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Ultrasound of peripheral veins

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Introduction

The widespread use of ultrasound that occurred in the last few decades has led to major changes in the diagnostic approach to diseases of the venous system leading to an almost complete replacement of venography that was previously considered the standard reference technique. Ultrasound enables visualization of deep and superficial veins of the lower and upper extremities, pelvis and abdomen. Ultrasonography is presently the standard test to confirm or exclude deep venous thrombosis (DVT) and to diagnose a number of clinically similar conditions. It is also used to diagnose chronic venous insufficiency and reflux and is vital for pre-operative assessment of varicose veins [(1-3)].

The most important indication is the assessment of deep vein thrombosis. Venous thromboembolism (VTE) is the third most common vascular disease and is the first cause of in-hospital death. The estimated incidence of DVT in the United States of America ranges from 300,000 to 600,000 [35]. The sensitivity and specificity of DVT sonographic assessment is greater than 90% in proximal peripheral veins.

In this chapter we will give an overview of peripheral venous anatomy, describe the technique of ultrasound examination, and present the clinical importance of ultrasound in diagnosing DVT. We will also review the use of ultrasound for pathological conditions of the superficial venous system, veins in the upper extremities and neck. Lastly, we will describe ultrasound findings of some conditions that can be mistaken for DVT

Clinical DVT and a diagnostic overview

VTE is a serious clinical condition with substantial morbidity and mortality. DVT is caused by stasis, injury to endothelium, or hypercoagulable states. Risk factors can be categorised as genetic and acquired:

- Genetic: family history, factor V Leiden, prothrombin G20210A, protein C deficiency, protein S deficiency, antithrombin deficiency.
- Acquired: Age, antiphospholipid antibodies, cancer, chronic disease, obesity,
- Transient acquired: Pregnancy, oral contraceptives, hormone therapy, hospitalization, immobilization, surgery and trauma [35].

Prior DVT is a risk factor for new (recurrent) DVT. A quarter to a half of patients have no known risk factors and more than 65% of cases of DVT remain clinically silent [(4-6)]. A clinical assessment of pre-test probability is useful to triage patients. The most commonly used clinical decision rule for risk stratification is the Wells score which allows to define the pre-test-probability based on a checklist of predisposing factors and commonly associated conditions **(Table 1)** [36,37**]**; among ambulatory patients with suspected DVT a score of ≥2 indicates that the probability of deep venous thrombosis is likely; a score of 1 or less indicates that the probability of deep venous thrombosis is unlikely.

Table 1 Pre-test probability (Wells Score) for DVT.

Factor	**Points**
Active cancer in last 6 months	1
Recently bedridden ≥3 days, or major surgery requiring regional or general anesthetic in the previous 12 weeks	1
Paralysis, paresis, or recent cast immobilization of lower extremities	1
Localized tenderness along distribution of deep venous system	1
Swelling of entire leg	1
Calf swelling ≥3 cm compared to asymptomatic calf (measured 10 cm below tibial tuberosity	1
Collateral superficial veins (non-varicose)	1
Pitting oedema (confined to symptomatic leg)	1
Previously documented deep-vein thrombosis	1
Alternative diagnosis at least as likely as DVT	-2

Clinical probability simplified score

- DVT “likely” - 2 points or more
- DVT is “unlikely” - 1 point or less

Many patient pathways include a D-dimer blood in the low risk patient with leg swelling. The D-dimer pathology test has a high negative predictive value and it can be used for screening; if negative, DVT can be safely excluded. If positive an ultrasound is indicated [(3, 9-21). The most serious complication of DVT is pulmonary embolism (PE), which is a life-threatening condition with high mortality. Symptomatic proximal DVT is associated with pulmonary embolism in approximately half of all patients, and even for those who survive the initial embolic event the risk of subsequent emboli and death remains high, especially in the older patients and those with malignancies or cardiovascular disease. Because anticoagulant therapy has been demonstrated to lower mortality related to PE, early and accurate diagnosis is crucial [(7)].

Since sensitivity and specificity of DVT clinical symptoms is poor; several imaging and measurement modalities have been developed to diagnose DVT including contrast venography, ultrasound and, more recently magnetic resonance venography. Older techniques such as plethysmography and radionuclide studies have been replaced by ultrasound. Contrast venography was long considered the reference DVT screening procedure since it provides good quality images of deep and superficial veins; but is no longer extensively used due to limited accessibility, and to concerns including the need to inject large volumes of contrast medium into a small veins and to avoid falsely negative results due to weak opacification, streaming artefacts and high interobserver variability. Contrast venography is still used in difficult cases when other diagnostic modalities are inconclusive, or when thrombosis of innominate veins or the superior vena cava is suspected [(1, 2, 8)].

Magnetic resonance imaging has recently shown itself to be a sensitive and specific test for DVT in the calf, thigh and pelvis. It is particularly useful because it can differentiate acute DVT from scarring in suspected recurrent DVT and can show extra-vascular causes of leg pain even when the clinical presentation is consistent with thrombotic venous obstruction or venous insufficiency. MRI scanners, however, are still not available in many centers; in addition the examination is expensive and requires a long acquisition time. Multidetector-row (multislice) CT allows accurate depiction of thrombus in peripheral veins and can be done during a chest CT for PE. However, it requires administration of contrast media, exposure to ionizing radiation, and is more expensive than ultrasound [(1, 2)].

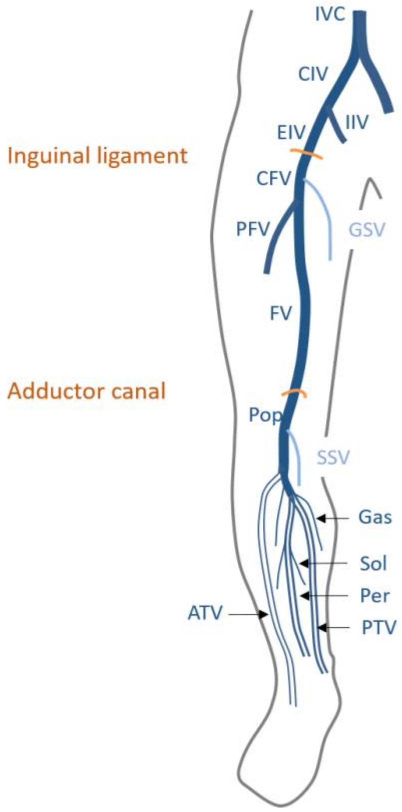
Over the past 20 years venous Duplex ultrasound has been the worldwide method of choice for DVT diagnosis. Venous Duplex exhibits many advantages over contrast venography: it is accurate, non-invasive, well tolerated by the patient. Both examination risk and costs are low; and the test can be repeated, whenever needed, with no harm for the patient. Venous testing is one of the most challenging applications of vascular ultrasound techniques, especially in cases of large legs, obese patients, and for the investigation of the veins below the knee for suspected recurrent DVTs or venous scars. Only high quality equipment should be employed; the accuracy of the examination is highly operator dependent; therefore, to achieve a reliable investigation an appropriate training and sufficient learning curve are mandatory to develop specific manual expertise and interpretation ability.

