

## *Chapter 24*

# *Ultrasound of Veins*

**Boris Brkljačić<sup>1</sup>, Sergio Castellani<sup>2</sup>, Colin Deane<sup>3</sup>, Christoph F. Dietrich<sup>4</sup>**

<sup>1</sup>Boris Brkljačić, MD, Professor of Radiology. Department of Diagnostic and Interventional Radiology, University Hospital “Dubrava”, Medical School, University of Zagreb, Zagreb, Croatia.

<sup>2</sup>Sergio Castellani. Department of Medical and Surgical Critical Care A.O.U. - Careggi, associate professor in cardiovascular diseases, chair of angiology, University of Florence, Florence, Italy.

<sup>3</sup>Colin Deane, PhD. Vascular Laboratory, Department of Medical Engineering and Physics, King’s College, London, UK

<sup>4</sup>Christoph F. Dietrich, MD, PhD, Professor. Med. Klinik 2, Caritas-Krankenhaus Bad Mergentheim, Bad Mergentheim, Germany.

**Content**

Content .....632  
 Introduction .....633  
 Clinical DVT and a diagnostic overview .....633  
 Venous anatomy and examination technique .....634  
 DVT .....638  
     Criteria for diagnosis .....638  
     Treatment and follow-up of DVT patients .....641  
     Other pathological conditions that can clinically mimic DVT.....642  
 Chronic venous disease and varicose veins.....645  
 Upper-extremity-veins and jugular vein examination.....648  
 References .....650

**Introduction**

The widespread use of ultrasound with colour and power Doppler has led to major changes in the diagnostic approach to diseases of the venous system. Ultrasound enables visualisation of deep and superficial veins of the lower and upper extremities, pelvis and abdomen. Ultrasonography is today routinely used 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 valuable in pre-operative assessment of varicose veins [1,2].

The most important field of ultrasonographic diagnostic is DVT. The ultrasonographic assessment has a very high level of sensitivity and specificity for DVT that can range up to 90%. The introduction of ultrasound in diagnosing DVT has resulted in a dramatic decrease in the number of contrast venographies performed. In fact, venographies have almost disappeared in modern radiological practice, and today are reserved only for a very limited number of indications [1,2].

In this review we will give a short overview of peripheral venous anatomy, describe the technique of ultrasound examination, and present the clinical importance of ultrasound in diagnosing the DVT. We will also present the possibilities of ultrasound in diagnosing the pathological conditions of the superficial venous system, veins in the upper extremities and neck, as well as other pathological conditions that can be clinically misdiagnosed as DVT, but can be clearly differentiated by means of colour Doppler ultrasound.

**Clinical DVT and a diagnostic overview**

DVT is a serious clinical condition with substantial morbidity and mortality. DVT is caused by injury to endothelium, circulatory stasis or hypercoagulable states, the last includes antithrombin, protein C or S deficiency; activated protein C resistance; or the prothrombin mutation G20210. Risk factors include leg fractures, major surgery (particularly hip-replacement), congestive heart failure, prolonged bed rest, pregnancy or malignant disease. A quarter to a half of patients have no known risk factors. It is estimated that in the United States the incidence of DVT is as high as 600,000 cases per year. The clinical diagnosis is not reliable and symptoms (including the typical combination of Homan's sign, localised oedema and tenderness) can overlap with many other pathological conditions such as muscle haemathoma, abscess, ruptured popliteal cyst and so on. In contrast, more than 65% of cases of DVT remain clinically silent and have no apparent clinical symptoms [3–5].

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 for those who survive the initial embolic event the risk for subsequent emboli remains high, especially in the older patients and those with malignant or cardiovascular disease. Because anticoagulant therapy has been demonstrated to lower mortality related to PE, early and accurate diagnosis is crucial [6]. Since clinical diagnosis of DVT is non-specific, several diagnostic modalities have been developed to diagnose DVT. These modalities include contrast venography, plethysmography, radionuclide studies, ultrasound and recently spiral (particularly multidetector-row) CT-venography and magnetic resonance venography.

The contrast venography was, for a long time, considered to be the gold standard DVT diagnosis because it provides good quality images of deep and superficial veins. In this procedure, contrast medium is injected into a dorsal vein of the foot and filling of the venous system is recorded. The

main phlebographic findings are: persistent filling defect, abrupt interruption of contrast in the vein, lack of opacification in all or some deep veins, and flow diversion with opacification of collateral branches. Negative aspects of this investigation include the need to administer the contrast medium, false-negative results due to the weak opacification and streaming artefacts and high interobserver variability. Also, the method is invasive and requires puncture of small dorsal veins of the foot. Today, contrast venography remains useful in difficult cases, if other diagnostic modalities remain inconclusive or when thrombosis of innominate veins or the superior vena cava is suspected [1,2,8].

Impedance plethysmography monitors the venous blood pressure and outflow at various points of the leg to determine if there are any blockages. The pneumatic cuff is placed around the middle of the patient's thigh, along with several electrodes on the patient's calf. While the cuff deflates, the electrodes record the electrical impedance, and changes in leg volume and blood flow are noted. Although the method is non-invasive and rapid, the sensitivity may be as low as 65% for DVT in the upper leg [1,2].

The main scintigraphic test available is radionuclide venography with technetium-99m-labeled synthetic peptide. The peptide P280 is a 26 amino acid dimer that binds with high affinity to the GPIIb/IIIa receptor expressed on activated platelets and can be labelled with 99mTc. Other tests include the I-125 fibrinogen accumulation test that is sensitive for recent DVT of the calf veins. However, the false-positive findings are very common, and the method has no advantages over ultrasound [1,2].

