15] They are divided into primary and secondary varicose veins. Primary varicose veins form due to vein wall weakness, primary valvular dysfunction, pregnancy, hormonal change and prolonged venous statis). Secondary varicose veins result from disorders of the venous system, such as deep vein thrombosis, superficial venous thrombosis, trauma and iatrogenic arteriovenous fistula. Secondary varicose veins may be totally indistinguishable from primary varicose veins. [13,14]
Venomegalia:	Diffuse dilation of one or more veins with an increased calibre of 50% or more compared to normal calibre. [6]
Venous aneurysm:	A localised saccular or fusiform dilatation of the vein, at least 50% greater size than the normal vein trunk. [2, 6]
Venous compression:	Narrowing or occlusion of the venous lumen as a result of extra-luminal pressure. [2]
Venous obstruction:	Partial or complete blockage to venous flow, which may be due to external compression or intra-luminal thrombus. [2]
Venous reflux:	An abnormal venous state, when venous flow in a vein is retrograde (flows away from the heart) and lasts longer than normal. [2]
Venous Leg Ulcer (VLU):	Full thickness defect of skin that does not heal spontaneously and is sustained by chronic venous disease, most frequently occurring in the ankle region.
Venous thromboembolism:	Blockage to venous blood flow due to a blood clot (thrombus).
Vulval varicosities:	Dilated veins in the labia majora and labia minora.
Treatment related Venous Terms	
ARTE	An overarching term describing ablation-related thrombus extension (including junctional extension) associated with any ablation modality including thermal, foam, mechanochemical, and