Venous anatomy and examination technique

The veins of the lower and upper extremities are divided into the deep and superficial venous system. The deep veins lie below the muscular fascia and travel alongside arteries.
The anatomy of the deep veins of the lower extremity is subject to some variability. Most of this is accounted for by the instances of duplications of the femoral and popliteal veins. The distal superficial femoral vein is duplicated over at least a short length in 15 to 20% of patients while the popliteal is duplicated in up to 35% of patients. The duplicated segments of the superficial femoral vein vary in length and rejoin the main venous trunk
The popliteal vein receives some muscular venous tributaries arising from the calf: the soleal and gastrocnemius veins, and some unnamed tributaries. If calf veins are to be examined the gastrocnemius, and soleal veins may be investigated since these have a DVT occurrence similar to the posterior tibial and peroneal veins [(22)]. The deep venous system of the calf includes 6-paired veins: posterior tibial veins, anterior tibial veins and peroneal (fibular) veins. The posterior tibial, peroneal (or together as the tibioperoneal trunk) and gastrocnemius and soleal veins converge to form the popliteal vein: the two anterior tibial veins cross anteriorly the interosseous membrane to join the popliteal vein 6 cm below the knee. Posteriorly, the tibial-peroneal trunk is divided into two true posterior tibial veins and into two peroneal veins (soon after their origin each tibial vein breaks into two paired tibial veins). The tibioperoneal trunk is difficult to visualize in the upper third of the calf. The posterior tibial-paired veins can be imaged as they migrate more superficially at the mid calf and then continue to the back of the medial malleolus. The paired peroneal veins lie deeper and closer to the fibula.
The anterior tibial is rarely the location of an isolated calf vein DVT and most protocols exclude this from a full lower limb examination. In the distal lower leg the deep system consists of plantar veins of the foot. The femoral vein belongs to the deep venous system, and the name “superficial femoral vein” is no longer accepted nomenclature and may be confusing [(23)]. Above the inguinal ligament the vein is the external iliac vein that when joined with internal iliac vein, becomes the common iliac vein. The right and left common

iliac veins converge to form the inferior vena cava [(1, 2)]. The superficial system includes two major veins and their tributaries: the great saphenous vein and the small saphenous vein. The deep and superficial systems are connected through the saphenofemoral and saphenopopliteal junctions and through several perforator veins. The great saphenous vein runs from the tibial malleolus and courses medially upwards to join the common femoral vein the saphenofemoral junction in the groin. The small saphenous vein runs superficially through the posterior calf from the lateral malleolus up to the popliteal fossa. The position of the saphenopopliteal junction is extremely variable; in approximately 30% of people it is situated at the level of the knee joint, in 50% it lays above the knee and in the other 20% it is located below the knee. Basic anatomy of lower extremity veins is shown in the [Figure 1].

Figure1 Lower extremity veins.



Proximal veins

IVC	Inferior vena cava
CIV	Common iliac vein
IIV	Internal iliac vein
EIV	External iliac vein
CFV	Common femoral vein
PFV	Profunda femoral vein
FV	Femoral vein
Pop	Popliteal vein

Calf veins

ATV	Anterior tibial veins
PTV	Posterior tibial veins
Per	Peroneal veins
Sol	Soleal veins
Gas	Gastrocnemius veins

Superficial veins

GSV	Greater saphenous vein
SSV	Short (or small) saphenous vein

Technique

For veins below the inguinal ligament - the common femoral and distal veins - direct compression using B-mode is the most accurate method for the detection of DVT. Full compression of a vein excludes thrombus. Colour flow and spectral Doppler can help to identify veins and challenges of flow but B-mode compression is definitive. For veins above the inguinal ligament, compression is not possible. Doppler investigation can be used to assess flow, it also aids in imaging of iliac vein and IVC patency. The procedure from the proximal to the distal veins is described below.

The iliac veins are examined with a linear (typically 2-8 MHz) curvilinear array (typically 1-5 MHz). Compression is only possible up to the inguinal ligament. Colour images and a flow waveform of the distal external iliac vein are obtained [Figure 2]). Phasic flow from respiration (and often cardiac changes) is indicative of a clear venous return to the IVC. Continuous flow is usually associated with proximal thrombus [Figure 3] or extrinsic compression ([Figure 4] or scarring. Evaluation of both sides is helpful to confirm asymmetry. In pregnant women, if non-phasic flow is detected, a change in patient’s position from supine to lateral decubitus often reduces pressure on the iliac veins allowing phasic flow to return [Figure 5]. Abnormalities can be more easily demonstrated asking the patients to make deep breaths, while carefully looking for a lack of respiratory modulation; even slighter side differences may be consistent with central (iliac or caval) venous obstruction and should prompt further evaluation by angio CT or angio MR to assess the obstruction site and extension and for timely institution of a thrombolytic treatment.

Figure 2 **Flow in the external iliac vein exhibits fluctuations from right atrial pressure changes indicating a good venous return.**

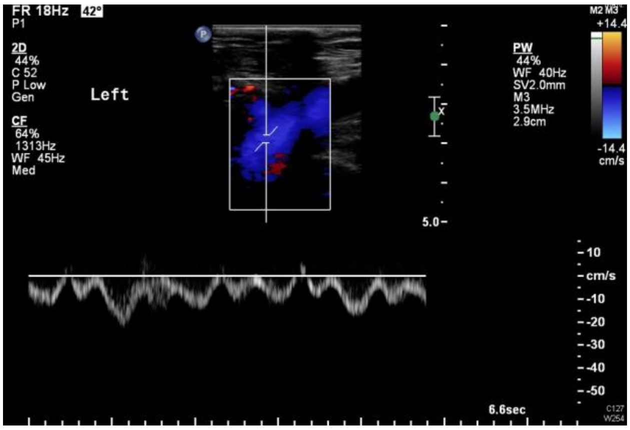
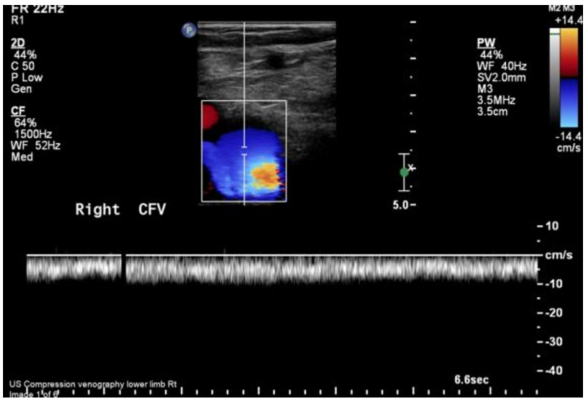


Figure 3 **The R CFV (a) shows continuous flow whereas the L CFV (b) shows phasic flow. The R EIV shows occlusive thrombus (c), flow from the R CFV is drained by collaterals and the normal variations on the R side are absent.**

a



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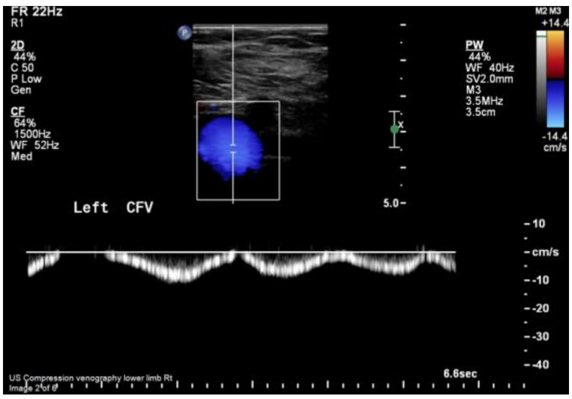
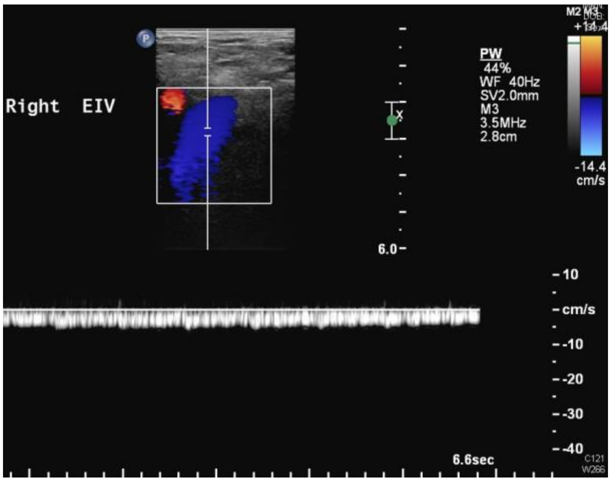


c

Figure 4 Flow in the R EIV (a) is constant suggesting proximal obstruction. A uterine fibroid (b) compresses the iliac veins causing venous obstruction and leg swelling.

