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Practical Venous Imaging of the Lower Extremity

Abstract:

Much has been written and published on the uses and applications of sonographic imaging in the evaluation of the patient with suspected or known lower extremity venous disease. Imaging protocols for venous duplex examination vary widely in their degree of completeness based on indications for referral, institutional traditions, and educational and skill level of the examiner. Frequently, a patient referred for sonography with a stated history of leg pain and swelling receives a limited examination of the deep system above the knee when, in fact, that is rarely the site of venous abnormalities causing the symptoms. It has been reported that less than 5% of patients referred to sonography with a suspicion of DVT actually have it. This paper presents the background information and rationale for a more comprehensive lower extremity venous duplex examination that is time-effective yet comprehensive enough to address the clinical indications for referral.

Understanding Venous Flow Relationships

The primary physiological function of the lower extremity venous system is to provide for the efficient and effectual removal of blood from the legs. Blood enters the leg, pumped by the kinetic energy of left ventricular contraction and by the forces of gravity that pull blood toward the foot. After perfusing the lower extremity tissues at the capillary level, blood crosses into the venules and begins its ascent through increasingly larger diameter venous vasculature until it reaches the inferior vena cava. This ascent, against the forces of gravity, occurs in two structurally and hemodynamically interconnected components of the lower extremity venous system: the deep veins and the superficial veins. These two anatomic systems are connected in series by a group of communicating and collateralizing veins. In addition to the structural integrity of the lower extremity venous system, the presence and proper functioning of two physiologic mechanisms are requisite to the efficient removal of blood from the legs: the muscle-pump and venous valvular function.

Muscle- Pump

The veins in the legs are thin-walled, distensible tubes that serve as conduits for blood returning to the right side of the heart. Unlike the arterial side of the circulatory system, there is no cardiac pump to provide energy to move the blood back toward the heart. The energy required to overcome the forces of gravity and propel blood toward the heart is provided primarily by the mechanical forces of the calf muscles as they contract during walking or other types of leg movement. Each muscular contraction “massages” blood upward past a set of valve cusps. In the presence of competent valves, the blood remains at this level until the next muscular contraction “massages” it up to the next level. This cephalad ratcheting-up of blood through the veins continues until it empties into the iliac veins and the inferior vena cava.

Fifteen to twenty percent (15-20%) of the body's total blood volume may be pooled in the lower extremities within 15 minutes of standing still¹. Proper functioning of the muscular-pump mechanism and the integrity of the venous valves are requisite to the healthy and efficient removal of this pooled blood. The muscle-pump mechanism also contributes to the control of the pressure in lower extremity veins. When there is absence or breakdown of this mechanism, an increase in blood volume in the venous vasculature of the lower extremity results in a subsequent increase in intravenous pressure.

Venous Valves

Another unique and important anatomic characteristic of both the deep and superficial veins are the valves, which play a critical role in helping keep the blood flowing toward the heart. Venous valves are bicuspid structures composed of two delicate valve cusps, each of which consists of a collagenous skeleton covered by a layer of endothelium. Local changes in intravenous pressure during muscle-pump activity force venous valves open to allow blood flow toward the heart and close together again to prevent the flow of blood back toward the feet. Defects in the structure or functioning of valves caused by genetic factors or by damage resulting from acute or chronic venous thrombosis and/or inflammation, may render them incompetent and, therefore, incapable of preventing the backflow of blood down the leg. This retrograde flow, or regurgitation, increases blood volume pooling in the lower extremity and, as with a defective muscle-pump mechanism, results in an increase in intravenous pressure.

There are also valves in the perforating veins that, in normal situations, prevent blood from flowing backward out from the high-capacitance deep system into the lower capacitance superficial system. These will be discussed below.