	cyanoacrylate ablation. This includes events also described as EHIT, postablation superficial thrombus extension, endovenous glue induced thrombosis, and endovenous foam-induced thrombosis.
Endophlebectomy:	Removal of postthrombotic residue from the venous lumen. [2]
Endovenous glue induced thrombus (EGIT):	Thrombus extension into the deep veins following cyanoacrylate closure (CAC). [16]
Endovenous heat induced thrombosis (EHIT):	The propagation of thrombus into the deep vein contiguous with the ablated superficial vein. [17] The EHIT classification include Class 1 (venous thrombosis to the junction but not extending into deep system), Class 2 (nonocclusive venous thrombosis with an extension into deep system of a cross sectional area less than 50%), Class 3 (nonocclusive venous thrombosis with an extension into deep system of a cross sectional area greater than 50%), and Class 4 (occlusive deep vein thrombosis of common femoral vein or popliteal vein). [18]
Deep vein sclerosis (DVS):	Sclerosis resulting from inadvertent entry of sclerosant into the adjoining deep veins. [19]
Flush Ligation:	Ligation of the great saphenous vein at its union with the common femoral vein.
High ligation (HL):	Ligation at the junction of the superficial vein and the deep vein, i.e. saphenofemoral junction, saphenopopliteal junction. Sometimes a residual stump is left when the procedure is performed at a distance from the deep vein (low ligation). [20]
High ligation and division:	Ligation and division of the great saphenous vein at its confluence with the common femoral vein, including ligation and division of all junctional tributaries. [2]
Ligation:	The surgical tying off of a vessel, usually of a large vein, is performed to prevent blood flow into a diseased or nonfunctioning vein.
Lymph NodeVvenous Networks (LNVN)/ Transnodal Lymph venous connection:	Are present in the groin, but not always visible on duplex ultrasound. In healthy people, they are small with diameters less than 1mm. LNVNs drain towards the saphenofemoral junction and into the pelvic veins. Reflux in the LNVN can be detected in both primary and secondary varicose veins but is more common in recurrent veins postsurgery (REVAS).
Microsclerotherapy:	Sclerotherapy treatment of spider veins.
Phlebectomy:	Removal of a vein segment through a small skin incision (includes mini/micro phlebectomy, stab avulsion, i.e. removal of vein segment via a small incision). [2]
Neovascularisation:	Defined as new blood vessel formation (angiogenesis) occurring in abnormal tissue or in an abnormal position; requires histological diagnosis. In the 2011 UIP consensus document, neovascularisation was defined as: "presence of multiple new, small tortuous veins in anatomic proximity to a previous venous intervention." In the context of varicose veins surgery, it refers to the presence of new veins situated at the site of the previous saphenofemoral junction or saphenopopliteal junction ligation. These veins may be newly formed or can arise from dilation of existing groin veins that were invisible on duplex ultrasound before the operation. [20]
Perforating vein ablation:	The destruction of a perforating vein by mechanical, chemical, or thermal means. [2]
Perforating vein interruption:	The disconnection of a perforating vein by mechanical, chemical, or thermal means. [2]
Perforating vein ligation:	The interruption of a perforating vein by mechanical means.[2]
Recanalisation:	The development of a new lumen with blood flow present in a previously obstructed/treated vein. [2] Characteristic appearances are of short, tortuous, small diameter vein with thickened walls and evidence of fibrosis or thrombosis. The blood flow may exhibit either venous or arteriovenous characteristics. This should be differentiated from untreated vein segments filled with small, localised fresh thrombus. Recanalised segments may demonstrate forward, reversed or bidirectional flow with compression manoeuvres. In most cases, significant reflux cannot be demonstrated in the recanalised segments. Inflow sources or drainage pathways include great saphenous vein tributaries, perforating veins, and vasa vasorum. [21,22]
Residual stump:	Almost always follows endovenous thermal or glue ablation procedures due to the position of the tip of laser fibre or catheter. May also be seen after surgery if great saphenous vein ligation has been performed at a distance from the common femoral vein (low ligation) instead of flush ligation. Where the terminal valves are competent, the stump receives inflow from its tributary veins that drain normally into the saphenofemoral junction. However, the stump may also become part of the reflux pathway if terminal valves and/or groin tributaries are incompetent, leading to recurrence of varicose veins with the involvement of anterior saphenous and great saphenous vein remnants or other varices. [20]
Sclerotherapy:	Obliteration of a vein (ablation) by chemical introduction (liquid or foam). [2]
Strip-track haematoma:	Of variable volume and may occur following the great saphenous vein stripping surgery. May be visible on grey-scale ultrasound with noticeable fascial distortion. The presence of strip track-haematoma is likely to stimulate endothelialisation within the fascial compartment, leading to revascularisation along the route of haematoma. [20]
Thrombus organisation:	Thrombus is invaded from the vein wall under the effect of cellular activities, during which the vasa vasorum in the intima and media become considerably dilated and new capillaries derived from these vessels traverse the intimal elastic lamina and invade the thrombus. [23]
Trapped Blood:	A collection of haemolysed blood that may appear following sclerotherapy of larger veins, laser or radiofrequency ablation. It consists of a disorganised fibrin mesh and blood cell debris. [24]
Vein stripping:	Removal of a long vein segment, usually most of the great saphenous vein or the small saphenous vein by means of a device. [2]

