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Ultrasound Evaluation of the Lower Extremity Veins

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Over the past 2 decades venous ultrasonography (US) has become the standard primary imaging technique for the initial evaluation of patients for whom there is clinical suspicion of deep venous thrombosis (DVT) of the lower extremity veins. It has replaced the venogram and other diagnostic studies such as impedance plethysmography, various radionuclide studies, and conventional CT because of its noninvasive nature, the ease with which it can be performed in skilled hands, and its proven efficacy. Compression US first was described as a means of diagnosing DVT in 1986 by Raghavendra and colleagues [1] and was introduced into clinical practice in 1987 by Cronan and colleagues [2–7] from the United States and Appleman and colleagues [8] from the Netherlands. In the ensuing years multiple articles were published establishing venous US as the examination of choice for the diagnosis of venous thrombosis, further refining the US technique and diagnostic criteria with gray-scale imaging, and also incorporating the newly developed technique of Doppler US.

This article addresses the role of duplex US and color Doppler US (CDUS) in today's clinical practice for the evaluation of patients suspected of harboring a thrombus in their lower extremity veins. Clinical presentation and differential diagnoses, technique, and diagnostic criteria for both acute and chronic DVT are reviewed. In addition, sonographic evaluation of venous insufficiency is addressed.

Clinical presentation and differential diagnosis

Acute DVT is a significant health problem affecting approximately 2 million people in the United States

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per year. Pulmonary embolism (PE) is the most dreaded complication of an untreated DVT and is reported to occur in about 50% to 60% of untreated cases with a 25% to 30% mortality rate [4,9–12]. Predisposing factors for DVT include prolonged bed rest, congestive heart failure, prior DVT, pelvic and lower extremity surgery, coagulopathies, immobilization, trauma, pregnancy, malignancies, indwelling catheters, intravenous drug abuse, dehydration, travel (probably because of prolonged immobility), and endothelial injury. In addition, oral contraceptive use, obesity, and systemic diseases such as systemic lupus erythematosus, lupus anticoagulant, nephritic syndrome, Behçet's disease, nocturnal hemoglobinuria, polycythemia vera, and hyperviscosity syndromes may predispose a patient to thromboembolism formation.

The signs and symptoms of DVT often are nonspecific, and thus the clinical diagnostic accuracy is poor. Extremity swelling, pain, and edema place the patient at risk for DVT. Significant thrombus can be present in asymptomatic patients, however. The clinical accuracy for the diagnosis of acute DVT has been reported to be about 50% in symptomatic patients [13–16]. Localized pain and calf tenderness are reported in approximately 50% of cases, but pain on forced dorsiflexion of the toes, the so-called "Homan's sign," is an unreliable diagnostic criterion and is present in only 8% to 30% of symptomatic patients harboring a DVT. Furthermore, the Homan's sign can be elicited in nearly 50% of symptomatic patients who do not have a DVT as a cause of their symptoms [4,17]. A palpable cord caused by a thrombosed superficial vessel can be present with or without involvement of the deep venous system [4]. US examination is highly sensitive and specific for the diagnosis of DVT: several series have reported sensitivities and specificities approaching 95% to 100% for the diagnosis of DVT in the proximal lower extremity. The most common causes for false-negative examinations are venous duplication, infrapopliteal thrombus, and nonocclusive focal or segmental DVT. Therefore, expeditious sonographic evaluation of patients suspected of having DVT may aid in rapid initiation of therapeutic measures to decrease the potential for developing complications such as PE or postthrombotic venous insufficiency. The limitations of US in accurately diagnosing calf vein thrombosis and DVT in obese patients is addressed later.

Examination technique and normal sonographic anatomy

The choice of transducer for evaluating the lower extremity veins depends on the patient's body habitus and the depth of the vessel to be studied. The examination is preferentially performed with a high-resolution 5- to 7.5-MHz linear array transducer. For very superficial veins (ie, the greater or small saphenous veins), a 10-MHz transducer may

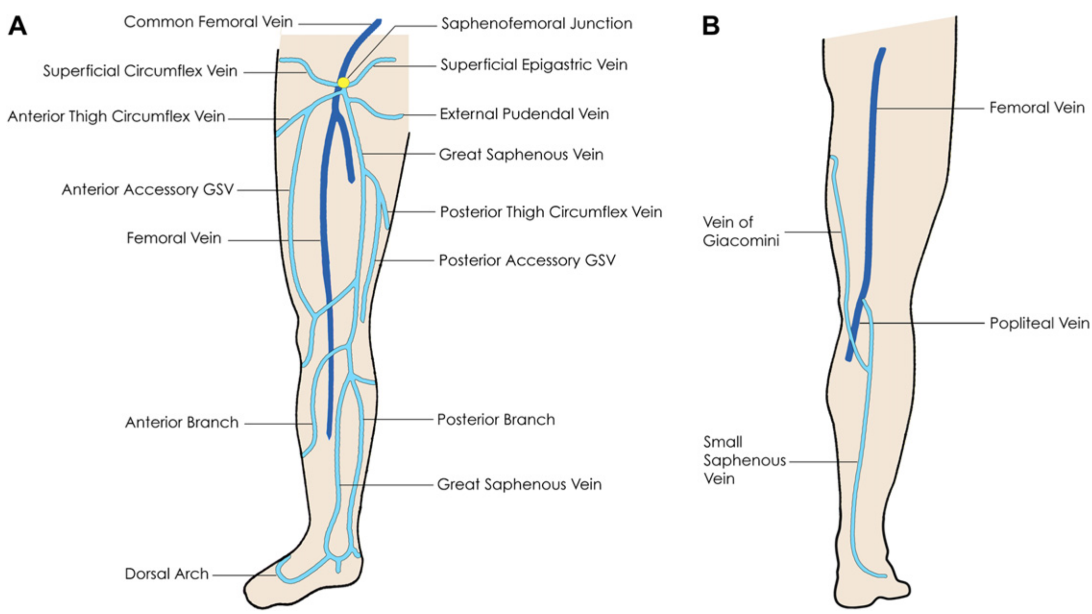

Fig. 1. (*A*) The great saphenous vein and its major tributaries. (*B*) The small saphenous vein. (*Adapted from* Weiss RA, Feied CF, Weiss MA. Vein diagnosis and treatment. New York: McGraw-Hill; 2001.)

be optimal. For large patients a lower-frequency (2.5- or 3.5-MHz) curvilinear transducer may be necessary. In general, the transducer with the highest frequency allowing adequate penetration provides the best spatial resolution. Appropriate gain settings must be applied to ensure that the vessels are free of artifactual internal echoes and that thrombus is not mistaken for echoes caused by slow-flowing blood. In addition, CDUS imaging parameters must be optimized for sensitivity to slow, low-volume flow.