Increased intravenous pressure in the leg veins may contribute to endothelial damage and cause venous stasis, two components of Virchow's triad that frequently contribute to acute venous thrombosis. Long-standing increase in intravenous pressure is the cause of many types of chronic venous pathology and will be described below. Duplex and color Doppler sonography offer a safe, quick, sensitive and accurate method of assessing the lower extremity for the presence of flow alterations that may cause these conditions.^{2 3 4 5}

Venous Anatomy and Flow Dynamics

The deep and superficial systems are connected in-series, which means that total blood volume entering the leg must pass through segments of both deep and superficial veins for complete and efficient venous return to take place. Via the communicating veins, inadequate flow return in one segment due to disease, dysfunction, or structural defect, places increased flow demands on the other system, resulting in alterations in flow dynamics in both deep and superficial systems. As predicted by Poiseuille's law of hemodynamics, increased flow demands in one segment will result in increased flow volume. Increased flow volume consequently increases lower extremity intravenous pressures that can result in a myriad of related clinical sequelae.⁶

Deep System

The deep veins begin in the dorsal venous rete of the foot and course upward along the bones in the leg. Because these veins are contained within deep fascial compartments and benefit directly from the muscular pump mechanism, they tolerate the high venous pressure in the lower extremity (80-90mm Hg at the ankle during quiet standing)⁷ better than the superficial system, without resulting in pathological stretching or distention of the veins. However, since these vessels are a direct anatomical conduit into the right heart, thrombus formation within these vessels poses a significant risk for pulmonary embolism (PE).

Posterior tibial veins: Begin in dorsal venous rete (foot) and communicate with the superficial veins by way of the communicating, or perforator, veins. The posterior tibial veins ascend along the medial and posterior aspect of the tibial crest. Each posterior tibial vein contains at least ten sets of valves.⁸ They are surrounded by the fascia of the leg. Any edema or swelling of these tissues can compress these veins making them difficult to evaluate sonographically.

Anterior tibial veins: Ascend from branches in the dorsum of the foot along the lateral aspect of the tibia beneath the interosseous membrane. The anterior tibial veins are rarely the site of clinically significant venous thrombosis and, in most protocols, are not routinely scanned.^{9 10}

Peroneal veins: Ascend from branches in the lower calf and follow the course of the peroneal artery along the posterior surface of the fibula. They join with the posterior tibial veins as they enter the popliteal vein. There are 7 valve sets in the peroneal veins.¹¹

Muscle plexus veins: The two muscle plexus veins that can be routinely seen with sonography are the soleus and gastrocnemius. The gastrocnemius plexus drains the blood from the gastrocnemius muscle and empties into the popliteal vein. These are seen as saccular venous structures in the medial calf region. The soleal plexus veins drain the soleal muscle and empty into the either the posterior tibial or peroneal veins near their termination into the popliteal vein. They are usually large enough in the popliteal space to be routinely imaged with sonography.¹² Visualization of the soleus plexus veins is important because deep vein

thrombi most commonly originate in these veins.¹³

Popliteal vein. Formed by the anastomosis of the anterior tibial veins and the tibio-peroneal venous trunk. It lies 1-2 cm from posterior surface of the distal femur. Up to two valve sets are typically present. Duplication of the popliteal vein occurs in approximately 25% of patients.¹⁴

Superficial femoral vein (SFV). Courses up the medial aspect of the leg to the level of the inguinal ligament. It typically lies medial and slightly posterior to the superficial femoral artery (SFA) in the groin and more directly lateral to the artery in the mid to lower thigh. It contains 2-5 valve sets. Like the popliteal artery, it is duplicated approximately 25% of the time.¹⁵

Deep femoral vein (profunda) (PFV). Receives numerous tributaries from the musculature of the upper leg. The deep femoral vein courses along the profunda femoris artery and joins the superficial femoral vein to form the **common femoral vein** in the groin.

External iliac vein. Begins at the anastomosis of the femoral veins at the level of the inguinal ligament. It courses into the pelvis and is joined by the **internal iliac vein** to form the **common iliac vein** at the level of the sacro-iliac joint. The common iliac veins unite to form the inferior vena cava.

Inferior vena cava (IVC). Returns blood to the heart from all structures below the diaphragm. It is formed by the junction of the two common iliac veins at the level of the fifth lumbar vertebra (L5). It courses through the retroperitoneum adjacent to right side of aorta. It enters the thoracic cavity and empties into the right atrium.