Magnetic resonance imaging has recently shown it self to be a sensitive and specific test for DVT in the calf and pelvis. It is particularly useful because it can show non-vascular causes of leg pain when the clinical presentation indicates venous obstruction or insufficiency. But MRI scanners are still not available in many centres, and the examination time is long and expensive. Multidetector-row (multislice) CT allows relatively accurate depiction of thrombus in peripheral veins in the same procedure performed for diagnosis of PE. It is expected that the method will spread with the wide use of MDCT scanners. However, it requires administration of large quantities of contrast media, and includes exposure to ionising radiation. The method is also more expensive than ultrasound [1,2].

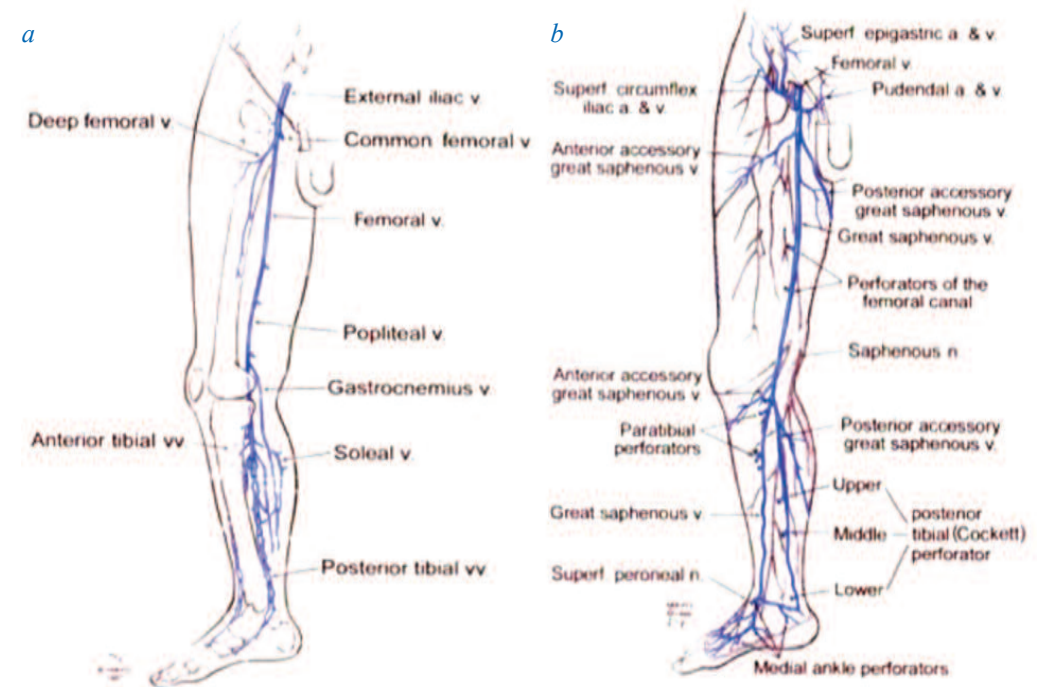
Colour Doppler ultrasound over the past 20 years has become the worldwide method of choice for diagnosing DVT. It has many advantages when compared with contrast venography: it is non-invasive, comfortable for the patient, has no ionisation radiation and it can be repeated as many times as necessary. The risk and the cost of examination are lowered and the sensitivity and specificity remain excellent. However, ultrasound has a small field of view that can be extended with panoramic imaging. It also requires a long-learning curve and experience with a high-degree manual expertise required to perform a good quality examination, especially in cases of recurrent DVT with post-thrombotic changes; the examination can be time-consuming and good quality equipment is essential. It is important to incorporate pre-test probability tests into the diagnostic work-up of patients with suspected DVT; it is also important do differentiate symptomatic and asymptomatic patients and to define the high-risk patient group [9–21].

### 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 follow the path of the arteries. In the lower leg the deep system consists of plantar veins of the foot and six paired veins in the calf: posterior tibial veins, anterior tibial veins and peroneal (fibular) veins. These veins converge to form the popliteal vein. At the level of Hunter's canal the popliteal vein becomes the superficial femoral vein and is joined with the profound femoral vein to form the common femoral vein. The superficial femoral vein belongs to the deep venous system, and the name "superficial" often causes confusion in clinical practice because the superficial femoral vein is actually a deep vein. It has therefore been suggested in the literature to call it simply the femoral vein. Above the inguinal ligament, it is called 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 consists of the two major veins and their tributaries: the greater saphenous vein and the lesser saphenous vein. The deep and superficial systems are connected through communicating veins (perforate veins). The greater saphenous vein runs from the medial (tibial) malleolus and courses upwards until the saphenofemoral junction in the inguinum. It can be seen on ultrasound examination during its whole course. The lesser saphenous vein runs from the lateral malleolus up to the popliteal fossa. The position of saphenopopliteal junction varies considerably; 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.

Figure 1 Anatomy of the lower extremity – deep venous system (a). Anatomy of the lower extremity – superficial venous system (b).

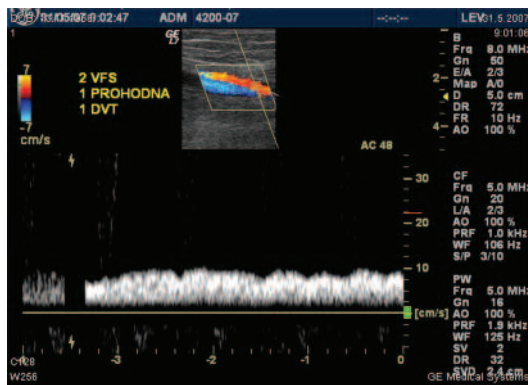


Ultrasound examination should always start with conventional B-mode examination that allows the visualisation of the venous lumen. With gentle probe compression directly over the lumen the vein can be completely compressed and thus differentiated from the adjacent artery. With the application of colour Doppler, the flow in the lumen can be seen, and direction of the flow assessed (Figure 2). By adding spectral frequency analysis the venous and arterial spectra can be very easily distinguished. Typical venous spectra are continuous, with low amplitudes and respiratory undulations (Figure 3), while the peripheral arteries have typical pulsatile triphasic spectra [1,2,8].