The lower extremity venous system consists of a superficial system, the saphenous veins and their tributaries, and a deep system. A more detailed description of the superficial venous plexus is presented later in the section discussing venous insufficiency. Briefly, the great saphenous vein (GSV) joins the common femoral vein (CFV) just superior to its bifurcation. It travels medially in the thigh and calf extending inferiorly to the level of the foot (Fig. 1A). It measures about 3 to 5 mm in diameter at the level of the saphenofemoral junction, tapering to about 1 to 3 mm in diameter at the level of the ankle (see Fig. 1A). Measurement of the GSV is important before harvesting for autologous vein grafting. The small saphenous vein (SSV) (formerly called the "lesser saphenous vein") extends from the ankle along the posterior aspect of the calf to insert at variable levels into the posterior proximal or mid popliteal vein (PV) (Fig. 1B). The diameter of the SSV tapers from 2 to 4 mm proximally to about 1 to 2 mm distally. Both the GSV and SSV may become enlarged and varicose in patients who have venous insufficiency or congestive heart failure. Thrombosis of these vessels may occur with ensuing superficial thrombophlebitis (Fig. 2).

The deep venous system includes the CFV, the deep femoral or profunda femoris vein, the femoral vein (FV) (previously called the "superficial femoral

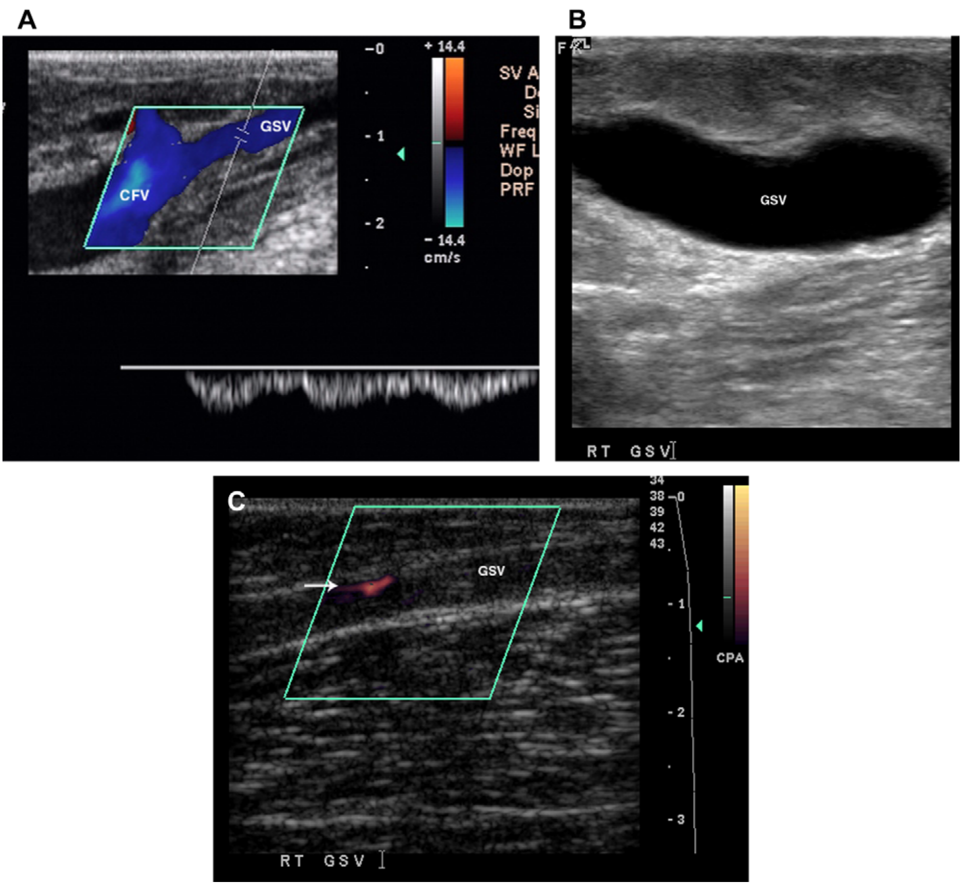

Fig. 2. (*A*) Normal greater saphenous vein/common femoral vein junction—Doppler gate in greater saphenous vein. (*B*) Large greater saphenous vein in a patient who has congestive heart failure. (*C*) Sagittal US of thrombosed greater saphenous vein with only a trickle of peripheral flow (*arrow*).

vein"), and the PV as well as the paired anterior and posterior tibial veins and the peroneal veins (Fig. 3). The CFV is the continuation of the external iliac vein and extends from the level of the inguinal ligament to the level of the bifurcation into the FV and profunda femoris vein. The profunda femoris vein lies medial to the accompanying artery and can be evaluated only in its most proximal portion. The CFV lies medial to the accompanying artery. The FV travels medial to the femoral artery through the adductor canal in the distal thigh. To avoid confusion among clinicians, the term "femoral vein" (FV) should be used instead of the previously designated term "superficial femoral vein" to describe that part of the deep venous system caudal to the bifurcation of the CFV. (In a survey performed by Bundens and colleagues [18], more than 70% of surveyed internists regarded the "superficial femoral vein" as part of the superficial and not the deep venous system and thus were under the assumption that superficial femoral vein thrombosis did not require treatment.) The PV is the continuation of the FV after its exit from the adductor canal in the posterior distal thigh. The PV is located anterior to its accompanying artery and courses through the popliteal space into the proximal calf. In general, the deep veins in the thigh are wider in diameter

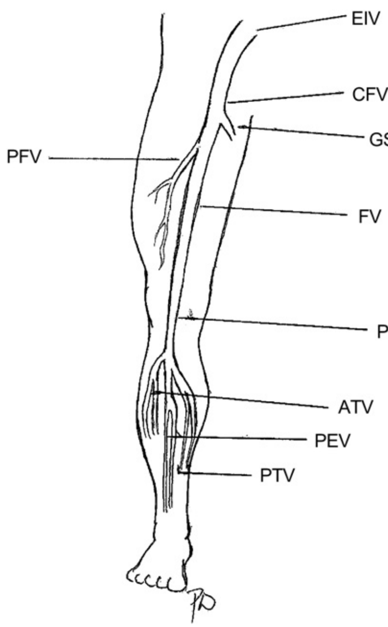

Fig. 3. The anatomy of the deep venous system. ATV, anterior tibial veins; CFV, common femoral vein; EIV, external iliac vein; FV, femoral vein; GSV, greater saphenous vein; PEV, peroneal veins; PFV, profunda femoris vein; PTV, posterior tibial veins; PV, popliteal veins.

than the accompanying artery unless the veins are duplicated. Duplication of the FV or PV can be seen in 20% to 35% of patients. Duplication of the FV can be segmental. It is important to mention these anatomic variants so that thrombosis in one of the duplicated limbs is not missed (Fig. 4). The paired anterior tibial veins are accompanied by their corresponding artery and arise from the popliteal vein traversing the anterior calf compartment along the interosseous membrane to the dorsum of the foot. The tibioperoneal trunk originates from the PV slightly distal to the anterior tibial veins and bifurcates into the paired posterior tibial veins and peroneal veins , both of which are accompanied by their respective arteries. The peroneal veins course medial to the posterior aspect of the fibula, whereas the posterior tibial veins course through the posterior calf muscles posterior to the tibia and along the medial malleolus. In addition, the muscles of the calf are drained by gastrocnemius and soleus veins, which do not have accompanying arteries.