Superficial System

The superficial veins are those that run beneath the integument and exterior to the deep fascia of the leg. Their exact course and configuration are quite varied but general anatomic considerations of this system are described here. While clinical sequelae associated with acute venous thrombophlebitis in the superficial system do not pose the same potential for morbidity and mortality as does deep vein thrombosis (DVT), chronic superficial venous disease can produce life long leg problems. These problems range from annoying cosmetic issues to debilitating swelling and ulcerations, to loss of limb due to chronic venous insufficiency or pathologic changes associated with post-thrombotic syndrome. Therefore, the superficial system should not be ignored in patients being evaluated with duplex sonography for venous problems in the leg. .

Greater saphenous (GSV). Arises from medial end of the dorsal venous arch at the foot and ascends in the ankle in front of the medial malleolus. It spirals around the medial aspect of the leg and crosses behind the femoral condyle. It continues up the medial aspect of the thigh and empties into the femoral vein at about 4cm below the inguinal ligament. It receives numerous tributaries throughout its course. Typical normal antero-posterior diameters range from 2-3mm in the calf and up to 6mm in

the thigh. The GSV consists of a single venous conduit in about 67% of patients, while a completely duplicated system is seen in only about 8% of patient. A branching double system has been identified in 18% of patients.¹⁶

Lesser saphenous (LSV). Begins at the lateral end of the venous arch on the dorsum of the foot and ascends behind the lateral malleolus. It extends up the back of the calf, passes between the two heads of the gastrocnemius muscle and empties into the popliteal vein.

Posterior Arch.

Superficial tributaries.

Perforators (Communicating Veins)

The perforating veins connect the deep and superficial systems and are an integral component in maintaining pressure and hemodynamic equilibrium in the lower extremity venous vasculature. In the normal leg, the muscular-pump mechanism and perforator valves keep blood flowing from the superficial system into the deep system. Most perforating veins have one valve; although they may be congenitally absent in some veins.¹⁷ When the valves fail to maintain this pressure-flow relationship either by congenital absence or by pathologic damage, increased blood volume in the leg may dilate the perforators; permanently damage the valve cusps resulting in perforator incompetence.

There are several sets of perforators in the leg and description of the exact anatomic configuration varies among anatomists, radiologists and vascular surgeons. The ones of immediate concern in venous duplex examination of the leg are the medial perforators, which connect the greater saphenous with the posterior tibial veins. The major medial perforators, known as Cockett's perforators are found along the medial-posterior aspect of the tibial crest. They course through the deep fascia of the calf and connect to the posterior arch, which empties into the greater saphenous vein. There are usually three perforating veins and they lie between 10 – 15 cm above the lower margin of the medial malleolus. Normal perforators measure between 0.5 – 4mm in antero-posterior (AP) diameter.¹⁸

Clinical Assessment

Understanding Clinical Signs Associated with Venous Disease

The diagnosis of peripheral vascular disease and an accurate assessment of its level and severity may be made by a taking a careful history and performing a focused physical examination to an extent not possible in many other disease states.¹⁹ In evaluating the patient with known or suspected lower extremity venous disease, patient history and clinical findings can focus the examiner's attention to the location and level of venous disease.

Symptomatology in Acute venous Disease

In a patient with acute deep vein thrombosis, the affected leg typically is swollen, red, warm to the touch and painful. The patient may complain of tenderness along the iliac vessels and femoral canal, in the popliteal space and over the deep calf veins, particularly when compressed by the examiner.²⁰ A positive Homan's sign (calf pain on dorsiflexion of the foot) has been traditionally used as a clinical finding in establishing the diagnosis of DVT, but it is a very nonspecific and certainly not pathognomonic for DVT. In fact, the positive predictive value of Homan's sign is only 33% and several authors have recommended its discontinuation in the evaluation of suspected DVT.^{21 22} In some cases of acute DVT, there are no appreciable leg symptoms at all. The patient may complain of sudden shortness of breath, hemoptysis, and pleuritic chest pain suggesting pulmonary embolism as the presenting symptom of peripheral venous pathology.²³ Epidemiological studies indicate that more than 95% of all pulmonary emboli arise from the deep venous system of the lower extremities.²⁴