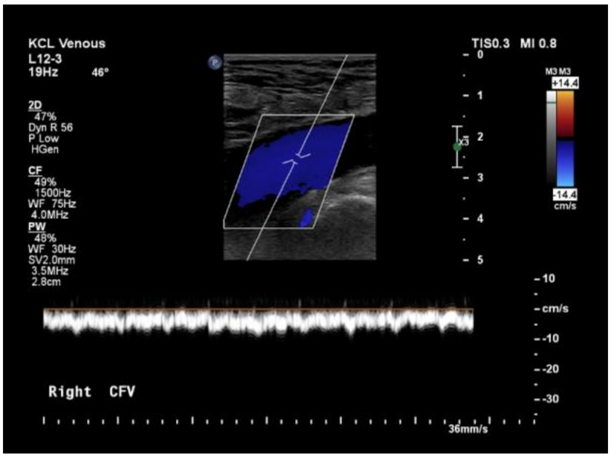
a



b

Figure 5 32 weeks pregnant woman: a R CFV constant flow pattern (a) turns to phasic when the woman lies on her side (b), thus allowing to relieve compression over her iliac vein.

a



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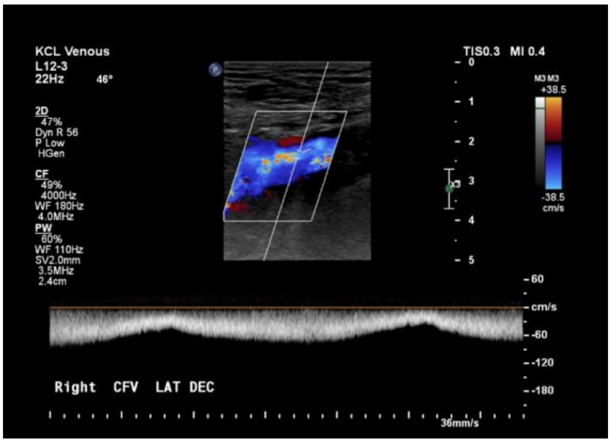
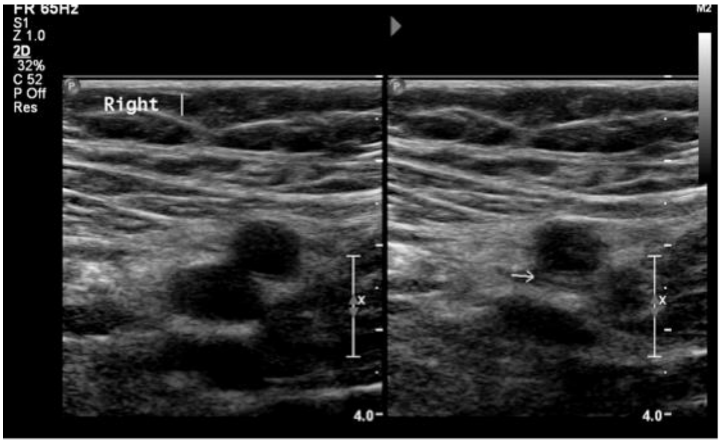


For the common femoral and distal veins, direct compression is used to detect DVT. Non-compressibility of the vein is the main criteria for the presence of DVT [Figure 6]. The proximal (femoropopliteal) veins are examined with the patient supine with as high a frequency linear probe as feasible. Large legs may require a curvilinear array. Ultrasound examination generally starts with conventional B-mode examination that allows the visualization of the venous lumen. When a gentle venous compression is applied by the operator through the transducer, the vessel lumen is completely obliterated [Figure 6a]. The presence of thrombus precludes full compression [Figure 6b].

**Figure 6	Effect of the compression ultrasound on a patent vein (a) and on a vein with occlusive thrombus (b). Transverse view of the femoral vein before compression (a, left image). Following compression the femoral vein is completely compressed (right image arrowed). The arterial walls do not collapse and remain apart because of the higher intra-arterial blood pressure. The popliteal vein (b) (left image arrowed) is more superficial than the artery. Thrombus prevents the vein walls from coming together with compression.**

a



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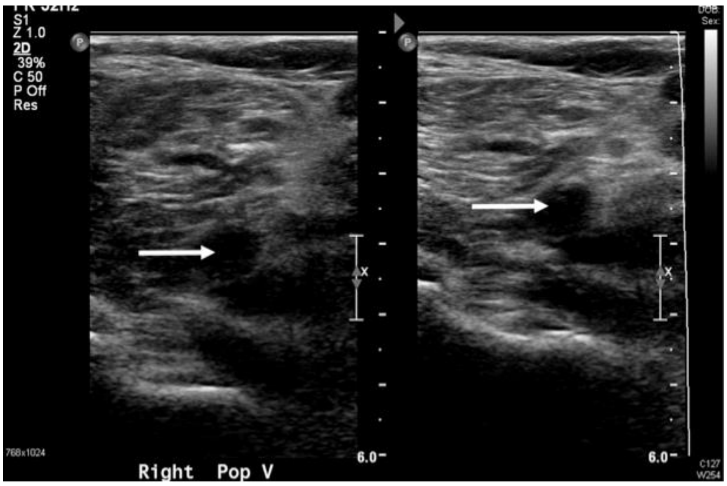


Different patient positions are recommended for optimal venous testing according to the vascular compartment to be analyzed. The use of a tilt table can facilitate patient positioning during the examination, since raising the head over of the couch increases venous pressure and aids venous visualization. Transverse sections of the femoral and popliteal veins are obtained scanning the patient in a supine reverse Trendelenburg position, at 30°. The operator should always check for duplicate femoral or popliteal veins. The great saphenous vein in the thigh can be examined with the patient in the same position. The affected leg can be externally rotated or the patient can be can be examined in a supine or prone position with the knee gently bent to facilitate the examination of the popliteal vein. Longitudinal images of the vein can identify the proximal and distal ends of thrombus thus allowing evaluating its length and time dependent changes at follow-up [Figure 7].

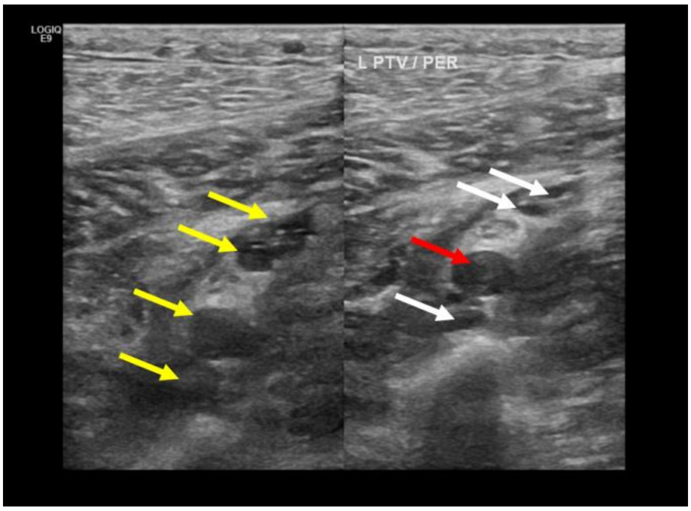
Figure 7 Acute thrombus in a profunda femoral vein extends to the common femoral but the femoral vein is patent. Note that the echogenicity of the thrombus is more echogenic than blood. Echogenicity of acute thrombus varies and is not a reliable sign to differentiate acute DVT from scarring.

The calf veins may be imaged placing the patient supine and tilted into reverse Trendelenburg with the limb externally rotated. Alternatively scanning is performed with the patient seated on the side of the bed with the leg dependent [Figure 8]. Supporting the lateral malleolus to relax the muscles enhances vein filling. In bed-ridden patients the calf veins are less distended and smaller sized. Compression should be applied on calf veins rather than directly on anterior tibial surface, since direct pressing on the tibia itself causes pain and tibial interposition prevents pressure from being transmitted to the vein/s.

Colour can help localizing the corresponding artery and the enhancement of color signal following distal augmentation can clearly depict the venous lumen. Colour Doppler can also show occlusion or recanalization [Figure 9].