Stab avulsion:	When a small stab wound or puncture is made to remove varicose veins. [25]
Venous ablation:	Removal or destruction of a vein by chemical, thermal, or mechanical means. [2] • Chemical ablation: endovenous injection of a chemical drug or solution to achieve endoluminal fibrosis and subsequent vein occlusion. • Venous thermal ablation: any endovenous technique employing heat energy to destroy the vein, including laser, radiofrequency or microwave. The goal is to deliver sufficient thermal energy to the wall of an incompetent vein segment to produce irreversible occlusion and fibrosis. • Nonthermal vein ablation: treatment for truncal venous reflux in varicose veins that does not use heat, currently these treatments include ultrasound guided foam sclerotherapy, mechanochemical ablation, and cyanoacrylate glue.
Venous fibrosis:	Ultimately occurs in areas of the organised thrombus. Vascularisation facilitates collagen growth. As collagen matures, the network of capillaries diminishes. Although the vein wall does not undergo complete fibrosis, there is an increase in collagen that separates the muscle fibres connected to the collagen within the thrombus. Haemosiderin can be deposited in various sections of the vein wall, the fibrosed thrombus, and the surrounding perivenous tissue. [23]
Venous occlusion:	Total obliteration of the venous lumen. [2]
Venous sclerosis:	Occurs in response to irreversible endothelial destruction from sclerotherapy procedure, leading to the formation of scar tissue or fibrosis within a vein. [25]
Other Clinical Conditions and Dermatological Manifestations	
Atrophie Blanche:	Presents as a small white patch on the skin caused by occlusion of dermal arterioles and infarction of the skin supplied by capillary vessels.
Corona Phlebectatica:	Fan shaped intradermal telangiectases (broken capillaries) on the medial or lateral aspects of the foot. [26]
Lipodermatosclerosis:	A chronic inflammatory condition of the lower legs, characterised by subcutaneous skin fibrosis and hardening (also known as sclerosing panniculitis, hypodermitis sclerodermaformis).
Lipoedema:	A complex, genetic and progressive condition characterised by the disproportionate accumulation of adipose tissue; lipoedema almost exclusively affects women. In most cases, the legs and buttock are affected with variable amounts of swelling, resulting in poor mobility and quality of life. Lipoedema typically begins at puberty or during other times of hormonal change and weight gain, such as pregnancy and menopause. In many patients, it is not diagnosed for years or is mistaken for obesity or lymphoedema. [27,28]
Lymphoedema	A condition where the accumulation of excessive amounts of protein rich fluid in the tissue results in swelling of one or more regions of the body. Primary lymphoedema results from genetic malformation of the lymphatics. Onset of swelling may not present until adolescence or adulthood. Secondary lymphoedema is much more frequent than primary lymphoedema. Secondary lymphoedema is acquired due to damage or destruction of lymph nodes or lymphatic vessels. This may occur following surgery, radiation therapy related to cancer treatment, recurrent cellulitis, or injury (trauma or surgery to other organs or structures in the body). [29] A positive Stemmer's test, indicated by the inability to pinch and lift a skinfold at the base of the second toe, is diagnostic for lymphoedema.
Matting:	Refers to vessels with a small diameter of less than 0.2 mm that appear sporadically or in well-defined patches. It occurs spontaneously or after superficial venous procedures including sclerotherapy, surgery, or endovenous laser therapy. Considered a major cosmetic complication of sclerotherapy and other superficial venous procedures. [30] Also referred to as 'flares', and telangiectatic matting.
Pigmentation:	Skin changes following deposition of pigment, and in the context of venous disease, pigmentation is due to deposition of haemosiderin in the dermis. Pigmentation is one of the earlier signs of venous hypertension and correlates with CEAP C4a. Chronic venous disease pigmentation should be differentiated from post-sclerotherapy pigmentation [24]
Stasis dermatitis/Stasis eczema:	Eczema/dermatitis due to venous stasis or venous hypertension. It often occurs in the lower leg frequently due to both primary and secondary varicose veins. [31]
Venous Anatomy Terms	
Communicating Veins:	Veins that communicate between two different points of the same venous system and should not be confused with perforating veins (i.e., deep-deep, superficial-superficial).
Deep veins (Deep Venous System):	Veins located below the deep (muscular) fascia with a parallel course to the accompanying arteries. [3]
Duplicated vein	Duplicated veins lie in the same territory as the main vein. Duplication of a deep vein of the lower limb (commonly associated with femoral vein and popliteal vein) occurs when the duplicated vessel is joined inferiorly and superiorly or has a common termination with the main vessel. [32] A true duplicated vein of the saphenous vein will run parallel with the main vessel and within the same saphenous compartment. [33]
Intersaphenous veins:	Communicating veins run obliquely between the great saphenous vein and small saphenous vein. The blood usually flows from the small saphenous to great saphenous network. They may act as pathways for reflux. [34,35,36]
Linton's line:	An imaginary vertical line drawn between the upper third of the calf and the superior border of the medial malleolus along the great saphenous vein to locate medial calf perforating veins. Initially described as an incision line for surgical ligation of clinically significant leg perforating veins. [37]
Nontruncal veins:	All epi-fascial veins of the superficial venous system (e.g., tributary veins, reticular veins and spider veins). [38]