Symptomatology in Chronic Venous Disease

Chronic venous insufficiency and post-thrombotic (or post-phlebotic) syndrome are conditions typically characterized by ankle edema, induration, stasis dermatitis, stasis pigmentation, and stasis ulceration. The difference between these clinically similar conditions is in the causative mechanism and venous hemodynamic sequelae. Chronic venous insufficiency is a generic term referring to presence of one or several of the above mentioned clinical findings in a patient's leg. Post-thrombotic syndrome is the presence of the same clinical findings in a patient with a history of acute DVT. About 20-50% of patients with diagnosed DVT develop post-thrombotic syndrome (PTS) within 1-2 years of symptomatic DVT, and severe PTS, which can include venous ulcers, occurs in 5-10% of cases.²⁵

Both chronic venous thrombosis and post-thrombotic syndrome are associated with venous outflow obstruction and/or venous valvular insufficiency. Specifically, the diagnosis of post-thrombotic syndrome demands the recognition and localization of venous valvular incompetence as well as venous outflow obstruction in a patient with a history of acute DVT. The post-thrombotic syndrome (PTS) develops as a result of the combination of venous hypertension due to persistent outflow obstruction or valvular incompetence and abnormal microvasculature or lymphatic function. Among factors potentially related to the development of PTS, recurrent ipsilateral thrombosis plays a major role.²⁶

The significance of the difference between these conditions may influence patient management decisions, but it is generally not important when performing a duplex examination in most medical imaging environments.^{27 28}

Occasionally, lymphedema, chronic inflammatory processes, metabolic disorders, collagen vascular diseases, neoplasia, allergic phenomena, and traumatic ulcers may cause some confusion.²⁹ Other causes of leg swelling are wide and varied as well and, many times, cannot be differentiated on a clinical

basis alone. Other etiologies of leg swelling include: lymphatic obstruction, congestive heart failure, nephrotic syndrome, hypoalbuminemic states, cirrhosis, and idiopathic edema.¹⁴

The type and pattern of leg pain in chronic venous disease can help distinguish it from acute venous disease as well as peripheral arterial disease. The leg pain is typically diffuse and is usually restricted to the calf or ankle. It may be described as aching, fullness or tiredness in the leg. It is not usually exacerbated by walking or other exercise. There may be pain and swelling in the thigh as well if ilio-femoral disease is present. Symptoms may be uni- or bilateral.

Pain in the legs after standing, walking or sitting with the feet dependent for an extended period of time is commonly reported by patients with chronic venous insufficiency or post-thrombotic syndrome. This type of pain is different from arterial claudication in that it does not dissipate after several minutes of rest. Frequently patients will state that their ankles appear normal in the morning when they get out of bed but, as the day goes on, they swell. Swelling is most pronounced in the evening, as are the feelings of aching, fullness or tiredness. Damaged venous valves and increased outflow pressure prevent the efficient reduction in blood volume from the lower extremity. As a result, there is increased intravenous pressure with subsequent extravasation of fluid into the fascia which causes swelling and dull, achy pain. Sudden onset of deep, intense pain is symptomatic of acute venous thrombosis or acute arterial occlusion.³⁰

Superficial Venous Incompetence (Varicose Veins)

Little more than visual inspection is required to diagnose varicose veins. Before they can be treated appropriately, however, the functional status of the iliofemoral valves, the deep venous valves, and the valves in the perforating veins must be determined.

Primary varicose veins usually affect the superficial venous system (greater saphenous vein lesser saphenous vein, and their tributaries). The deep veins remain normal. Occasionally, primary varicose veins may also involve the perforating veins. The exact cause is unknown; however, the development of varicose veins is hereditary.³¹ Abnormal wall weakness, increased distending force, and multiple, small arteriovenous fistulas have been suggested as possible contributing factors. The condition is associated with venous valvular incompetency. The valves in the greater saphenous vein, in the iliac vein, and at the saphenofemoral junction are often absent or incompetent.