In a patient suspected of having DVT, the deep venous system should be evaluated from just above the inguinal ligament to the trifurcation of the popliteal vein in the upper calf, every 2 to 3 cm, with compression. In addition, evaluation of the external iliac veins and calf veins, including anterior and posterior tibial veins and peroneal veins, can be performed. The iliac vein, CFV, and FV are best evaluated with the patient in a supine position (Fig. 5A). The popliteal veins can be studied with the patient supine and the leg flexed 20° to 30° and externally rotated or in a decubitus position with the study side up (Fig. 5B). The anterior tibial veins are best identified initially at the posterior medial ankle. When followed up the leg, the peroneal veins, which originate from the lateral ankle, are posterior to the anterior tibial veins. The anterior tibial veins run between the tibia and fibula anteriorly.

According to the American College of Radiology practice guidelines and technical standards, lower extremity Duplex sonography should include compression, color, and spectral Doppler sonography with assessment of phasicity and venous flow augmentation [19]. A complete examination includes evaluation of the full length of the CFV and saphenofemoral junction, the proximal portion of the deep femoral vein, the FV, and the PV. These veins are imaged in the transverse plane with and without compression with mild transducer pressure every 2 to 3 cm from the level of the CFV to the distal FV in the adductor canal and the PV to the popliteal trifurcation. In obese patients compression in the adductor canal may be difficult. Sometimes a

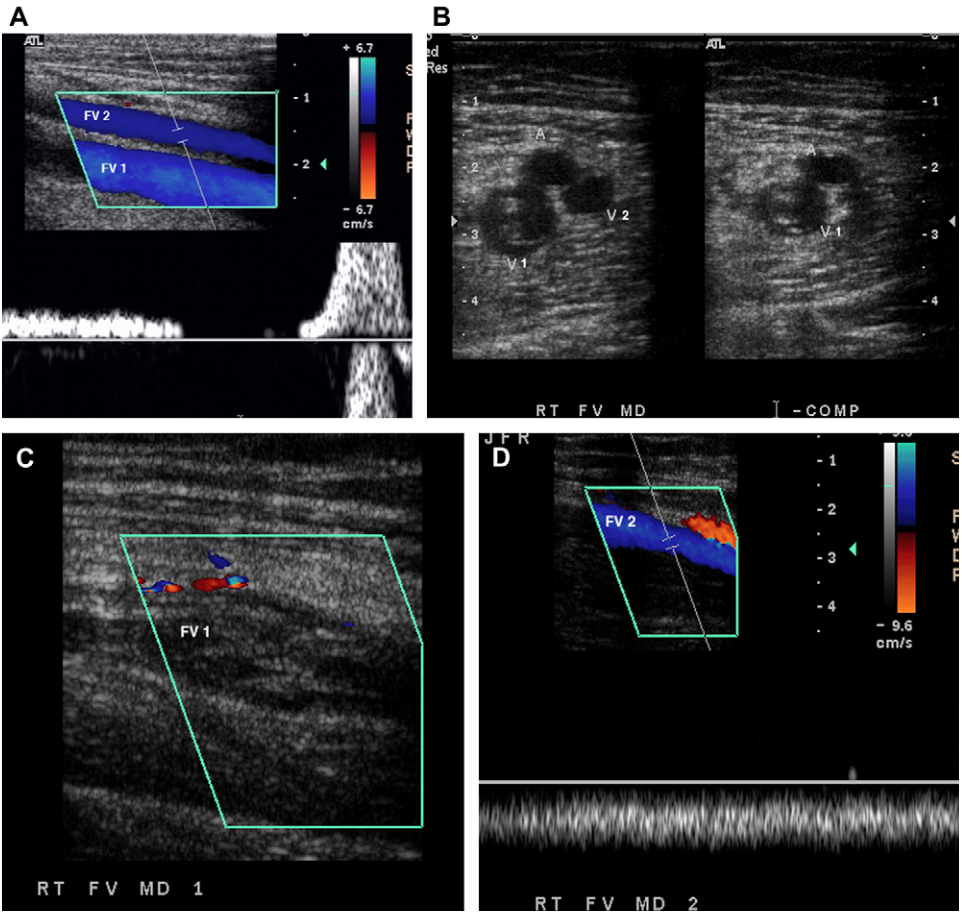

Fig. 4. (*A*) Sagittal color Doppler US of duplicated femoral vein (*FV*). (*B*) Transverse US of duplicated femoral vein demonstrating noncompressibility of thrombosed limb (*V1*). (*C*) Sagittal color Doppler US limb of a duplicated femoral vein (*FV1*). (*D*) Sagittal color Doppler US of normal flow in duplicated femoral vein (*FV2*).

two-hand technique, pushing the leg into the transducer with the free hand from behind, may help achieve adequate compression (Fig. 5C). A normal pulse of color flow Doppler signal in a suboptimally compressed or poorly visualized segment is reassuring of at least partial patency and excludes the possibility of occlusive thrombus. Compression of a normal vein completely collapses the venous lumen (Figs. 6 and 7), whereas the presence of DVT prevents coaptation of the vein walls (see Figs. 6 and 7). In the authors' laboratory, color flow Doppler US is used routinely because it speeds the identification of the vessels and facilitates identification of deeply located vessel segments. Color should fill the entire lumen of normal veins, but color flow is diminished or absent in partially or completely thrombosed veins (Fig. 8). Sometimes, particularly in veins with slow flow, squeezing the calf will augment venous flow, resulting in complete color filling of a vessel. Doppler spectral analysis is performed to assess phasicity and augmentation responses following squeezing of the calf or plantar flexion of the foot and is helpful as a secondary means of assessing patency of the more proximal veins, particularly the iliac veins (see Fig. 8E, F). If a thrombus is identified in any segment of the veins, augmentation is not performed to avoid detaching the clot and thereby causing a PE. Because of the risk of superficial thrombophlebitis extending into the deep venous system, the authors also routinely study the GSV at its junction with the CFV (see Fig. 8F–H). Because of its increased sensitivity to low-velocity, low-volume flow, pwer Doppler US sometimes is useful in identifying early recanalization of thrombosed veins or in detecting minimal flow in nearly occluded venous segments, deep vessels, or the smaller calf veins. Power Doppler, however, does not indicate directionality of flow and is affected adversely by patient motion or calcifications.