Paired vein (vena comitans/satellite vein):	Two or more veins accompanying an artery. They are usually present with the deep arteries of the extremities, such as the peroneal, posterior tibial and anterior tibial veins of the calf. [39]
Perforator veins (PV):	Connects between the superficial and deep venous system.
Reticular Veins:	Dilated bluish intradermal veins, usually 1-3mm in diameter.
Saphenofemoral Junction (SFJ):	Located at the saphenous opening and correspond to the orifice of the great saphenous vein. [34] From a clinical perspective, the saphenofemoral junction refers to the terminal section of the great saphenous vein where it is joined by other superficial veins such as ASV, and SCIV, SEV, SEPV, PAGSV (refer to anatomical abbreviations below)
Saphenopopliteal Junction (SPJ):	Where the small saphenous vein terminates directly into the popliteal vein. Is situated at variable levels within the popliteal fossa and may be hypoplastic or absent in 25% of the legs. [39]
Spider Veins (or thread veins):	A confluence of dilated intradermal venules of less than 1mm in diameter, also known as telangiectasia. They appear as fine pink, red, purple or bluish lines just below the skin.
Superficial veins (Superficial Venous system):	Veins located superficially to the deep fascia and are not paired with an artery, including the great and small saphenous veins and their branches (collectively called superficial veins of the lower limb). Normally, approximately 10–20% of venous blood of the leg is returned via these superficial veins.
Truncal veins:	The saphenous veins and their major accessory veins (i.e., the great saphenous, small saphenous, anterior saphenous and posterior accessory great saphenous veins and the Giacomini vein). In cases of saphenous aplasia or hypoplasia, the primary connecting tributary which runs epifascially and in a straight course also constitutes the truncal vein contiguous with the saphenous vein. [21, 38]
Anatomical abbreviations (organised from approximately proximal to distal)	
IVC:	Inferior Vena Cava
CIV:	Common Iliac Vein
EIV:	External Iliac Vein
IIV:	Internal Iliac Vein
SGV:	Superior Gluteal Vein
IGV:	Inferior Gluteal Vein
SEV	Superficial Epigastric Vein
SCIV:	Superficial Circumflex Iliac Vein
SEPV:	Superficial External Pudendal Vein
CFV:	Common Femoral Vein
FV:	Femoral Vein (note: Superficial Femoral Vein (SFV) should not be used for this vein)
Pop V	Popliteal Vein
DFV (formerly PFV):	Deep Femoral Vein (formerly known as profunda femoris vein)
PSV:	Persistent Sciatic Vein
SFJ:	Saphenofemoral Junction
GSV:	Great Saphenous Vein (note: Long Saphenous Vein (LSV), Greater Saphenous Vein and Internal Saphenous Vein are not recommended)
ASV (formerly AAGSV/AASV):	Anterior Saphenous Vein (previously known as Anterior Accessory of the Great Saphenous Vein or Anterior Accessory Saphenous Vein) [40]
PAGSV/PASV:	Posterior Accessory of the Great Saphenous Vein (used interchangeably with Posterior Accessory Saphenous Vein)
LNVN	Lymph Node Venous Network
PAV:	Posterior Arch Vein (also known as the Posterior Accessory of the Great Saphenous Vein of the Lower Leg)
ATCV:	Anterior Thigh Circumflex Vein
PTCV:	Posterior Thigh Circumflex Vein
AAV:	Anterior Arch Vein (also known as Anterior Accessory of the Great Saphenous Vein of the Lower Leg)
SPJ:	Saphenopopliteal Junction
SSV:	Small Saphenous Vein (note: Short Saphenous Vein, Smaller Saphenous Vein and External Saphenous Vein are not recommended)
TE:	Thigh Extension of the Small Saphenous Vein (also known as Cranial Extension of the Small Saphenous Vein)
PV:	Perforating Vein
SNV	Sciatic Nerve Varices
SSA:	Small Saphenous Artery