Secondary varicose veins involve the deep and communicating veins. The most common etiologic factor is previous deep vein thrombosis. Arteriovenous fistula, (congenital or acquired), and Klippel-Trenaunay syndrome (deep vein hypoplasia or aplasia) are other causes of secondary varicose veins. Deep vein obstruction may or may not be present. In thrombosed veins, some thrombi may gradually resolve with time by the action of thrombolysis; others may undergo the process of recanalization. Venous valves are destroyed by these thrombi, resulting in venous valvular incompetency. This event is followed by progressive dilatation of collateral veins, which results in secondary varicose veins.³²

Physical Examination of the Legs

Visual Inspection

Approximately 5% of patients with DVT develop chronic venous insufficiency (post-thrombotic syndrome)³³. Damage to the venous valves by acute thrombosis, trauma or chronic incompetence permits reflux of blood into the calf veins and increases intravenous hydrostatic pressure. As a result, the veins become dilated and extravasation of serous fluid into the soft tissues causes swelling.¹⁰ As compared to the acutely thrombosed leg, the post-thrombotic leg usually is not red and hot, however comorbidity such as focal cellulitis and pigmentation changes (stasis dermatitis) may complicate the clinical assessment. As stated above, the typical picture of a patient with chronic venous insufficiency includes ankle edema, induration, stasis dermatitis, hyperpigmentation and ulceration. Venous ulcers most commonly occur on the lower calf or ankle and typically do not penetrate the fascia. Even large, infected venous ulcers tend to remain confined to the superficial tissue. In a patient with a known history of thrombophlebitis, these clinical signs are sufficient to establish a diagnosis.⁹

Palpating Perforators

When a perforating vein becomes dilated and its valves become incompetent, superficial veins become large and rounded under the skin. This effect is known as a “blow-out” and the enlarged, flaccid perforator vein can usually be palpated along the medial aspect of the anterior tibial crest as indentations. These “divots” lie in a position where one would expect to find the Cockett perforators and are an exceptionally useful landmark when searching for incompetent perforators with sonography. Prior to the advent and acceptance of duplex ultrasound as an integral tool in the practice of vascular surgery, most surgeons relied on palpation of perforators prior to intervention. Identification of these locations in the leg remains an important part of the pre-treatment evaluation in patients with chronic venous disease.

The location of perforating veins also corresponds well to the site of skin pigmentation and ulcer formation.³⁴ In fact a recent study showed that 86% of venous leg ulcers have some degree of reflux in the local area usually related to perforator anatomy. Treatment of the local hemodynamic abnormalities may be an important factor in the healing of the ulcers and in the prevention of their recurrence.³⁵

Duplex Examination

Using Duplex Sonography to Sort it All Out

Duplex sonography is well established as a highly sensitive and specific diagnostic modality in the evaluation of the peripheral venous vasculature.^{36 37 38} When compared to ascending phlebography (contrast venography), duplex ultrasound has a very high sensitivity (>100%) and specificity (97%) in

diagnosing femoral and popliteal vein thromboses; and 85% and 83% respectively for calf vein thromboses.³⁹ The following duplex examination protocol, when combined with a focused patient history and physical examination of the legs, can increase diagnostic output of a lower extremity venous ultrasound examination by assessing deep, superficial and perforator vein structure and hemodynamics in an integrated psychomotor and cognitive manner. In laboratories that require hardcopy documentation of all normal as well as abnormal findings, this protocol may not be time efficient enough to be performed on every patient.

Deep system:

Graded compression: using well-established two-dimensional real-time sonography protocols the deep veins are visualized in a transverse plane of section and compressed and released. Normal veins will be easily compressible (in most patients) and the lumen will be obliterated on the image. Inability to completely obliterate the lumen is consistent with the presence of venous thrombus, whether acute or chronic. The following venous segments should be evaluated with graded compression:

Thigh: Common femoral vein
Sapheno-femoral junction
Proximal superficial femoral vein
Mid superficial femoral vein
(Distal SFV is difficult to image as it dives into Hunter's (adductor) canal.

CDI is used to presumptively rule out significant thrombus in this segment by demonstrating normal venous flow patterns, particularly a good strong response to distal augmentation.

Popliteal space:

Popliteal vein
Tibio-peroneal trunk
Soleal veins
Gastrocnemius veins

Calf:

Posterior tibial veins
Peroneal veins
(Anterior tibial veins are not routinely imaged.)

Color Doppler Imaging (CDI): CDI is used to demonstrate hemodynamic status in all venous segments outlined above. Familiarity with determination of flow direction using CDI is imperative. The color bar on the image serves as reference standard for flow direction related to the transducer.