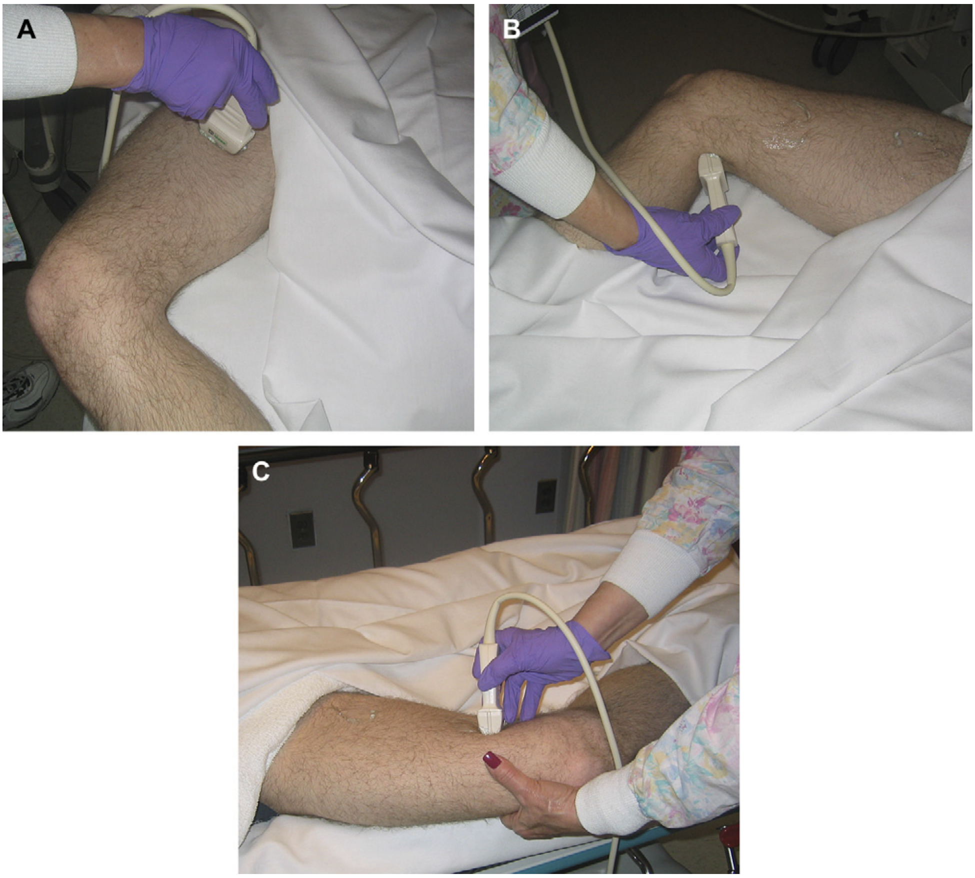

Fig. 5. (*A*) Transverse scan demonstrating the imaging technique of the femoral vein with the patient in a supine position. (*B*) Transverse scan demonstrating the imaging technique of the popliteal vein with the leg flexed and externally rotated. (*C*) "Two-hand technique" to visualize the femoral vein (FV) in the adductor canal.

The normal veins are easily compressible by slight transducer pressure (see Figs. 6 and 7). If one cannot compress the veins because of swelling or pain, one can attempt indirect evaluation using a Valsalva maneuver, observation of the presence or absence of respiratory phasicity, and augmentation techniques. Following a Valsalva maneuver, a normal vein expands to more than 50% of its diameter from baseline. The Valsalva maneuver has the most pronounced effect on the veins near the inguinal ligament. Proximal pelvic DVT or external compression of the pelvic veins blunts the normal dilatation of the CFV following a Valsalva maneuver. Evaluation of respiratory phasicity and augmentation also can provide indirect assessment of adjacent vein segments. Proximal (cephalic) obstruction caused by pelvic venous thrombosis or external compression of the pelvic veins by ascites, pelvic mass, or hematoma dampens respiratory phasicity in the CFV (Fig. 9). Distal (caudal) DVT blunts the normal augmentation of venous flow achieved by squeezing the leg inferior to the point of insonation. Valsalva maneuvers also can be used to evaluate venous insufficiency. With the patient supine, a temporary reversal of flow occurs if the valves are incompetent. (This phenomenon is discussed in more detail at the end of the article). Likewise changes in the venous spectral tracing can indicate more proximal cardiovascular disease. For example, in patients who have elevated right atrial pressure caused by congestive heart failure or tricuspid insufficiency, the spectral venous waveform can be quite pulsatile, mimicking a waveform more typical of the hepatic veins or inferior vena cava (Fig. 10) [20].

Controversial issues vis-à-vis the examination technique

US evaluation of the calf veins remains controversial because of its unproven clinical value and cost effectiveness, and the American College of Radiology practice guidelines do not require their evaluation. It has been shown that isolated calf vein

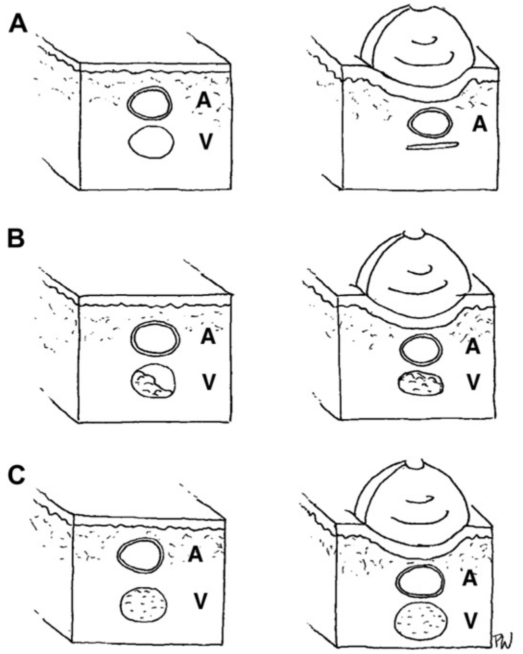

Fig. 6. Transverse compression of normal and thrombosed veins. (*A*) Compression of a normal vein completely collapses the venous lumen. (*B*) Compression of a nonocclusive, thrombosed vein partially collapses the venous lumen. (*C*) Compression of a completely thrombosed vein prevents venous coaptation.

thrombosis rarely causes significant pulmonary emboli and thus is assumed to be a benign, self-limiting disease [21]. Hence, patients who have isolated calf DVT rarely receive anticoagulation therapy in clinical practice because the risk of bleeding from the anticoagulation therapy is considered to be higher than the risk of PE even though several studies have shown that approximately 20% of calf DVTs ultimately extend into the more superior deep venous system in the thigh [21]. In addition, US evaluation of the calf veins is less accurate than evaluation of veins in the thigh because the veins in the calf are much smaller; therefore it is difficult to be certain that all of the calf vessels, some of which are duplicated, are free of thrombus. Other studies stress the clinical importance of calf vein thrombosis and argue that the 4% to 10% risk of potential bleeding with anticoagulation therapy is safer than the 20% to 30% risk of propagation and potential 5% risk for development of PE [22,23]. In a study by Badgett and colleagues [24], a complete venous examination including calf veins altered clinical management in about 30% of patients. The practice in the authors' laboratory is to evaluate the calf veins only in patients who have focal, persistent, or worsening calf symptoms.