Definition of chronic venous disorder, chronic venous disease, and chronic venous insufficiency

For clarification and consistent reporting, the terms surrounding the diagnosis and management of chronic venous disease have been defined in a consensus document. [2] *Chronic venous disorder* was defined as the full spectrum of venous system abnormalities, whereas c*hronic venous disease* refers to individuals with these abnormalities, but who also require investigation and/care due to their signs and symptoms. The

term *chronic venous insufficiency* (CVI) was used to define the subset of individuals who have advanced signs and/or symptoms, with functional venous abnormalities producing oedema, skin changes or venous ulcers. [2]

The term Chronic Venous Disease (CVD) will be the dominant term used in this clinical guideline as it addresses the diagnostic workup with the use of duplex ultrasound (US), also known as venous insufficiency ultrasound (or chronic venous insufficiency ultrasound), for individuals who require investigation and potentially intervention to alleviate signs and symptoms.

Contributing risk factors

Reported risk factors for CVD include age, sex (female), positive family history, pregnancy, hormones, parity, height, obesity, prolonged standing and prolonged sitting (sedentary lifestyle), genetic factors, decreased physical activity, and previous deep vein thrombosis (DVT). [41-43] Without treatment, CVD becomes more severe over time. [41]

Signs and symptoms

CVD occurs due to ambulatory venous hypertension, resulting from venous obstruction, weakening of the vein wall, malfunctioning valves and/or deficiency in the muscle pump. [21, 43-44] Depending on the intensity of haemodynamic disturbance, CVD may not produce any signs or symptoms at the early stage. However, when clinical manifestations do occur, there is a broad spectrum of possible venous signs and symptoms **(Images 1-9)**.

Venous signs are visible manifestations which include dilated veins (telangiectasia, reticular veins, varicose veins), leg oedema, skin changes, (eczema, sclerosis, hyperpigmentation, dermatitis, atrophie blanche, lipodermatosclerosis) and ulcers. [2, 42, 44-46]

Venous clinical symptoms are complaints related to venous disease, which may include fatigue, leg swelling, abnormal sensations (tingling, itching, aching, burning, pain, feeling of throbbing or heaviness), muscle cramps or restless legs. [2,31 45-47] These symptoms may suggest, but are not pathognomonic of CVD, therefore correlation with venous clinical signs, and the results of diagnostic investigations are often required. [2] It is common to have patients presenting with one or two symptoms of CVD but with no clinical signs during physical examination and no apparent abnormalities on duplex US.

The CEAP (Clinical-Eetiology-Anatomy-Pathophysiology) classification is an internationally recognised reliable and reproducible classification system for classifying patients based on signs and symptoms. It standardises reporting and guides the therapeutic strategy by describing the following classes of chronic venous disease: [5] Refer to article for full explanation https://www.jvsvenous.org/article/S2213-333X(20)30063-9/fulltext

- C0: no visible or palpable signs of venous disease
- C1: Telangiectasias or reticular veins
- C2: Varicose veins
- C2r: Recurrent varicose veins
- C3: Oedema
- C4: Changes in skin and subcutaneous tissue secondary to chronic venous disease
- C4a: Pigmentation or eczema
- C4b: Lipodermatosclerosis or atrophie blanche
- C4c: Corona phlebectatica
- C5: Healed
- C6: Active venous ulcer
- C6r: Recurrent active venous ulcer