Figure 2 Normal colour Doppler image of the femoral vein.



Figure 3 Normal continuous venous spectrum in femoral vein.



Ultrasound examination should include the evaluation of the pelvic, femoral, popliteal and calf veins. In the case of bilateral femoral or iliac thrombosis, the inferior cava vein should be examined, as well as the internal jugular vein, to assess the possibility for implanting a cava filter. With the patient in the supine position the deep veins can be easily seen to the level of popliteal trifurcation, but the peripheral veins in the calf are often collapsed and can be seen only in the standing position or with the use of manual compression applied distally to the probe. Manual compression on the distal segment of the calf results in a faster blood flow to the proximal part of the leg and this results in the increase (augmentation) of the flow that can be clearly documented on spectral analysis as a sudden sharp increase in the normally flat basal venous spectra. This increase in

flow with distal compression is a very important clinical test that excludes the presence of thrombus in the segment between the position of the probe and the distal site of compression.

Compression of the veins (gentle pressure with the probe directly on the underlying segment of the vein) of the leg is the most accurate method for detection of DVT. The non-compressibility of the vein is the main criteria for the presence of DVT. The other signs that should be looked for are the spectral response to distal manual compression and response to the Valsalva manoeuvre.

The examination should begin with the patient comfortably lying down. The iliac veins are examined with a curved probe with a frequency of 3–5 MHz. Femoral veins and proximal greater saphenous veins are usually examined with a linear probe with a frequency of 5–10 MHz. The applied frequency of the probe depends on the patient's constitution and the position (depth) of the veins. The location of greater saphenous vein (GSV) is very shallow, just 1 cm below the skin, so the pressure of the probe during examination should be very gentle. The popliteal vein is examined with the patient in prone or in a side position. The calf veins should be examined preferably in the standing position. Posterior tibial veins and peroneal veins can be examined from the medial side of the calf and can be seen in one field of view, and the anterior tibial veins can be examined from the anterior side of the calf. When the calf veins cannot be seen properly, this could be due to thrombosis, anatomical variation or a technically inappropriate examination. Soft tissue expansion and oedema can lead to vein collapse and could be the reason for non-adequate visualisation of the veins. It is important to know that examination only with conventional B-mode ultrasound is not sufficient for making or excluding the diagnosis of DVT. Intraluminal thrombosis, particularly acute, can often look anechogenic and can be overlooked on B-mode examination. Therefore, compression ultrasonography, colour Doppler examination, and spectral analysis should all be a part of the examination. 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 as it allows the easy and fast visualisation of venous collaterals and anatomical variations. It is also useful in diagnosing DVT: if the thrombus is present there is no flow and the lumen does not fill with colour. In partial thrombosis or during recanalisation only a small amount of flow is visible around the thrombus so it can be very helpful in assessing how much the lumen is open.

The low-extremity veins normally have respiratory dependant spectra that manifest as mild undulatory changes during the breathing cycle. The Valsalva manoeuvre is a very important tool in the evaluation of venous flow. When performing the Valsalva manoeuvre intra-abdominal pressure is raised and peripheral venous flow is transiently diminished. This can be seen as widening of the venous lumen and a lack of flow on the colour Doppler study. Upon ceasing the Valsalva manoeuvre, augmentation of flow is present. The normal femoral vein widens during the Valsalva manoeuvre for 50–100% when compared with its basal diameter. In partial thrombosis this widening is less prominent and in complete occlusion it is completely absent. To perform the Valsalva manoeuvre good cooperation with the patient is mandatory, and apart from DVT other pathological conditions such as external compression of the iliac veins by a tumour, haematoma or ascites can also result in a similar lack of normal response. In heart failure the reactivity of peripheral veins to changes in intra-abdominal pressure is decreased, and with right-heart failure the retrograde increase in pulsatility in lower extremity veins is present with a loss of normal continuous venous spectra [1,2].



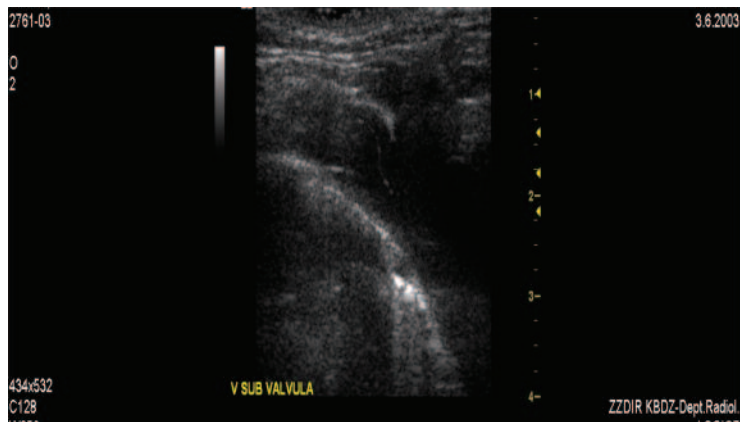
DVT

Criteria for diagnosis

The most important parameter for diagnosing DVT is non-compressibility of the venous lumen detectable on the conventional B-mode examination. Adding colour Doppler helps the examiner to visualise the flow in the small veins, detect collaterals and visually evaluate the effects of compression and the Valsalva manoeuvre. The accuracy of duplex colour Doppler in detecting DVT approaches 99% above the knee region 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 manoeuvre. Therefore, whenever there is a pathological response to the Valsalva manoeuvre at the upper-tight veins and the veins are normally compressible one should look for ascites, expansive lesions in the lower abdomen or iliac vein thrombosis. Similarly, when there is a pathological response in upper-tight veins one should look for pathology of the popliteal vein or calf veins [1,2,12].