Another controversy in the radiologic and surgical literature centers around the necessity of routinely performing bilateral lower extremity venous US examinations versus the appropriateness of performing a unilateral examination if symptoms are limited to one leg. Some studies recommend routine performance of bilateral venous US studies of the lower extremities because thrombus can occur in either leg in up to 23% of patients who have bilateral symptoms [25]. A study by Cronan [3] concluded that the likelihood of finding DVT in patients who have bilateral lower extremity symptoms is related to their predisposing risk factors, and that bilateral lower extremity sonography is indicated only in patients who have a risk factor for DVT, not in those who are at risk only for cardiac disease.

Other studies suggest that examination of an asymptomatic leg is unnecessary because, although thrombosis may occur in the asymptomatic leg in up to 14% of patients, these patients typically have DVT in the symptomatic leg as well [25–28].

An additional concern centers on the issue of performing limited examinations. Some authors have suggested a limited compression examination of the FV and PV because of the low rate of reported isolated FV thrombosis. Several studies, however, have shown that omitting the FV and evaluating only the CFVs and PVs would lead to a decrease in sensitivity because isolated thrombosis of the FV occurs in 4% to 6% of cases [26,29]. Another issue concerns the usefulness of performing augmentation in all patients being evaluated for DVT. A study by Lockhart and colleagues [30] questions the usefulness of performing venous flow augmentation maneuvers with duplex sonography because of its lack of clinical benefit and the added discomfort for the patient who has swollen and painful legs. The authors recommend using augmentation as an adjunct only in uncertain cases rather than as a routine diagnostic component of the lower extremity DVT examination.

Finally, the recent development of multislice and/or multidetector techniques in CT has led to a reconsideration of the role of CT in the diagnosis of DVT when included as part of a pulmonary angiography study. A study by Kim and colleagues [31] demonstrated that multislice helical CT detected DVT that had been missed by venous sonography in five patients, but US detected DVT that was missed by CT in one patient. On the other hand, Kim and colleagues reported [31] that indirect multidetector CT is as accurate as sonography in the detection of femoropopliteal DVT and has the advantage over sonography of being able to evaluate the pelvic veins and inferior vena cava. Particularly in a patient undergoing

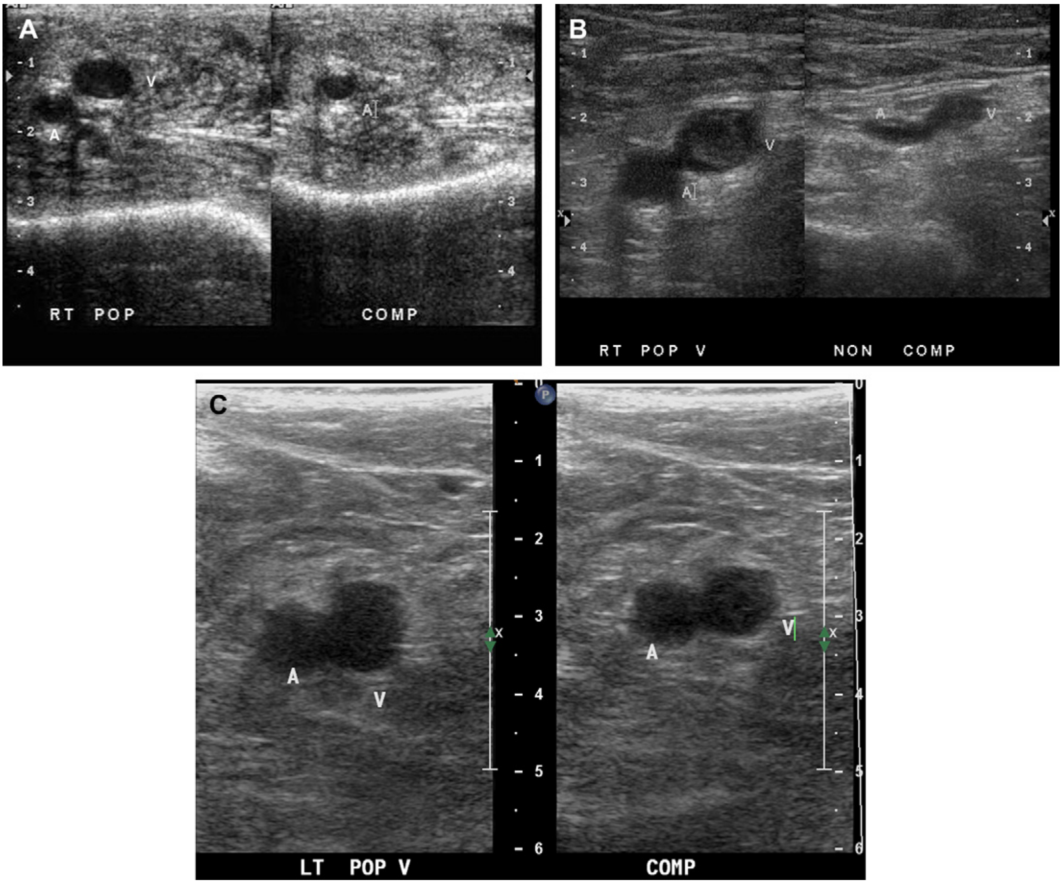

Fig. 7. Transverse sonograms demonstrating compression response of normal and thrombosed veins. (*A*) Compression of a normal popliteal vein completely collapses the venous lumen. Only the popliteal artery (*A*) remains on compression view. (*B*) Compression of a nonocclusive thrombosed popliteal vein partially collapses the venous lumen (*V*). (*C*) Compression of a completely thrombosed popliteal vein prevents venous coaptation.

CT pulmonary angiography in the ICU, the use of indirect CT venography of the lower extremities may be a reasonable alternative to sonography [32]. One must, however, consider carefully the increased radiation dose from multidetector CT when considering the use of this imaging approach to diagnose DVT.