Pathophysiology

Venous hypertension is the underlying cause of CVD. It occurs when there is a disturbance in the physiological drainage of venous blood from the leg to the heart, against the force of gravity. Blood from the lower limb is normally returned to the heart via a system of deep veins which are fed by superficial veins either through direct junctions or via perforating veins. Of the many various causes, valvular incompetence and diminished calf muscle function are the two major causative factors responsible for the development and progression of CVD. As the pump fails to empty the blood in the venous system, ambulatory venous hypertension becomes the sequela which further produces a series of histopathological changes to the vein wall and destruction of the valve leaflets.[44-47]

Venous valves and valvular incompetence

Venous valves are present in the lower limb, serving to maintain venous function, by acting as mechanical gates that allow blood to travel towards the heart, without peripheral or reverse flow leakage (venous reflux). [21, 47-48] Typically, there are no valves present in the iliac veins, one (or no) valve(s) in the common femoral vein (CFV), two to four valves in the femoral vein (FV), approximately a dozen valves in the deep calf veins and some perforating veins in the foot.

In relation to the superficial venous system, both the great saphenous vein (GSV) and the small saphenous vein (SSV) have a set of terminal and preterminal valves near their junctions with the deep venous system. The terminal valve is situated very close to this junction and prevents reflux from the deep system into the superficial system. The preterminal valves (previously known as subterminal valves) are situated slightly more distal to the terminal valves. [49]

The number of valves increases towards the distal veins in order to counteract increased hydrostatic venous pressure. [35] At rest, the valves usually remain open, and closure of valves is triggered by muscular activity. Venous reflux (or pathologic reverse flow) resulting from valvular incompetence can occur in superficial, deep and perforating veins (PVs) of the lower limb in either a segmental or axial pattern.

The cause of valvular failure can be primary, secondary or congenital. [2] Primary valvular incompetence is more common and occurs as a result of idiopathic valve dysfunction, [2] whereas secondary valvular incompetence develops from recanalisation during or after previous deep vein thrombosis (DVT), superficial thrombophlebitis (STP), arteriovenous fistula (AVF) or congenital venous malformations. [8, 41, 43] Congenital valvular agenesis refers to congenitally absent or abnormally developed valves. [2] Secondary venous reflux is more common in the deep veins due to thrombotic event. [46] The pathway of CVD related signs and symptoms due to DVT is often called postthrombotic syndrome. [8]

Varicose veins are a manifestation of the combined negative effects of hydrostatic pressure and valvular dysfunction which disrupts the normal vein wall structure due to weakening and/or loss of elasticity in the connective tissue of the vein wall and damage to the valve leaflets. This leads to local vein dilatation and tortuosity. [8,41,46] Venous function is further impaired due to reactive collagen deposition leading to fibrosis and scarring. [41] This pathophysiological process may present in either an ascending pattern (originating distally in distal veins, such as tributaries, perforating veins or lower section of the saphenous vein with upward propagation to the more proximal veins and/or junction), or a descending pattern (reverse order to ascending pattern). [42]

Origins of venous insufficiency

Pathophysiology of CVD in the large leg veins can be described by the origin of the venous insufficiency.

1. Saphenofemoral and Saphenopopliteal Insufficiency (superficial veins)
   Varicosities in the saphenous distribution usually start where superficial veins communicate with deep veins, such as at the saphenofemoral junction (SFJ) and saphenopopliteal (SPJ) junction and in the perforating system. [8] The SFJ is the site where the GSV drains into the CFV; the SPJ is the site where the SSV drains into the popliteal vein. [8,43] At both sites, the terminal valve can be damaged by constantly elevated venous pressure against it, leading to valvular functional loss over time.