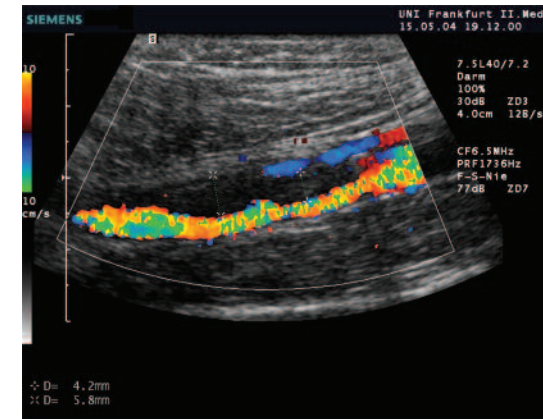
The thrombus forms and develops on the venous valve (Figure 4) and grows and spreads from that point. It can grow to fill the lumen only partially or completely and thus the thrombosis can be only partial or complete. The complete filling of the lumen with thrombus results in a loss of Doppler signal, non-filling of the lumen with colour, loss of augmentation on distal compression and loss of respiratory spectral variations in the segments proximal to thrombosis. As in the calf, deep veins always follow concomitant arteries, and the open collaterals, with no concomitant artery, can be easily differentiated from the occluded deep vein [1,2].

Figure 4 Normal B-mode image of venous valve.



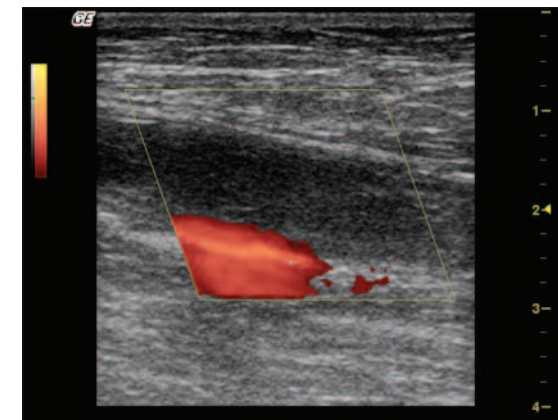
In partial DVT one part of the lumen is filled with thrombus (Figure 5). The vein is only partially compressible and around the thrombus the lumen fills with colour and Doppler spectra can be detected. The respiratory spectral changes are absent and a response to the Valsalva manoeuvre is present, but diminished.

Figure 5 Partial DVT of the femoral vein.



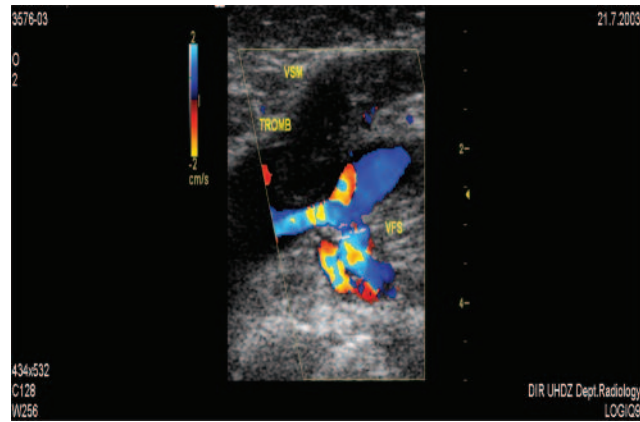
Complete DVT can be focal or diffuse. Often the longer segments of the veins are affected and in more severe cases all deep veins of the lower extremities can be thrombosed (Figure 6).

Figure 6 Complete acute DVT of femoral vein demonstrated in power Doppler.



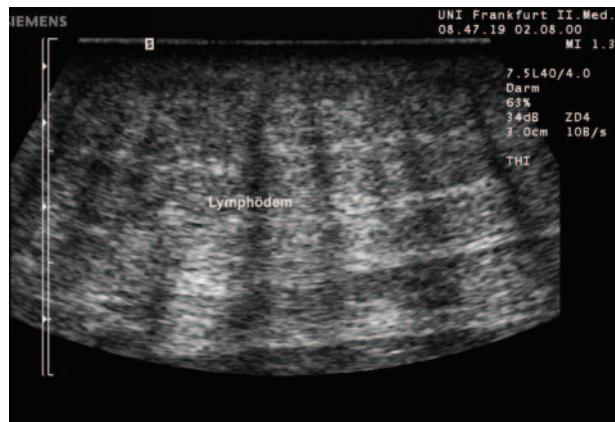
Focal DVT is a localised change when only a short segment of vein is affected. The most common place for focal DVT is on saphenofemoral junction (Figure 7), but it can also develop in the iliac, popliteal or one of the calf veins. From the calf veins the most commonly affected are the posterior tibial veins, and the least commonly affected are the anterior tibial veins.

Figure 7 DVT of the saphenofemoral junction.



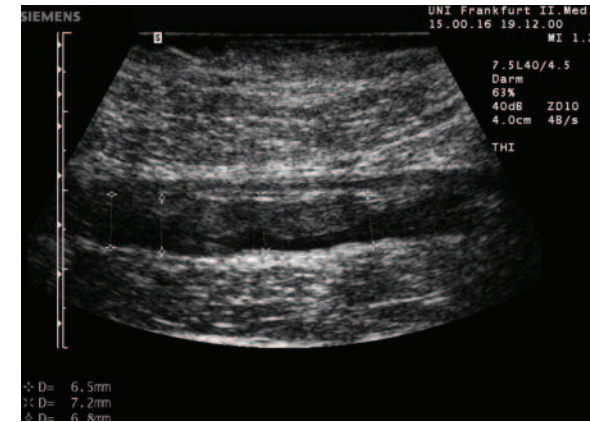
There are some signs that can help to differentiate acute from chronic DVT. In acute complete DVT the vein is completely non-compressible, distended and the thrombus is usually hypoechoic and has a smooth shape (Figure 6). Severe soft tissue oedema is usually present (Figure 8).

Figure 8 Severe soft-tissue oedema accompanying DVT.



In chronic DVT the vein walls are irregular and thickened, the lumen is filled with more echoic material (Figure 9) and calcifications of the lumen can be present. This process can be regarded as vein scarring [1,2].