Figure 8 Calf vein DVTs a In the uncompressed transverse scan (left), the posterior tibial and peroneal veins (yellow arrows) are visible. With pressure applied to the probe, one peroneal artery does not compress (red arrow) while the others do (white arrows). Soleal and gastrocnemius veins (b) are common DVT sites (frequently coexisting with more proximal DVT). At onset, calf thrombosis can also occur with pain. In this example the gastrocnemius vein (b) is very distended and there is slight deformation indicating a recent thrombus.

a



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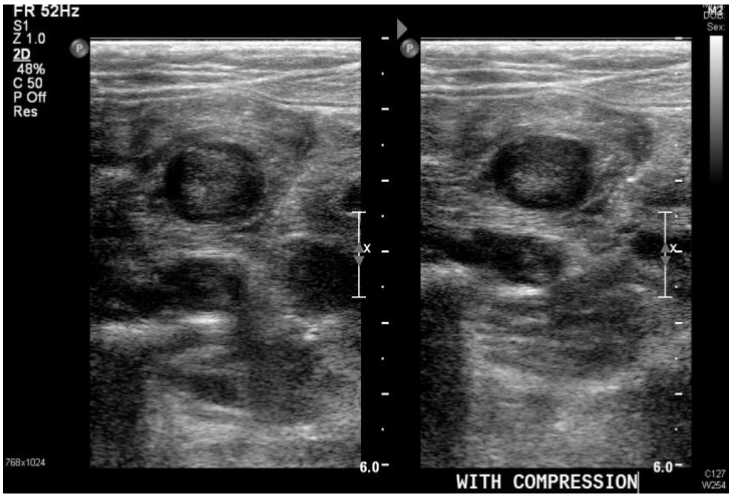
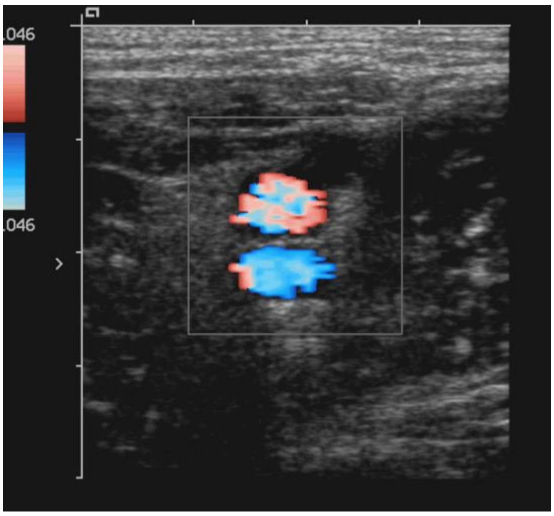


Figure 9 **Colour Doppler can effectively contribute to DVT diagnosis: The artery is color-coded red and one branch of the posterior tibial vein is coded blue (a). The other venous branch is shallower than the artery, it does not display any flow inside the lumen due to thrombotic occlusion. Colour Doppler shows flow signals surrounding the thrombus (b), consistent with incomplete occlusion (similar to a venogram); more irregular recanalization can be observed after healing with flow signals surrounding the scars (arrowed in the femoral vein) (c).**

a



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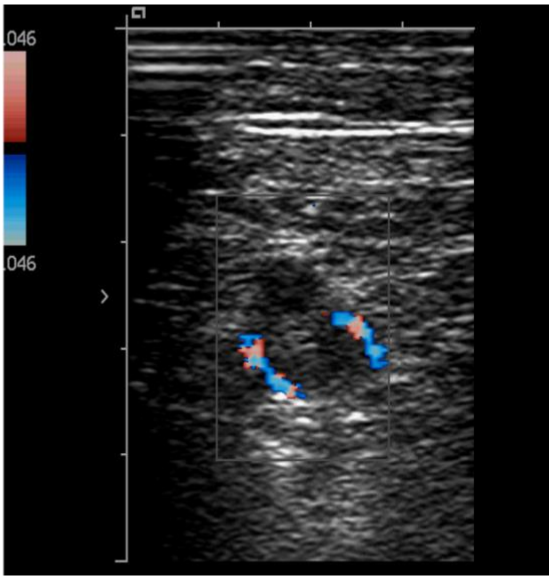

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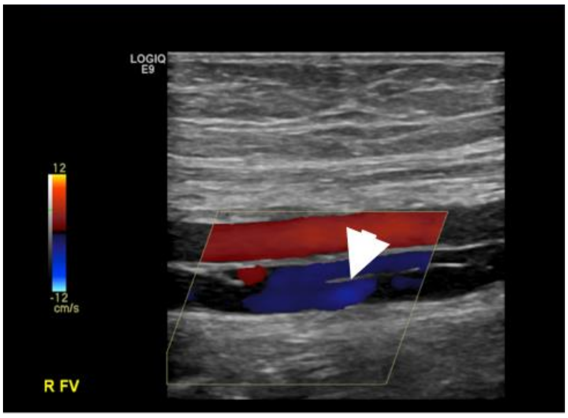
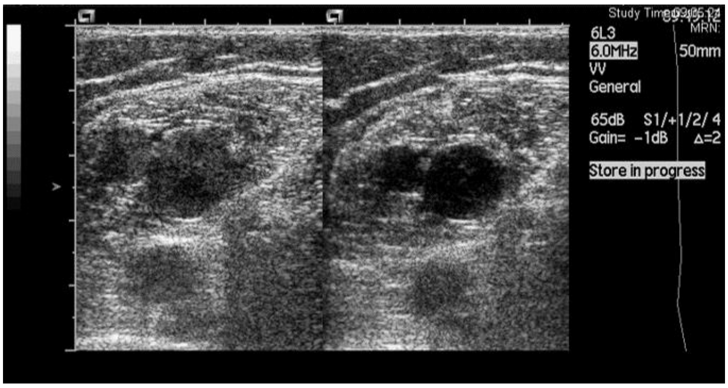


The calf veins may not be fully visualized, due to thrombosis, anatomical variations or to technical limitations. In patients with abnormal body habitus or in the presence of edematous legs, the veins are deeper. A vein may fail to be detected due to extrinsic compression causing small veins to collapse. It is important to confirm thrombus by compression techniques. Static images can be misleading. Intraluminal thrombus, particularly in the acute phase, can look anechoic and conversely, slow-moving blood can appear echogenic [Figure 10]. Evidence from compression ultrasonography, colour Doppler examination, and spectral analysis should be used where required. These are the key elements in the ultrasound diagnosis of DVT as well as in any other venous pathology. Colour Doppler is complementary to compression ultrasonography and can directly demonstrate the presence of DVT if there is no flow evidence or a venous filling defect. In partial thrombosis or during recanalization only a small amount of flow is visible around the thrombus thus allowing to assess and quantify the residual viable lumen [Figure 9].

Figure 10 Gastrocnemius veins. The image on the left shows brighter echoes in the vein. Although this might appear to be thrombus it is caused by stasis of blood and an increase in scattering as rouleaux form. After squeezing the calf (R), new blood enters which has weaker scattering as a result of movement.



Deep vein thrombosis (DVT)

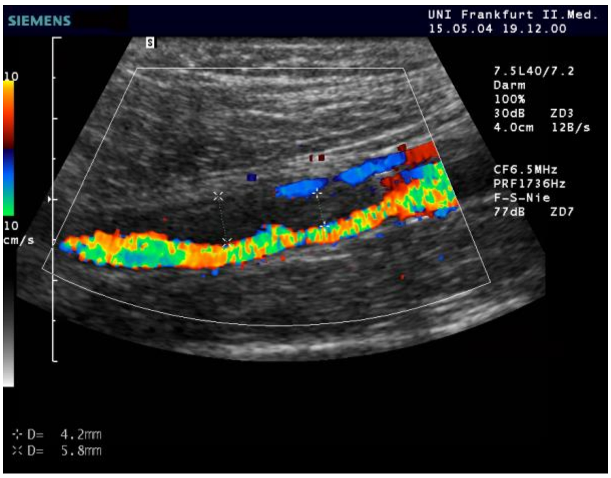
Diagnostic criteria

The most important parameter for diagnosing DVT is non-compressibility of the venous lumen at conventional B-mode examination. In the acute phase the venous thrombus tends to be soft and, despite its lack of complete compressibility, its shape can change following probe compression. The material frequently distends the vein. The echogenicity of the material is quite variable, and heterogeneity is commonplace. A single segment or multiple venous sites can be simultaneously involved; in many proximal DVTs, a venous thrombus is simultaneously found in the calf. Isolated calf DVT is often the earliest phase of the disease and so isolated calf DVT is frequent. In some patients superficial veins are the primary DVT source. This may be the case, for example when an early thrombus is found in the proximal small saphenous vein, with subsequent propagation to deep popliteal compartment through the saphenopopliteal junction [Figure 12]. Colour Doppler can provide further evidence to the examiner to demonstrate filling defects, and to visualize the flow in the veins, and for collaterals and small veins detection. The accuracy of duplex colour Doppler in detecting DVT approaches 99% above the knee and is over 81% in examination of the calf veins. The obstruction of the proximal veins due to thrombosis or external compression leads to the loss of respiratory spectral variations in the distal segments, as well as a lack of response to the Valsalva maneuver in the central veins.