Sonography of acute deep venous thrombosis

The hallmark gray-scale findings of acute DVT include noncompressibility of the vessel as well as direct visualization of the thrombus (see Fig. 6; Figs. 11 and 12). Thrombus may be anechoic or hypo- or hyperechoic. Anechoic or hypoechoic thrombi are thought to be more recent, although the distinction is not precise. Clot echogenicity alone cannot be used to assess the age of a clot [33]. In addition, very slowly flowing blood may be sufficiently echogenic to mimic the appearance of clot (Fig. 13A) [34]. In this instance, however, the compression US is normal (Fig. 13B). Changes in vein caliber with respiration or Valsalva maneuver are lost also when thrombus is present. Thrombi may occlude the lumen completely or partially, and they may be adherent to the wall or free floating (Fig. 14). On CDUS a filling defect can be seen indicating nonocclusive thrombus, or there may be complete absence of flow (Fig. 15).

In a large, pooled series of DVT studies, the accuracy of compression US for DVT has been shown to reach 95% with 98% specificity [4]. In other series sensitivities and specificities range from 88% to 100% and from 92% to 100%, respectively. Additional studies report a sensitivity of 95%, specificity of 99%, and accuracy of 98% for color Doppler flow imaging [35,36]. Sensitivities for isolated calf vein thrombosis are lower, ranging from 60% to 80% [4,37,38]. Protocols and techniques

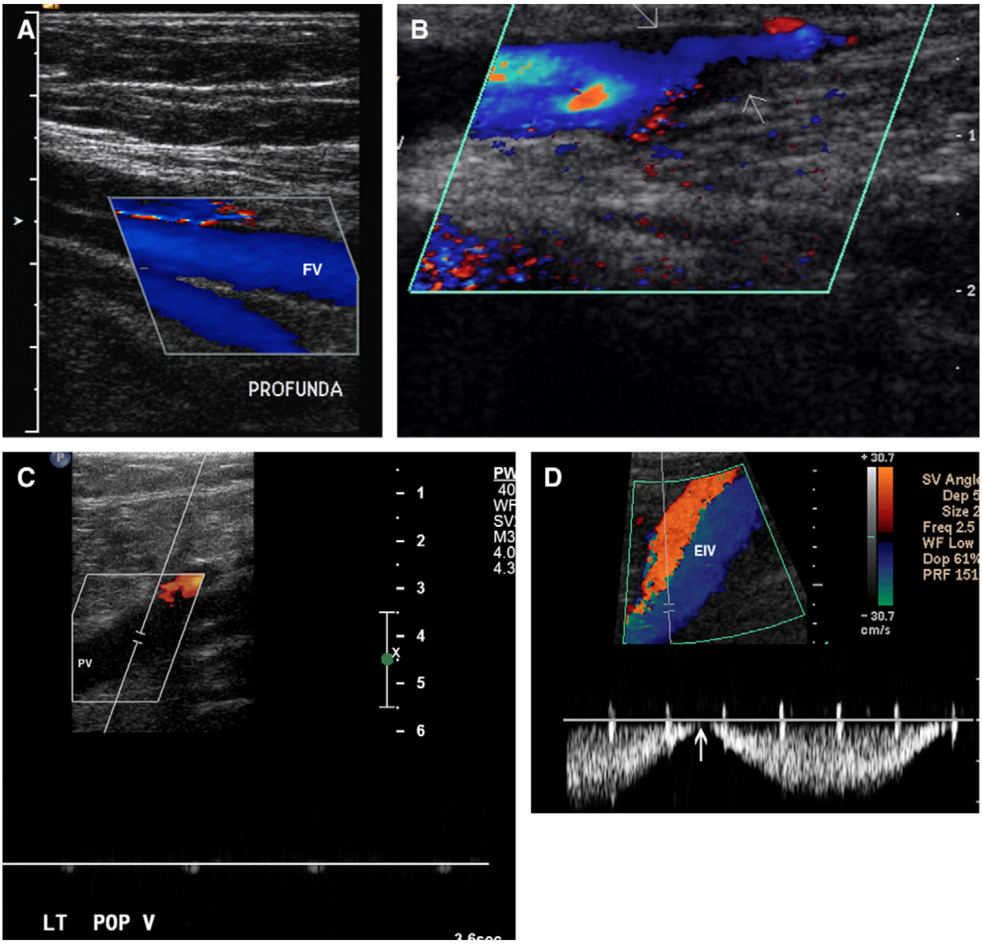

Fig. 8. (*A*) Normal veins demonstrating color flow filling the entire vessel lumen. FV, femoral vein. (*B*) Partial venous thrombosis demonstrating focal absence of color flow (*arrows*). (*C*) Occlusive venous thrombosis demonstrating complete absence of color flow in the popliteal vein. Also note absence of venous waveform on duplex spectrum. (*D*) Normal respiratory phasicity of the right iliac vein. Note return of flow to baseline during inspiration (*arrow*). (*E*) Augmentation maneuver demonstrates surge of flow in right popliteal vein when squeezing the calf (*arrow*). (*F*) Normal color flow Doppler appearance of the junction of the greater saphenous vein (*GSV*) and the common femoral vein (*CFV*). (*G*) Thrombosis of the left greater saphenou vein (*GSV*) near the junction with the common femoral vein (*arrow*). (*H*) Six days later the greater saphenous vein thrombosis has extended into the common femoral vein (*V*) as demonstrated on the transverse compression images.

for evaluation of the calf veins have not been established definitively.

Pitfalls and limitations of diagnosis of acute deep vein thrombosis

The classic clinical signs of DVT cannot be trusted because a variety of abnormalities can mimic the signs and symptoms of acute DVT. Cronan and colleagues [6] showed that in approximately 10% of patients studied for acute DVT, ancillary findings were diagnosed by sonography along with a normal deep venous examination. US can readily distinguish those entities from venous pathology.