Figure 9 Chronic, long lasting DVT with echogenic intraluminal thrombus.



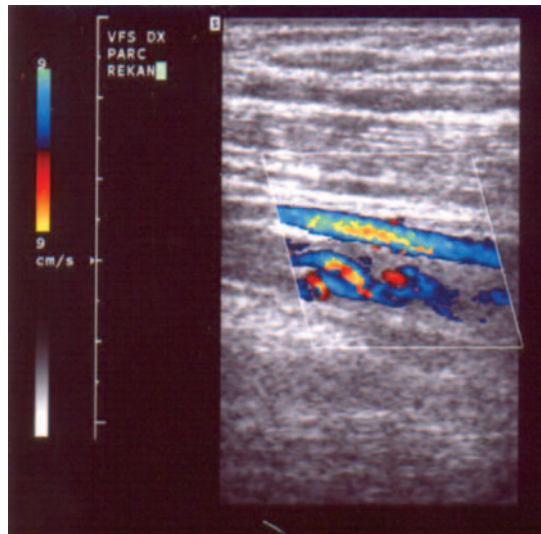
Sometimes it can be difficult on the basis of conventional B-mode alone to differentiate the chronically changed vein from the surrounding soft tissue. Chronic DVT often results in only partial recanalisation with destruction of the valves and developing chronic deep venous insufficiency and reflux.

Both complete and partial DVT can result in pulmonary embolism and it is the most serious complication of DVT. Most often, the source of pulmonary emboli is in diffuse DVT of the iliofemoral region. Isolated focal calf DVT would rarely result in PE. The propagation of the thrombus from the calf into the proximal segments of the upper-thigh appears in approximately 20% of the cases. Whether the calf veins should be routinely examined during examination and the isolated calf thrombosis treated remains the subject of debate. More recently, calf veins are examined in most institutions. But they should definitely be examined if the patient specifies pain or discomfort in that area [13,22,23].

### Treatment and follow-up of DVT patients

Basic treatment for DVT is anticoagulant therapy. Initial parenteral treatment with heparin is followed with oral anticoagulant therapy. Close monitoring of coagulation tests during therapy is mandatory. It is believed that early and appropriate treatment lowers the risk of pulmonary embolic incidents and the risk of developing chronic venous insufficiency [13,19,20,24]. In a third of DVT cases complete restitution of flow is present and in the other two-thirds damage of the valves with some deep venous insufficiency develops. Colour Doppler ultrasound allows the assessment of the recanalisation process (Figure 10) or eventual propagation of the thrombus as well as assessment of possible complications [2,13,25].

Figure 10 Partial recanalisation of the femoral vein during therapy.



For iliofemoral DVT, it is recommended to examine the inferior vena cava to exclude tumour and compression from other adjacent organs as a cause of DVT, as well as to assess the possibility for cava filter placement [2].

**Other pathological conditions that can clinically mimic DVT**

One of the great advantages of ultrasound is that it is an excellent tool for analysing structures and organs adjacent to the veins. There are a number of pathological conditions that can cause clinical symptoms that are similar to DVT. The clinical diagnosis of DVT is non-specific and it is estimated to be correct in less than 50% of patients. Because of this it is extremely important to recognise other differentials to provide the patient with appropriate treatment [2,27].

The most common pathological conditions that mimic DVT include simple or complicated popliteal cysts (Figure 11), haematomas (Figure 12) in the muscle or ruptures of muscles, muscular abscesses (Figure 13), different vascular anomalies, pseudoaneurysms (Figure 14) either spontaneous or iatrogenic, benign or malignant tumours (Figure 15), inguinal lymphadenopathy and generalised oedema of the soft tissue due to different causes. In the inguinal region iliopsoas bursitis can mimic DVT (Figure 16). All these pathological states should be kept in mind when examining patients with suspected DVT [2,27].

Figure 11 Ruptured popliteal cyst, dual imaging.

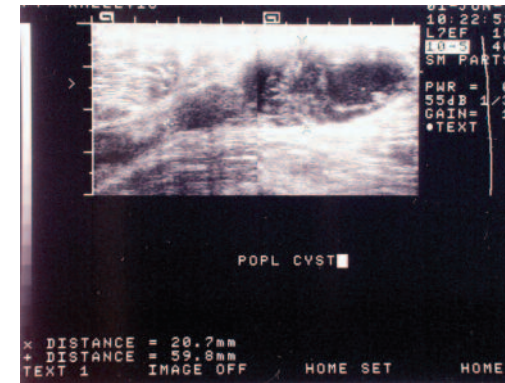


Figure 12 Haematoma in the gastrocnemius muscle.

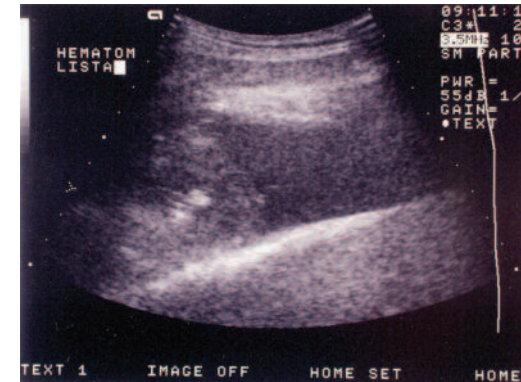


Figure 13 Abscess in lateral vastus muscle.

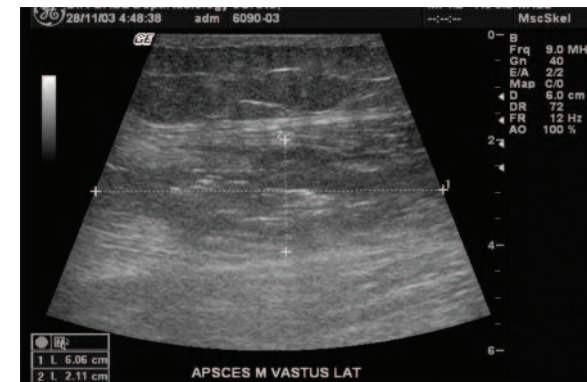




Figure 14 Iatrogenic pseudoaneurysm of peroneal artery.