Most thrombus originates from a venous valve. As thrombus increases it can grow directly into or develop in additional veins with intervening normal vein segments. Acute thrombus can grow to fill the lumen partially or completely. Complete occlusion of the lumen by thrombus results in a loss of spectral Doppler signal, non-filling of the lumen with colour, and loss of respiratory spectral variations in the segments ahead of thrombus (i.e.: cephalad, closer to the heart).

In non-occlusive DVT part of the lumen is filled with thrombus [Figure 11]. Colour Doppler shows some flow in the residual lumen and spectral Doppler flow signals can be detected from it. Respiratory changes may be normal, blunted or absent depending on the degree of obstruction.

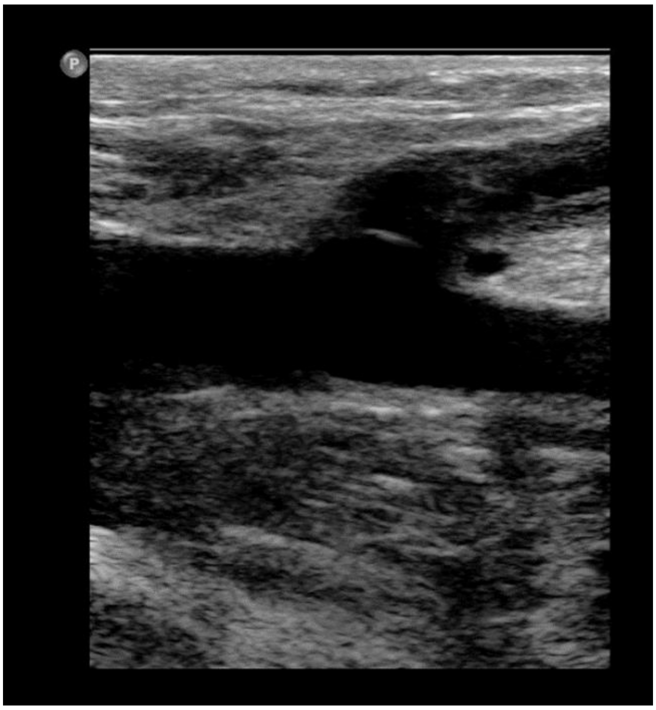
Figure 11 Nonocclusive acute DVT of the femoral vein.



Isolated DVT may occur at the saphenofemoral junction [Figure 12] and saphenopopliteal junction, but it can also develop in any veins, especially calf veins [(22)]. When a thrombus is found close to the level of the saphenofemoral junction, it is important to measure its location from the SFJ and to describe whether the head of thrombus intrudes into the common femoral vein.

Figure 12 Thrombus in GSV flush to a CFV (a). Colour flow shows extension of a GSV thrombus into the CFV lumen with flow returning through a small residual lumen (b).

a



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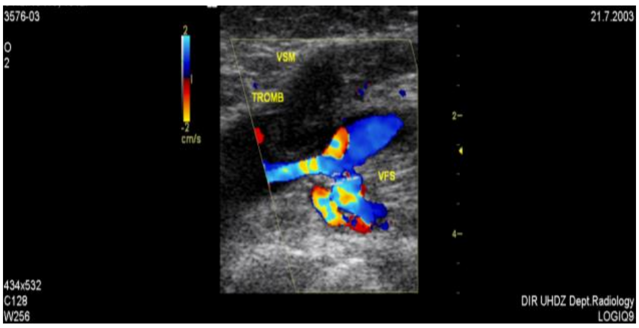


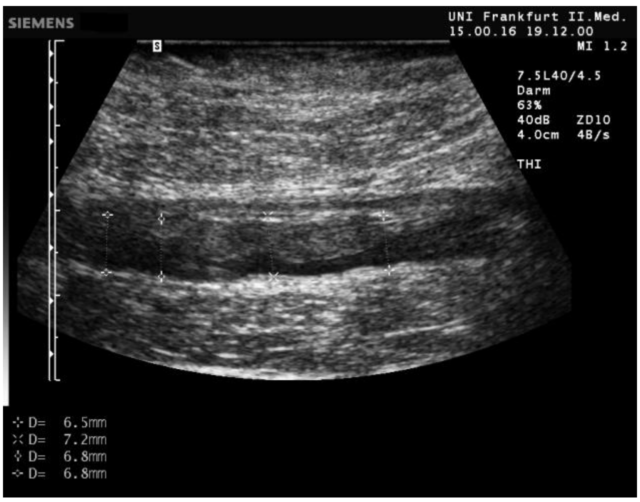
Evaluating the age of intraluminal material

As acute DVT ages, it changes from thrombus to fibrin and collagen over weeks to months [(24)]. A recent consensus favoured the term chronic post thrombotic change for the material, which remains after healing of a DVT to indicate its chronic and fibrotic and it is no longer a thrombus containing fibrin and platelets [3]. The group also did not endorse the term subacute thrombus since it is poorly defined. Subacute has no ultrasound equivalent and may rarely be used when there is a previous baseline scan identifying acute DVT and the follow up study in question shows some evolution but not enough to diagnose chronic post thrombotic change.

There are signs that can help to differentiate acute from chronic post thrombotic change including deformability, attachment to the vein wall, lumen size and the presence of collaterals. In acute DVT the vein is non-compressible and distended, the intraluminal material is soft, deformable during compression, and has a smooth shape. Acute DVT may be poorly attached to the vein wall, perhaps with a free-floating component.

In chronic post thrombotic change (formerly chronic DVT or residual venous thrombosis) the scarred vein may be small or normal sized, its walls are frequently irregular, retracted or thickened. Residual material in the lumen is stiffer, non-deformable and irregular. There may be intraluminal webs or bands. It may rarely be calcified.

Figure 13 Chronic post thrombotic change (previously termed residual venous thrombosis or chronic DVT or scarring) with irregular intraluminal material. The echogenicity is heterogeneous

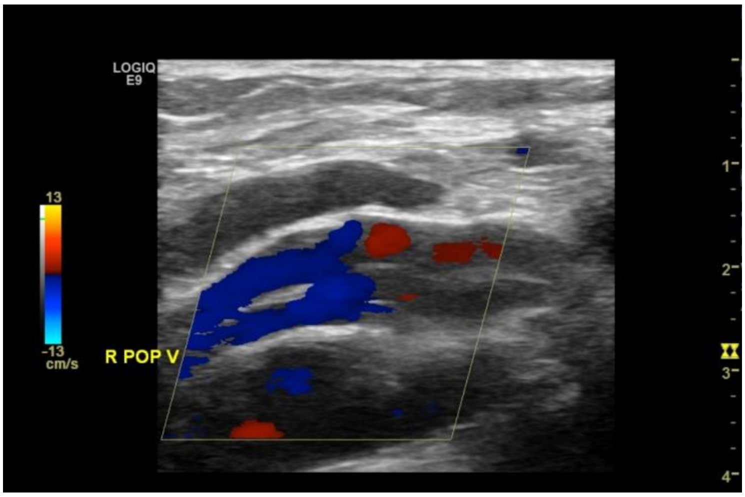


Any acute DVT can produce a pulmonary embolism and it is the most serious complication of DVT. Isolated calf DVT is rarely large enough to produce symptomatic PE, however, calf DVT is not entirely benign since there is risk of propagation into the proximal segments. Interestingly calf DVT is the most common site of DVT found at autopsies after death from PE (it is the residua after the larger DVT embolizes). Calf veins are examined routinely in many institutions but they should definitely be examined if the patient specifies pain or discomfort in that area [(13, 25, 26, 39, 40)]. If found, calf DVT should be evaluated serially for up to two weeks to determine if propagation, resolution or stability has occurred. If propagation above the calf is found, the patient should be treated, and serial imaging may cease.