1. Normal venous flow is spontaneous and phasic with respiration in femoral and popliteal veins. It may not be in the tibial (calf) veins.
2. Assessment of calf veins require increased Doppler sensitivity settings and may require distal compression to demonstrate color filling of the vessels.

- a. Anterior tibial veins do not respond to distal augmentation techniques.
 - b. Anterior tibial veins are not routinely interrogated with CDI.
3. Provocative maneuvers (proximal and distal compression/augmentation) are performed to assess direction of flow during maneuver and after release of compression. Valsalva maneuver and “sniff” can be used but the effectiveness of these techniques are more dependent on patient compliance and ability to perform the maneuver without effecting movement of the leg being examined.
- a. Distal compression (augmentation): Normal response is an increase flow volume and velocity in during maneuver; no flow reversal upon release of compression. Significant flow reversal (> 1 seconds in duration) upon release indicates “spontaneous regurgitation” a finding consistent with deep venous insufficiency or valvular incompetence.
 - b. Proximal compression: No flow reversal is seen in normal vessels during compression. Flow reversal (>0.5 seconds in duration) is consistent with deep venous insufficiency.
 - c. The optimal sites to assess deep vein flow reversal are in the posterior tibial vein following removal of distal compression with the patient standing, and in the popliteal vein during a Valsalva maneuver. Retrograde flow more than 0.5 s does not occur in subjects with normal venous function.⁴⁰

Superficial System

Graded Compression: As with the deep system, the superficial system is examined in cross section using real-time imaging. The GSV should be scanned from groin to the distal most branching, which is frequently below the knee and may extend as far as the ankle. Being located directly under the integument, it can be easily compressed in most patients. In fact, the weight of the transducer and the examiner’s hand can compress the GSV resulting in artifactual absence of the vessel.

The most important segment of the GSV to examine for the presence of thrombus is at the level of its junction with the femoral vein. Thrombus in this location has a propensity to propagate into the deep system resulting in frank DVT if not treated with anticoagulants.⁴¹

Imaging of varicose veins, or painful, localized, thrombosed varicosities adds little to the information provided with this protocol. Evaluation of patients requiring vein-mapping prior to surgery, injections or other therapeutic interventions for varicose veins is beyond the scope of this paper.

Color Doppler Imaging: Determination of the status of superficial venous competence is an important part of this protocol. While images and CDI of each segment may not be recorded or hard-copied, information about the integrity of the valves is important in proper patient management. For

example, in the patient who present to the laboratory with a swollen leg and a referral to “rule out DVT”, ruling out the presence of acute venous thrombosis does not answer the still present question of why the patient’s leg is swollen. Evaluation of superficial system competence is necessary to preclude other venous etiologies of leg swelling.

Normal flow in the GSV is usually spontaneous but low flow volume may necessitate increasing system Doppler sensitivity and/or the use of distal manual compression to demonstrate flow. As with deep vein incompetence, the presence of spontaneous regurgitation indicated severe venous incompetence, whereas flow reversal only with proximal compression suggests a milder level of incompetence.

Perforators

Color Doppler Imaging. Using the external landmarks described above (indentation along the tibial crest, particularly in areas of hyperpigmentation or venous ulceration) incompetent perforators are relatively easy to find. They are characterized by large (>4mm) serpiginous structures arising from the posterior tibial veins coursing outward toward the posterior arch. With either proximal or distal compression, a rush of color can be seen filling these vein segments. Determination of the direction of flow (normal flow is into the deep from the superficial, therefore, usually away from the transducer) is not always possible or even helpful in cases of gross incompetence. In fact, flow reversal in perforator veins is seen in 21% of normal limbs, therefore the identification of retrograde flow in the perforators with CDI is not specific for the presence of perforator incompetence⁴² However, the identification of large, dilated perforators (> 0.4mm) that fill easily with color during either proximal or distal compression indicating higher mean and peak flow velocities, volume flow, and venous volume displaced outward, are duplex characteristics of incompetent perforators.⁴³

The identification of incompetent perforators with duplex sonography has also come to play a role in planning less invasive surgical procedures for treatment of chronic venous disease, such as, subfascial endoscopic perforator surgery (SEPS). Duplex imaging has proven to be as accurate and contrast venography in assessing the number, function and localization of Cockett perforators prior to SEPS.⁴⁴

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