Another mechanism of valve impairment is fibrosis secondary to venous thrombosis, where the valve does not return to normal function after vein recanalisation. Impairment of terminal valve function and refluxive forces in the vein result in vein dilation, also increasing pressure on the next upstream valve, leading to a cascading effect of multiple valve failures. [41, 46]

2. Deep Venous Insufficiency with Perforator Vein Insufficiency
   Valve damage and subsequent deep venous insufficiency may occur due to DVT or increased venous pressure secondary to congestive heart failure. Both mechanisms ultimately result in a combination of valve damage and increased venous pressure working to exert retrograde pressure on the perforating veins and subsequent reversed venous flow, which in turn can transmit increased pressure to the connected superficial vein segment. This increased pressure in the superficial vein, facilitates sequential valve failures along its length. [41] Deep venous insufficiency may also be idiopathic. [8] By contrast, a third of the CFV reflux is caused by volume overload and dilatation of the saphenous venous system, which usually undergoes normalisation after the obliteration of the GSV. [50]
3. Pudendal Vein and Pelvic Vein Insufficiency
   Varicose leg veins can be due to vulvar varicosities rather than with reflux of the leg veins. Vulvar varicosities may be associated with clinical symptoms and signs suggestive of pelvic congestion, including uterine retroversion and dyspareunia, menorrhagia, and more commonly occur in multiparous women and those who have had haemorrhoids and vulvar varicosities associated with pregnancy. [8] The pudendal and pelvic veins connect to the GSV and other superficial veins of the leg via small tributaries. These small communicating veins include the superficial external pudendal vein (SEPV), the superficial epigastric vein (SEV), and the superficial circumflex iliac vein (SCIV). The increased venous pressures associated with pudendal vein and pelvic vein insufficiency is transmitted through these communicating veins, leading to GSV reflux and varicosities. [41]
4. Calf Muscle Pump Causes
   The calf muscle pump assists the drainage of venous blood from muscular compartments of the leg to the heart. As the calf muscles contract, the veins are compressed, pumping the blood forward. If there is calf muscle pump weakness, as which can occur with obesity or leg immobility, there will be reduced mechanical action in pumping the blood forward, leading to venous stasis, vein dilatation and valve incompetence. Another calf pump mechanism that can damage valves is activity related sudden vasodilatation such as which can occur with repetitive squats with weights. The sudden, associated increase in blood volume can precipitate valve function over time in predisposed individuals. [41, 47]

5. Plantar Venous Pump and Static Foot Disorder
   The plantar veins of the foot also play an important role in the physiological venous return of the lower extremity. When the foot muscle pump contracts, 50% of the perforating veins of the foot permit flow from the deep to the superficial veins. The impaired foot muscle pump function may lead to CVD. Furthermore, static foot disorder refers to a condition where there is abnormal alignment or positioning of the foot while standing or walking, often due to weakened or imbalanced muscles and ligaments. This can lead to problems such as arch collapse, flat feet, or excessive pronation. While static foot disorder and varicose veins are distinct conditions, they can potentially be interconnected. The altered foot mechanics and misalignment associated with static foot disorder may contribute to increased venous pressure and impaired circulation in the legs, which can exacerbate the development or progression of varicose veins. [51-52]
6. Recurrent Varicose veins
   Recurrent varicose veins may result from failed treatment, neovascularisation posttreatment, or the progression of disease with new incompetence in previously untreated vessels and perforating veins (PV). [35]

Venous hypertension and the microcirculation

The effect of venous hypertension on the microcirculation is different to the insult on the larger veins. The microvessels also have valves which, similar to the large veins, become incompetent, elongated, dilated and become tortuous. With these changes, the endothelium of the microvessels becomes

dysfunctional resulting in oedema and skin changes. [43] The changes in microvessels have been linked to reduction of normal nutrition to skin cells and the subsequent development of venous leg ulcers. [8]