Treatment and follow-up of DVT patients

Treatment for DVT is generally anticoagulation [(27)]. Close monitoring of coagulation tests during therapy depends on the medications used. It is believed that early and appropriate treatment lowers the risk of pulmonary emboli and the risk of developing chronic venous insufficiency [(13, 19, 20, 28)]. Thrombolytic therapy is increasing used for iliofemoral DVT. Routine re-evaluation of the leg during anticoagulation is not needed unless there is a clinical change and the results will alter therapy. Imaging at the end of anticoagulation is helpful to establish a new baseline since in case the patient presents with leg symptoms as recurrent DVT is a very important complication. In a third of DVT cases complete restitution of flow occurs and in the other two-thirds damage of the valves with some deep venous insufficiency develops. Colour Doppler ultrasound allows the assessment of the recanalization process [Figure 14] as well as assessment of possible complications such as recurrent thrombosis [(1, 13)].

Figure 14 Partial recanalization of a popliteal vein during therapy.



Other pathological conditions that can clinically mimic DVT

One of the advantages of ultrasound is that it is an excellent tool for analyzing structures and organs adjacent to the veins. There are a number of other conditions that can mimic DVT. The clinical diagnosis of DVT is therefore non-specific and it is estimated to be correct in less than 50% of patients. Because of this it is extremely important to recognize other diseases so as to provide the patient with appropriate treatment [(1, 30)] (Table 2). The most common pathological conditions that mimic DVT include simple or complicated popliteal cysts [Figure 15], hematomas [Figure 16] in the muscle or rupture of muscles, muscular abscesses [Figure 17], iliopsoas bursitis [Figure 19] pseudoaneurysms [Figure 18], benign or malignant tumours, inguinal lymphadenopathy and oedema of the soft tissue due to a variety of causes. In this latter category swelling from chronic venous insufficiency and right heart failure are common.

Table 2 DEEP VEIN THROMBOSIS MIMICS (differential diagnosis in approximate order of frequency).

- Cellulitis
- Torn calf muscle
- Ruptured Baker's Cyst

- Subfascial hematoma
- Ruptured plantaris muscle
- Prolonged limb dependency
- Lymphedema
- Lymphangitis
- Nerve entrapment
- Arterial pseudoaneurysm

Figure 15 Extended field of view of a ruptured Baker's cyst

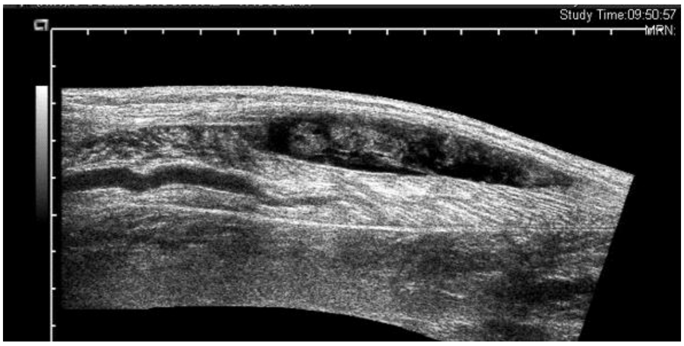


Figure 16 Plantaris tendon tear or rupture with hematoma between gastrocnemius and soleus in an extended field of view image.

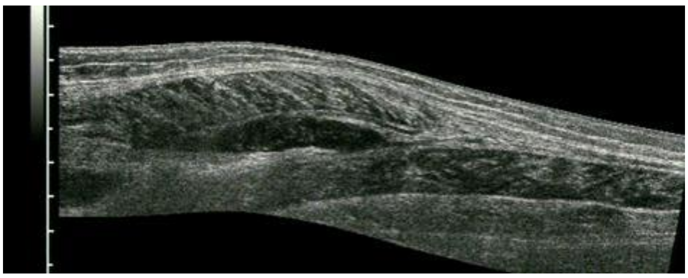


Figure 17 Abscess in vastus lateralis muscle.

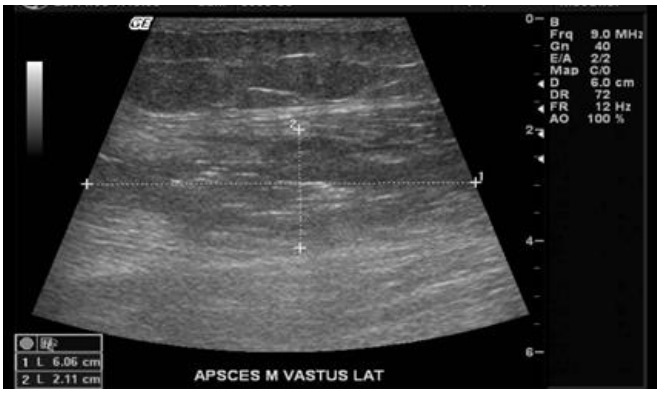
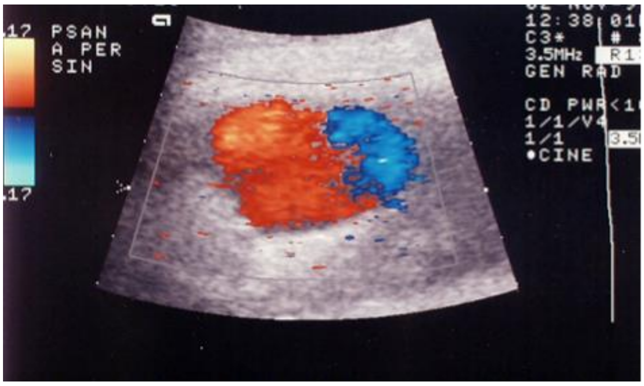


Figure 18 Iatrogenic pseudoaneurysm of peroneal artery (a). Popliteal haematoma with compression on adjacent vessels (popliteal vein thrombotic occlusion; popliteal artery stenosis) (b).

a



b

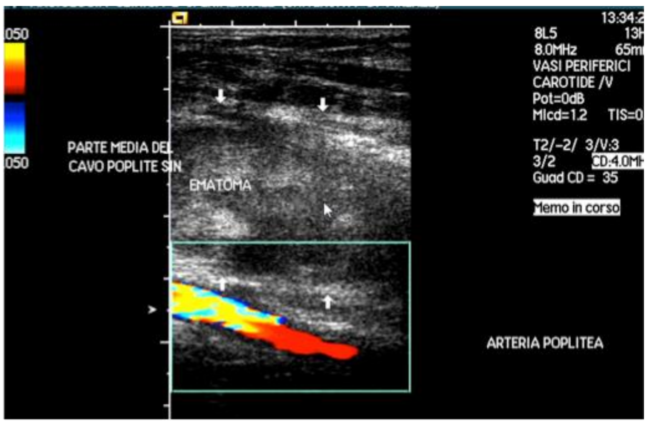
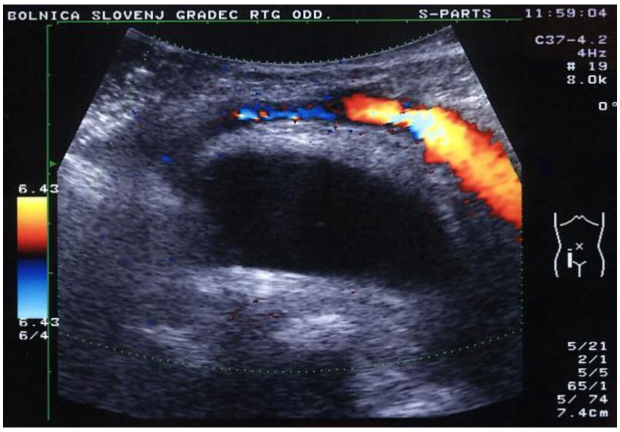