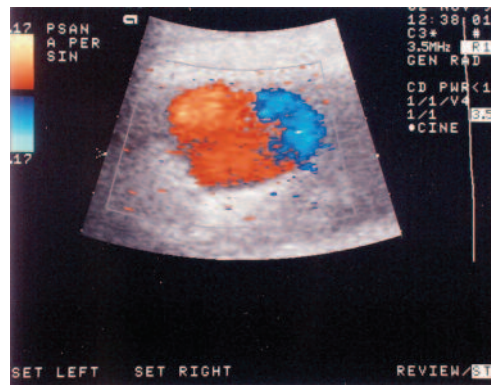


Figure 15 Dual imaging, large sarcoma of the muscles of the calf; the patient was referred to ultrasound to rule out DVT.

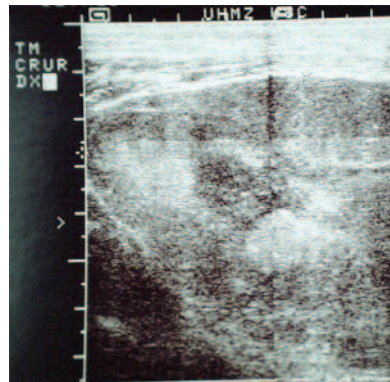
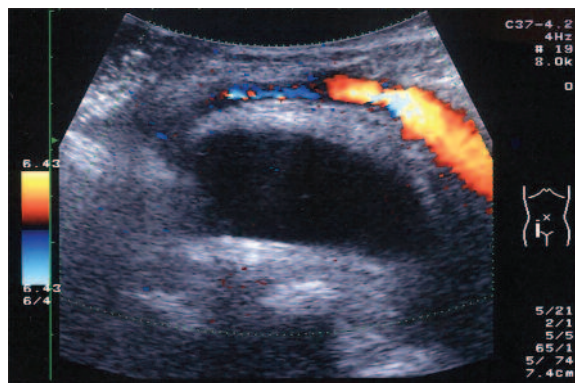


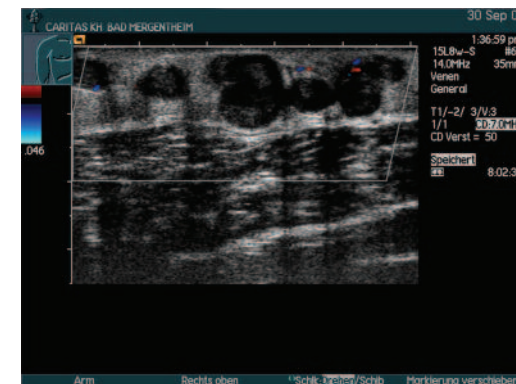
Figure 16 Iliopsoas bursitis clinically mimicking DVT.



**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 cosmetically displeasing 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 that are usually present in the medial side of the leg. The underlying cause of varicose veins is chronic venous obstruction and/or valvular incompetence. They may thrombose and cause symptoms of superficial thrombophlebitis (Figure 17).

Figure 17 Thrombosed varicose vein.



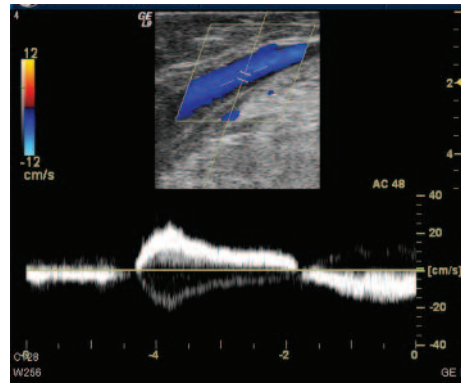
Aetiologically, they can be primary when no venous obstruction is present and dilatation and valvular failure is a result of increased hydrostatic pressures. Primary varicose veins are more common in female patients, overweight patients and in patients in professions involving standing for long periods 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. Prolonged standing, pregnancy and obesity all lead to increased hydrostatic pressures that can cause chronic venous distension and secondary valvular incompetence anywhere within the superficial venous system. If proximal junction valves become incompetent, high pressure passes from the deep veins into the superficial veins. Thus, the normal scenario in which one-way valves direct the flow of venous blood upward and inward from small 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 or 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. DVT initially produces an obstruction to the flow, but recanalisation is often not complete, the “scarring” occurs with consequent valvular damage and the vein finally becomes a valveless channel that delivers high pressures from above. With colour duplex Doppler it is possible to detect and grade valvular incompetence. The patient is asked to perform a Valsalva manoeuvre. In normal veins abrupt cessation of the blood flow is observed following a brief deflection produced by valve closure. After Valsalva release,



flow augmentation toward the heart is detected. Valvular incompetence is indicated by sustained retrograde flow induced by the Valsalva manoeuvre (Figure 18). Reflux can also be tested with probe compression on the vein when, after sudden release of compression, reversed flow persists and indicates incompetent valves. Valvular incompetence can be graded using the time of reflux duration. Less than 0.5 seconds of reversed flow is considered within normal range and if it lasts longer than 0.5 seconds this presents a pathological finding indicating reflux [2,28,29].

Figure 18 Valvular incompetence is indicated by sustained retrograde flow induced by Valsalva manoeuvre



Ultrasonographic evaluation of varicose veins is very important prior to planned surgery. The 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. The saphenofemoral junction is assessed for competency and its position is noted; the femoral vein is examined to exclude prior DVT. The course of the great saphenous vein and its major tributaries is followed by noting their size and whether there is any reflux. Perforator communication between the greater saphenous vein and the deep system is identified and their competency assessed. A perforator diameter of more than 4 mm is incompetent (Figure 19) 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 20).