Prevalence and socioeconomic burden

The CVD is the most common vascular disease, with reported prevalence rates for women ranging from 25-60% and for men ranging from 15-49%. [53-55] The CVD and its associated symptoms and/or complications can be responsible for chronic pain, disability and decreased quality of life, leading to loss of working days, and early retirement. A recent systematic review study on global prevalence found that the most common manifestations of CVD were telangiectasia and reticular veins (26%) and varicose veins (19%). Additionally, the review reported that 22% of individuals with varicose veins would progress to develop a venous leg ulcers (VLU) within six years. [55] In Australia, VLU make up 12% of the estimated 420, 000 cases of chronic wounds in hospitals and residential care facilities. [56] This poses a health burden on the population, as well as an associated social-economic burden. [57]

In relation to the economic burden, and using VLU as an example, 2012 Australian data estimates that the total number of chronic leg ulcer (CLU) cases (n=49,098) in private and public hospitals and residential facilities represented an economic burden of US$802.55 million (approximate AUD $830.79 million). [58] Looking at the overall picture, Australian Medicare data demonstrates that in the 2022 calendar year the diagnosis and treatment of CVD disease accounted for 0.14% of the total Medicare expenditure, and varicose vein treatments accounted for 41.9% of all vascular treatments. The expenditure associated with treatment of CVD decreased by 17.8% from 2012-2022 due to the availability of less costly endovenous treatment methods compared to conventional surgeries. Interestingly, the Medicare expenditure on duplex US scans investigating CVD is 7.7 million more than CVD treatments. [59] Therefore, duplex US examination is a large contributor to the CVD economic burden and should be performed to the best standards to reduce unnecessary repeat scans and ensure it generates accurate and comprehensive information, ultimately optimising management and treatment outcomes.

The role of duplex ultrasound in chronic venous disease

Duplex US is the most common imaging technique used in the management of CVD and has been recommended, based on strong levels of evidence, as the primary diagnostic imaging of choice to investigate CVD. [46, 60]

It:

- supports the diagnosis and is helpful for decision making of different treatment options; [44, 49]
- defines associated pathology such as acute DVT, chronic postthrombotic change and superficial venous thrombosis (SVT) with accuracies of 95-97%; [8,21, 63]
- allows the assessment of venous haemodynamics in the veins of lower limb;
- identifies the incompetent veins (including the extent) in the deep, superficial and perforating veins; [8, 21, 46]
- maps the anatomy and morphology of normal and abnormal veins; [46, 61]
- can be used postoperatively to evaluate the success of treatment and to identify complications; [47]
- and can use customised protocols to address the points above. [63]

The advantages of duplex US are that it is regarded as safe, noninvasive, cost effective, reproducible and diagnostically accurate. [8.45] It utilises greyscale (B-mode), colour Doppler and pulsed -wave Doppler (also known as spectral Doppler) ultrasound imaging. Greyscale imaging allows the visualisation of the venous anatomy and its patency. Colour and spectral Doppler imaging demonstrate venous flow with directions for identifying venous reflux and determining patency of the deep, superficial, and perforating veins.[21] Greyscale imaging also allows accurate placement of the spectral Doppler sample volume within the vessel, and colour Doppler assists with the identification of obstruction, turbulence, and the direction of venous and arterial flow. [8] With optimised settings, duplex US is sensitive in detecting even small amounts of reflux in the veins or vein segments.

The visualisation of veins and their haemodynamics may be limited by obesity, severe oedema, and barriers to a good sonographic window such as casts, dressings, indwelling catheters, or limitations in the patient's range of movement (such as joint contractures). Diagnostic quality may be impacted in confused or mentally impaired patients due to lack of cooperation [62] or an inability to tolerate the optimal positions for assessment (i.e. erect or semierect) or the manoeuvres required to assess for venous reflux.[62]
To achieve accurate results, duplex US should be performed by personnel with training, technical expertise and clinical knowledge and using a standardised protocol. [63] Across Australia and New Zealand, there is heterogeneity in how sonographers undertake duplex US to investigate CVD. [64] This clinical guideline will provide education and guidance to sonographers in an effort to standardise practices.