Figure 19 **Iliopsoas bursitis clinically mimicking DVT.**



Chronic venous disease and varicose veins

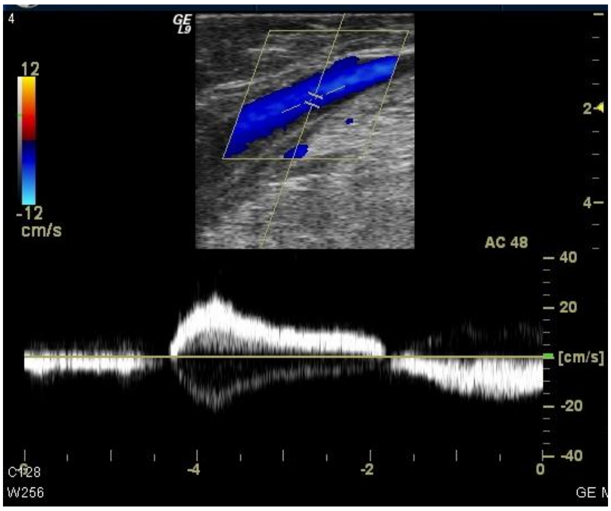
Lower extremity venous insufficiency presents with venous dilatation and occurrence of the retrograde flow through incompetent venous valves. It is a common condition and it is estimated that between 30–60% of adults have some form of true lower extremity venous insufficiency. This incidence increases with age. The clinical manifestations vary from only cosmetic to symptomatically disabling. Clinical symptoms of chronic venous disease are leg swelling, pain and skin changes (eczema, pigmentation or ankle ulceration). Varicose veins are tortuous dilated superficial veins whose location varies depending on which underlying incompetent vein or veins are affected. Reflux and varicose veins may be a sequel of chronic venous obstruction and/or valvular incompetence. Superficial veins may thrombose and cause symptoms of superficial thrombophlebitis [Figure 20].

Figure 20 **Thrombosed varicose vein. The tortuous superficial vein is imaged in sections in the superficial 1cm of tissue with occlusion evident on B-mode.**

Obstruction may increase venous pressure, but dilatation and valvular failure is more often a result of reflux from hydrostatic pressure. Primary varicose veins are more common in female patients, overweight patients and in patients in professions involving standing for long periods of time. Heredity is important to determine susceptibility to primary valvular failure, but the specific genetic factors responsible for varicosities have not yet been clearly elucidated. If valves become incompetent, high pressure may be transmitted from the deep system into the superficial veins. Thus, the normal scenario in which competent valves direct the flow of venous blood upward and inward from superficial veins to larger superficial veins, than into the deep veins and centrally to the heart is lost.

Secondary valvular incompetence is the most common consequence of venous outflow obstruction caused by either intravascular thrombosis of from extrinsic compression. In patients with an obstruction to venous outflow, varicosities must not be ablated because they are important bypass pathways that allow blood to flow around the obstruction. Often the acute DVT does not completely recanalize. “Scarring” occurs with consequent valvular damage and the vein finally becomes a valveless channel that delivers high pressures from above. Colour Doppler can detect valvular incompetence, but spectral Doppler is needed for quantification. Both techniques require provocative maneuvers to demonstrate reflux. For proximal leg veins, a Valsalva maneuver can be used. Distal augmentation and release are also used for some or all veins. This can be done manually or via a dedicated compression device. In normal veins abrupt cessation of the blood flow is observed following a brief deflection produced by valve closure. After release, valvular incompetence is indicated by retrograde flow lasting longer than normal [Figure 21]. Valvular incompetence can be graded using the time of reflux duration (reflux time). Superficial vein reflux is abnormal when it lasts longer than 0.5 seconds [(31)]. Deep venous reflux is pathologic when it is greater than 1 s. A detailed protocol for reflux is needed to determine the sites of disease, location and severity of reflux and sources of varicosities.

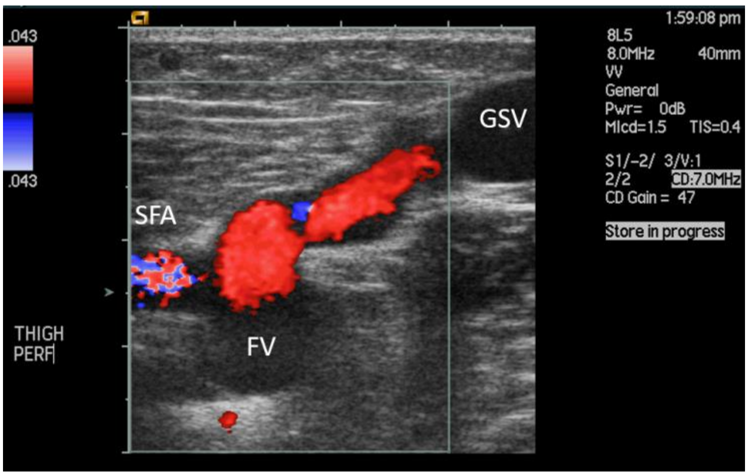
Figure 21 Valvular incompetence is indicated by sustained retrograde flow induced by Valsalva maneuver,



Sonographic evaluation of the superficial system is very important prior to planned surgery. The protocol consists of evaluating the deep system for scarring and obstruction, the superficial and deep system for reflux, mapping of the size of the great and small saphenous veins, and identifying sources of refluxing superficial veins (and, for some, perforating veins). The reflux examination is performed with the patient in a standing position with the weight supported on their contralateral limb. The leg to be examined is flexed and turned slightly outwards. In some protocols, perforators are assessed. Their communication between the saphenous veins and the deep system is identified and their competency assessed. A perforator diameter of more than 4 mm is generally incompetent [Figure 22] and those with a diameter less than 3 mm are likely to be competent. Using colour Doppler, reverse flow can be noted in perforator veins as well [Figure 23]. The nomenclature of the superficial system has been updated removing eponyms for veins, e.g. Dodd's perforator.

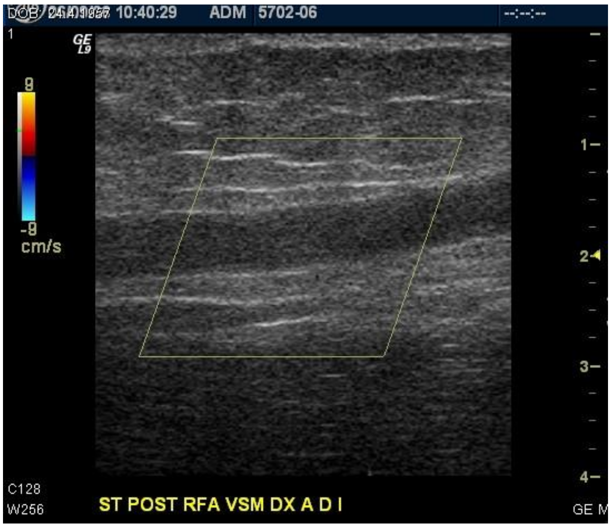
Figure 22 **Very dilated perforator between deep and superficial systems, measuring 11 mm in transverse diameter.**

Figure 23 **Colour flow shows reflux from a femoral vein (FV) to the greater saphenous vein (GSV) in a thigh perforating vein.**



Varicose veins can be treated conservatively, by sclerotherapy, laser ablation, Radiofrequency (RF) ablation and surgical extirpation. Success of the procedure can be monitored by ultrasound [Figure 24]. In pre-operative assessment it is important to note the proximal and distal reflux point as it determines the length of the vessel segment that needs to be removed (stripping). Prior to treating perforator veins, it is helpful to note the position of dilated perforator veins, even to mark their position on the skin. Colour Doppler ultrasound is also a good technique for post-operative follow-up when it is necessary to evaluate recurrent varicosities and post-operative findings. A post ablation study to identify the ablated segment, distance from the saphenofemoral junction, and patency of the superficial inferior epigastric vein is easily accomplished. Post ablation deep vein thrombosis is a rare but reported complication [(32)].