Figure 19 Very dilated Dodd's perforator, measuring 11 mm in transverse diameter.

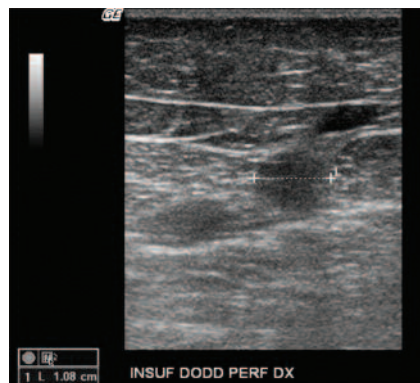
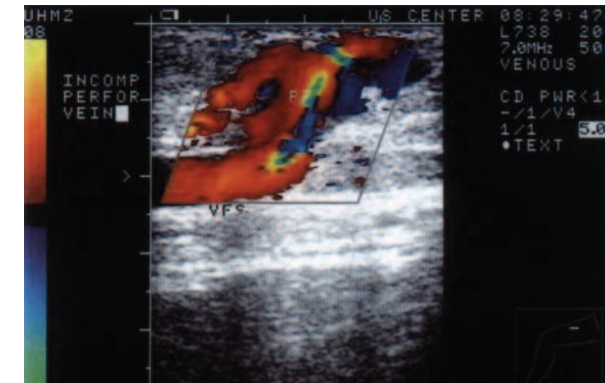


Figure 20 Colour Doppler indicates reverse flow through the dilated perforator.

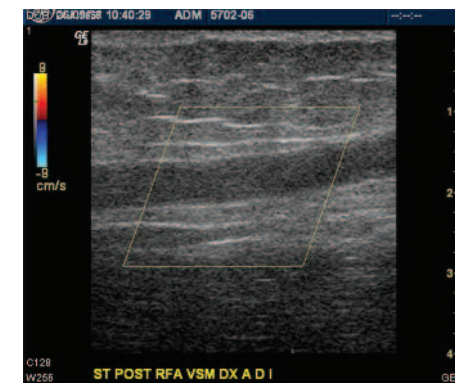


The location of the perforator veins in relation to the varicosities is noted because incompetent segments can be easily traced directly to the varicosities.

The patient is then turned away and the lesser saphenous vein is examined. The location of the termination of the lesser saphenous vein is established. The size and competency of the saphenopopliteal junction is assessed and its relationship to posterior calf varicosities is noted. The popliteal vein should be examined to exclude prior DVT and evidence of deep vein reflux.

Varicose veins can be treated conservatively, by sclerotherapy, laser ablation, RF ablation and surgical extirpation. Success of the procedure can be monitored by ultrasound (Figure 21). 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). It is also important to note the position of dilated perforator veins and 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 finding of the great saphenous vein. Deep reflux should be tested as well as possible perforator dilatation and reflux [2,28,29].

Figure 21 Completely thrombosed greater saphenous vein after successful radiofrequency ablation.

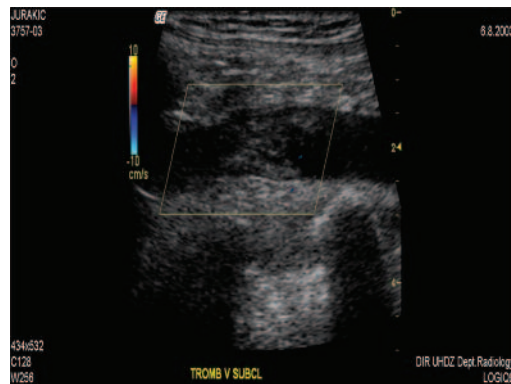


### Upper-extremity-veins and jugular vein examination

Thrombosis of the upper-extremity veins is far less common than the lower extremities. The most common is subclavian vein thrombosis, which can be associated with strain during exercise, thoracic outlet obstruction syndrome, underlying hypercoagulation state or iatrogenic complication. Colour duplex Doppler has a high specificity and sensitivity to detect upper-extremity venous thrombosis of 92% and 78%, respectively. The subclavian vein, axilar and brachial veins, as well as superficial veins (cephalic and basilic vein) should all be examined.

As the subclavian vein can be only partially compressed during examination the respiratory variability of lumen diameter and filling with flow on colour Doppler study are the main signs of its normal patency. In cases of acute thrombosis the subclavian vein is distended, without colour filling and often, especially in partial thrombosis, the thrombus can be clearly visible (Figure 22).

Figure 22 Acute thrombosis of the subclavian vein.



There is no response to changes of intrathoracic pressure induced by the Valsalva manoeuvre or sniffing test, or it is greatly diminished. In the case of loss of respiratory spectral undulation the proximal obstruction at the level of superior vena cava should be suspected.

The internal jugular vein is situated laterally from the common carotid artery in the neck. Its complete or partial thrombosis can be caused by iatrogenic procedures, hypercoagulation states (malignant diseases or sepsis) or it is 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 23). As it can be used for autogenic tissue reconstruction of the carotid artery, its integrity can be easily pre-operatively assessed by colour Duplex ultrasound [30–32].

Figure 23 Thrombosis of internal jugular vein.