A variety of conditions that may cause clinical signs and symptoms indistinguishable from DVT may be caused by other underlying pathology such as adenopathy (groin swelling), arterial aneurysms, or stenoses/occlusions (Fig. 16), soft tissue tumors, hematomas or abscesses, muscle tears, and diffuse soft tissue edema (Fig. 17). Proximal obstruction by an extrinsic mass, such as adenopathy, pseudoaneurysm, or hematoma, may limit the compressibility of veins and produce the same clinical signs of DVT (see Fig. 9B; Fig. 18). Simple

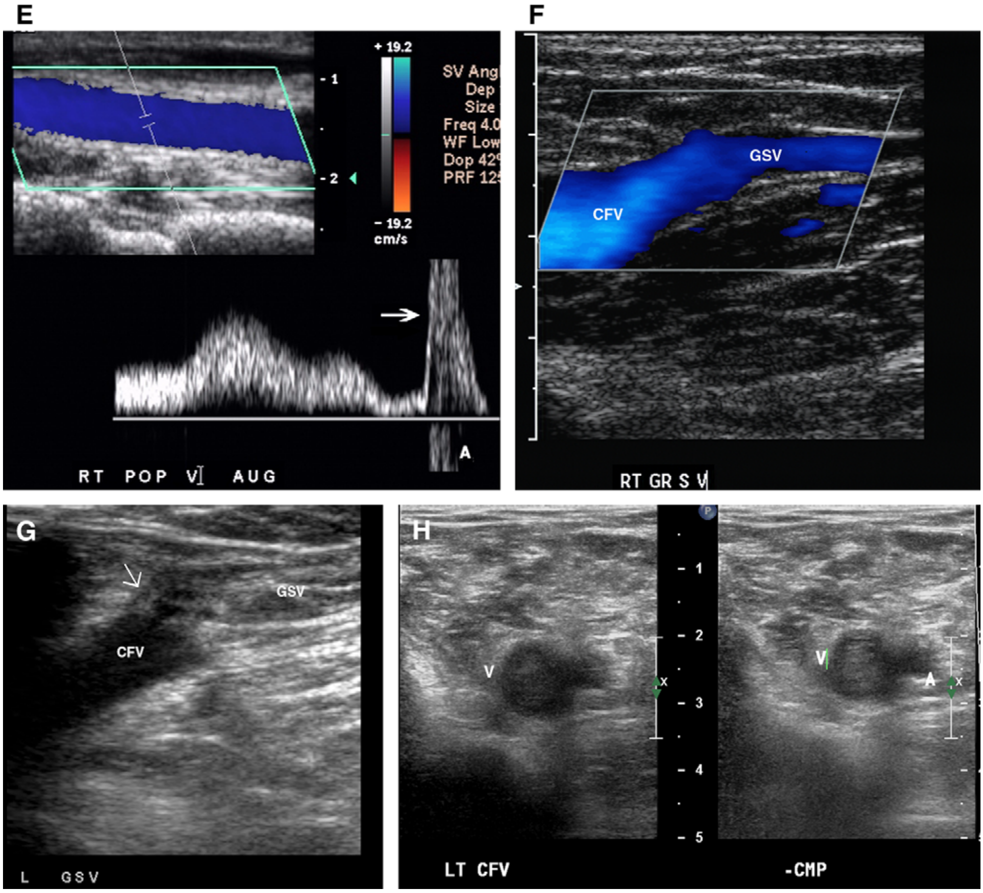

Fig. 8 (*continued*)

popliteal synovial cysts or cysts that communicate with the posterior bursa around the knee (Baker's cysts) can, when ruptured, cause mass effect and simulate the pain and swelling associated with popliteal DVT (Fig. 19). Popliteal artery aneurysm may cause calf pain and swelling (Fig. 20). Congestive heart failure or tricuspid insufficiency with secondary venous distension and leg swelling and edema may cause a clinically false-positive result. Often in these patients a very pulsatile venous waveform is observed (see Fig. 10) [20]. A patent SSV filled with thrombus may be mistaken for a thrombosed PV (Fig. 21). Acute or chronic clot may be difficult to differentiate, as discussed later.

False-negative US studies can occur in obese patients or in patients who have very edematous extremities. The FV in the adductor canal region may be difficult to evaluate. Small nonocclusive thrombi in the profunda femoris vein may be missed. Thrombosis of a duplicated FV (see Fig. 4B) may go undetected, and more proximal iliac vein thrombosis often is difficult to demonstrate. Loss of phasicity in the CFV or distal iliac vein should be noted as a secondary sign indicating a more proximal thrombosis or compression by masses or collections (see Figs. 16 and 18).

It is important to recognize these pitfalls and limitations to lessen the chance of potentially severe consequences of an erroneous or missed diagnosis of DVT. False-negative imaging results may deprive the patient of appropriate anticoagulation therapy and potentially result in clot propagation, PE, and death, whereas a false-positive result may result in unnecessary anticoagulation with the associated risk of bleeding.

Sonography of chronic deep venous thrombosis

The differentiation of acute or chronic DVT by imaging methods and clinical parameters often is difficult. In about one fourth of patients, the DVT resolves completely after adequate treatment regimens. Serial studies, however, have shown