Figure 24 Ablated greater saphenous vein after successful radiofrequency ablation.

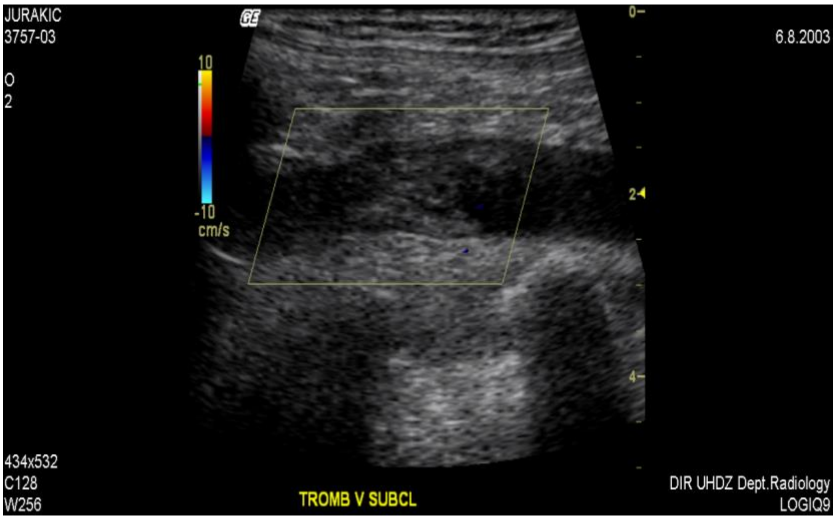


Upper-extremity-veins and jugular vein examination

Thrombosis of the upper-extremity veins is less common than the lower extremities. Subclavian vein thrombosis is associated with central venous lines, strain during exercise, thoracic outlet obstruction syndrome, and underlying hypercoagulable state. Thrombosis may begin centrally and extend peripherally to the axilla or arm, or rarely propagate centrally from the arm or axilla. Colour duplex Doppler has a high specificity and sensitivity to detect upper-extremity venous thrombosis of 92% and 78%, respectively [(33)] with even higher success reported in a structured patient pathway [(34)]. The jugular, subclavian, axillary and brachial veins, as well as superficial veins (cephalic and basilic vein) should be examined. Linear or sector probes can both be used depending on the vein investigated. The patient should be supine and the arms should be symmetrically positioned.

The jugular veins can be compressed. For all central upper extremity veins and especially the subclavian veins, which cannot be compressed, the diagnosis relies on a combination of grey scale, colour and spectral Doppler. Arm veins can simply be compressed. The respiratory variability of lumen diameter on grey scale, absence of a filling defect by grey scale and colour Doppler study are the main signs of a normal vein. In cases of acute thrombosis the vein is distended, without colour filling and often, especially in partial thrombosis, the thrombus can be clearly visible [Figure 25].

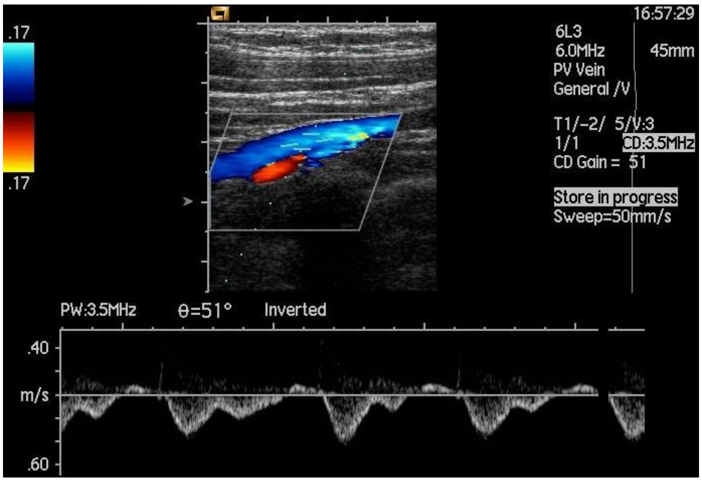
Figure 25 Acute thrombosis of the subclavian vein.



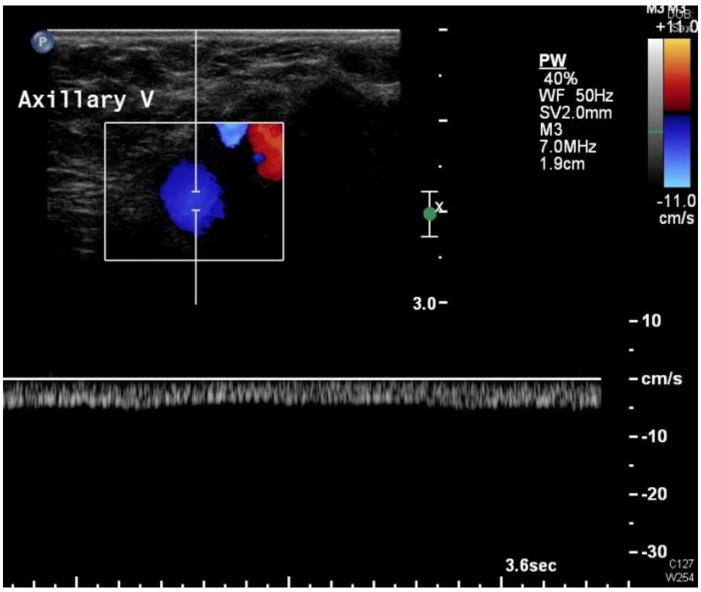
With obstruction, there is diminished or continuous spectral Doppler waveform changes by breathing, by the Valsalva maneuver or by sniffing. Normal pulsatile cardiac changes may be blunted or absent as well [Figure 26]. Comparing side to side is helpful to determine milder but important asymmetry. In the case of loss of bilateral respiratory spectral undulation the proximal obstruction at the level of superior vena cava should be suspected.

Figure 26 **Normal phasic flow in a subclavian vein showing good venous return to the R atrium (a). Constant flow in an axillary vein in a patient with subclavian vein occlusion (b).**

a

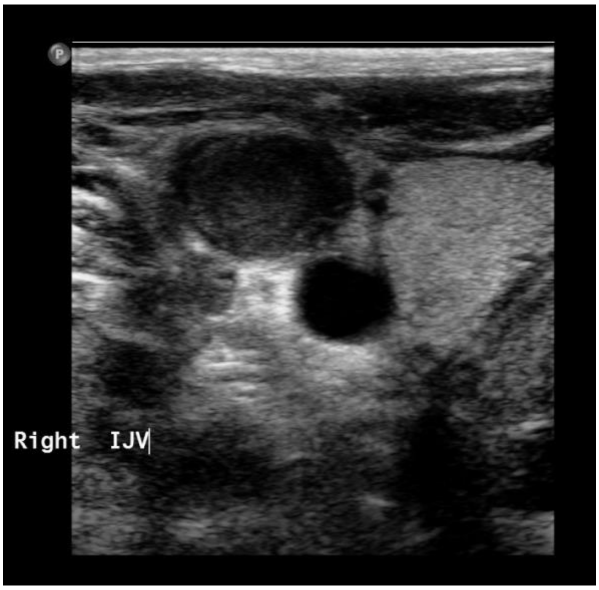


b



The internal jugular vein is situated lateral to the common carotid artery in the neck. Its complete or partial thrombosis can be caused by iatrogenic procedures such as lines and pacemakers, hypercoagulable states (malignant diseases or sepsis) or it can be idiopathic. This vein is easily visible by ultrasound, and in the case of thrombosis it is distended, non-compressible; with visible intraluminal material and no detectable flow [Figure 27]. When obstructed loss of normal phasicity and cardiac pulsatility may be present. Reversal in the jugular vein is also possible with central obstruction since the neck is a rich source of collaterals from one side to the other.

Figure 27 Thrombosis of an internal jugular vein.



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