## References

1. Gooding GAW. Ultrasound of deep venous thrombosis. In: Goldberg BB, Pettersson H, Eds, *Ultrasonography*. NICER, Oslo 1996;583-611.
2. Brkljačić B. Doppler of peripheral veins. In: Brkljačić B. *Vascular Ultrasound*. Medicinska naklada, Zagreb 2010; pp. 113-60
3. Di Minno G, Mannucci PM, Tufano A, et al. The first ambulatory screening on thromboembolism: a multicenter, cross-sectional, observational study on risk factors for venous thromboembolism. *J Thromb Hemost* 2005;3(7):1459-66.
4. Oger E, Mottier D. Incidence and risk factors for venous thromboembolism. *Rev Prat* 2007;57(7):711-20.
5. Zurawska U, Parasuraman S, Goldhaber SZ. Prevention of pulmonary embolism in general surgery patients. *Circulation* 2007;115(9):302-7.
6. White RH. The epidemiology of venous thromboembolism. *Circulation* 2003;107:4-8.
7. Cronan JJ. Controversies in venous ultrasound. *Semin Ultrasound CT MR* 1997;18:33-8.
8. Lewis BD, James EM, Welch TJ et al. Diagnosis of acute deep venous thrombosis of the lower extremities: prospective evaluation of colour Doppler flow imaging versus venography. *Radiology* 1994;192:651-5.
9. Cronan JJ. Controversies in venous ultrasound. In: Zwiebel WJ, Pellerito JS (Ed) *Introduction to Vascular Ultrasonography*. Elsevier-Saunders, Philadelphia 2005; pp. 467-478.
10. Goodacre S, et al. Systematic review and meta-analysis of the diagnostic accuracy of ultrasound for DVT. *BMC Med Imaging* 2005;5:6
11. Rose SC, Zwiebel WJ, Nelson BD et al. Symptomatic lower extremity deep venous thrombosis: accuracy, limitations and role of colour duplex flow imaging in diagnosis. *Radiology* 1990;175:639-44.
12. Singh RS, Galt SW. The role of ultrasound in the management of extremity venous disease U: Zwiebel WJ, Pellerito JS (Ur) *Introduction to Vascular Ultrasonography*. Elsevier-Saunders, Philadelphia 2005; pp. 403-414.
13. Gaitini D. Current approaches and controversial issues in the diagnosis of DVT via duplex Doppler ultrasound. *J Clin Ultrasound* 2006;34(6):289-97.
14. Subramaniam RM, Snyder B, Heath R, Tawse F, Sleigh J. Diagnosis of lower limb deep venous thrombosis in emergency department patients: performance of Hamilton and modified Wells scores. *Ann Emerg Medicine* 2006; 48(6):678-85.
15. Michiels JJ, Gadisseur A, Van der Planken M. A critical appraisal of noninvasive diagnosis and exclusion of DVT and PE in outpatients with suspected DVT and PE: how many tests do we need? *Int Angiol* 2005;24(1): 27-39.
16. Effeney DJ, Friedman MB, Gooding GAW. Real-time ultrasound in the diagnosis of iliofemoral venous thrombosis: real-time ultrasound diagnosis, normal criteria and clinical application. *Radiology* 1984;150:787-92.
17. Polak JF, Culter SS, O'Leary DH. Deep veins of the calf: assessment with colour Doppler flow imaging. *Radiology* 1989;171:481-5.
18. Rose SC, Zwiebel WJ, Miller FJ. Distribution of acute lower extremity deep venous thrombosis in symptomatic and asymptomatic patients: Imaging implications. *J Ultrasound Med* 1994;13:243-50.
19. Gottlieb RH, Widjaja J. Clinical outcomes of untreated symptomatic patients with negative findings of sonography of the thigh for deep venous thrombosis: our experience and review of the literature. *Am J Roentgenol* 1999;172(6):1601-4.
20. Geerts WH, Pineo GF, Heit JA, et al. Prevention of venous thromboembolism: the seventh ACCP conference on antithrombotic and thrombolytic therapy. *Chest* 2004;126(3): 338S-400S
21. Kassai B, Boissel JP, Cucherat M, Sonie S, Shah NR, Leizorovicz A. A systematic review of the accuracy of ultrasound in the diagnosis of deep venous thrombosis in asymptomatic patients. *Thromb Haemost* 2004;91(4):655-66.
22. Elias A. Extended lower limb venous ultrasound for the diagnosis of proximal and distal vein thrombosis in asymptomatic patients after total hip replacement. *Eur J Vasc Endovas Surg* 2004;27(4):438-44.
23. Cronan JJ. Deep venous thrombosis – one leg or both legs? *Radiology* 1996;200(2):323-4.
24. Prandoni P, Noventa F, Ghirarduzzi A, et al. The risk of recurrent venous thromboembolism after discontinuing anticoagulation in patients with acute proximal DVT or pulmonary embolism. A prospective cohort study in 1626 patients. *Haematologica* 2007; 92(2):199-205.
25. Frieria A, Gimenez NR, Caballero P, Mollini PS, Suarez C. Deep vein thrombosis: can a second ultrasound examination be avoided. *Am J Roentgenol* 2002;178(4): 1001-5.
26. Elias A, Mallard L, Ellias M, et al. A single complete ultrasound investigation of the venous network for the diagnostic management of patients with a clinically suspected first episode of DVT of the lower limbs. *Thromb Haemost* 2003;89(2):206-7.
27. Brkljačić B, Mišević T, Huzjan R, Brajčić H, Ivanac G. Duplex-Doppler ultrasonography in the detection of lower extremities deep venous thrombosis and in the detection of alternative findings. *Coll Antropol* 2004;28(2):761-767.
28. Athanasoulis CA, Yucel EK. Venous reflux: assessing the level of incompetence. *Radiology* 1990;174:326-7.
29. Masuda EM, Kistner RL, Ekolof B. Prospective study of duplex scanning for venous reflux: comparison of Valsalva and pneumatic cuff technique in the reverse Trendelenburg and standing positions. *J Vasc Surg* 1994;20:711-20.
30. Gooding GAW, Woodruff A. Colour Doppler imaging in the subclavian-axillary region and upper extremity. *Clin Imag* 1994; 18:165-172.
31. Gaitini D, Kaforti JF, Pery M, Engel A. high-resolution real-time ultrasonography. Diagnosis and follow-up of jugular and subclavian vein thrombosis. *J Ultrasound Med* 1998;7:621-7.
32. Kundson GJ, Wiedermeyer DA, Erickson SJ, i dr. Colour Doppler sonographic imaging in the assessment of upper-extremity deep venous thrombosis. *Am J Roentgenol* 1990; 154:399-403.