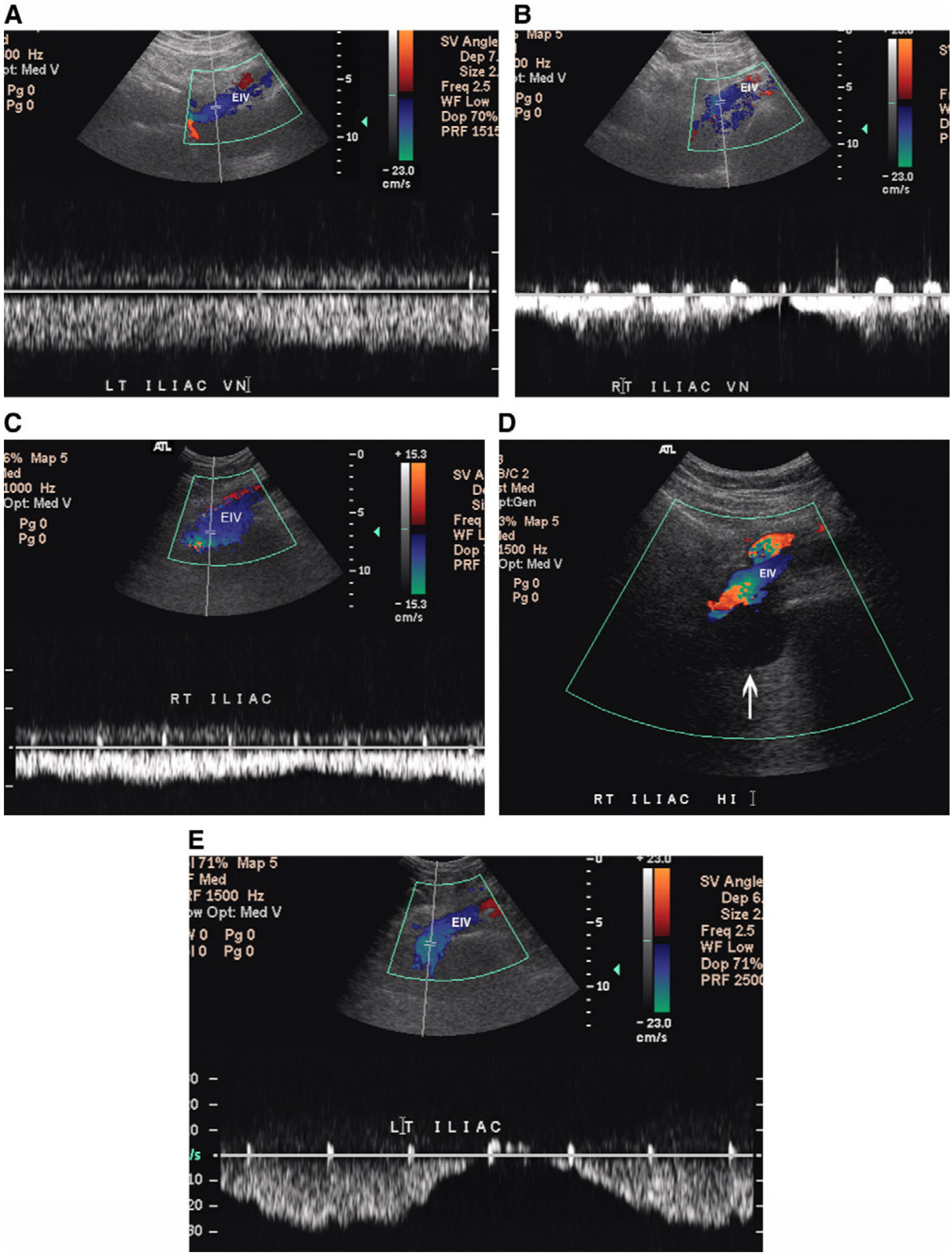

Fig. 9. (*A*) Dampened flow in left external iliac vein (*EIV*) caused by more proximal thrombosis. (*B*) Normal respiratory phasicity flow in right external iliac vein (*EIV*). (*C*) Dampened flow in right external iliac vein (*EIV*) caused by compression by fluid collection with loss of respiratory phasicity. (*D*) Right groin fluid collection (*arrow*). (*E*) Normal respiratory phasicity in left external iliac vein (*EIV*).

persistent abnormal findings on compression US 6 to 24 months later in 54% of patients who had acute DVT [4]. Without an US evaluation showing resolution of the acute DVT, recurrent DVT may be indistinguishable from chronic disease. On compression US both acute and chronic DVT may show noncompressibility of the vessel. In chronic DVT, the vessel walls often are thickened with decreased compliance (Fig. 22A). Veins may recanalize to a certain extent after an acute DVT (Fig. 22B). Residual clot, however, may manifest as organized echogenic material along the vein walls and mimic the sonographic appearance of acute clot. Because noncompressibility of a vein is a sonographic sign of

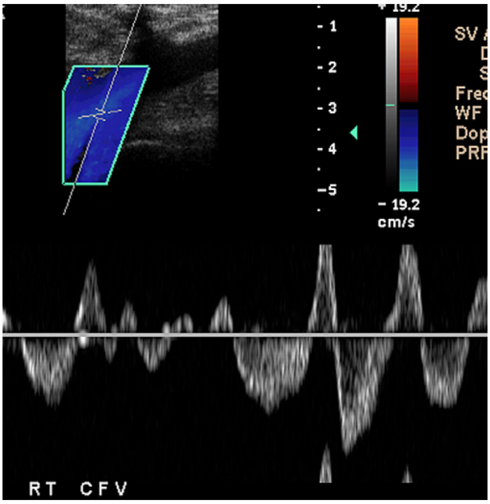

Fig. 10. Pulsatile spectral waveform in right common femoral vein (*CFV*) caused by tricuspid insufficiency.

both chronic and acute DVT, care must be taken to differentiate the two entities so that patients do not receive unnecessary anticoagulation therapy for a chronic condition. Some US findings that may help distinguish acute from chronic DVT are atretic segments (inability to identify a normal vein next to the artery), echogenic weblike filling defects within the vein (see Fig. 22B), collateral vessels (Fig. 23B), and valvular damage with reflux and resultant chronic deep venous insufficiency. Chronic thrombus usually is more echogenic and may calcify (Fig. 23A). Chronic thrombi may demonstrate thickened vein walls with a reduced venous diameter (see Fig. 22A). In addition, the size of the vein is helpful. In acute DVT the vein usually is enlarged, whereas in chronic DVT it is normal, small, or diminutive. Recent studies demonstrating the contribution of in vitro US elasticity imaging to assess clot age may be promising for clinical use in the future [39].

Chronic venous insufficiency

Venous insufficiency is a common but often undiagnosed condition with a prevalence estimated at 25% in women and 15% in men [40–43]. The clinical significance of varicose veins is not merely cosmetic, because chronic venous insufficiency may cause significant pain and swelling of the lower extremities and ultimately may result in debilitating skin changes ranging from discoloration and induration to cutaneous and soft tissue breakdown (ie, venous stasis ulcers). Treatment costs are estimated to be as high as 3 billion dollars per year in the United States [44] and to comprise up to 1% to 3% of the total health care budget in developed countries [45–47]. Recently developed minimally invasive catheter techniques have revolutionized treatment options for patients who have varicose veins. Hence, it is important that radiologists become familiar with the US technique for evaluation of venous reflux.

Early on, patients who have venous reflux may present with leg pain and swelling and may not have obvious visible varicosities. Such patients often are referred for lower extremity venous US examinations to rule out DVT, and both clinician and radiologist may overlook the diagnosis of venous insufficiency. Hence, venous insufficiency is

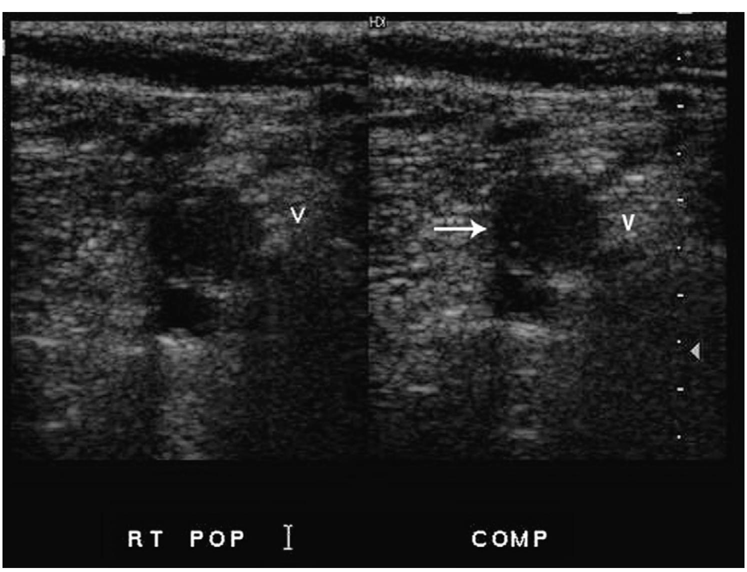

Fig. 11. Complete venous thrombosis. Transverse views of the right popliteal vein demonstrate noncompressible vein caused by acute thrombus (*arrow*).

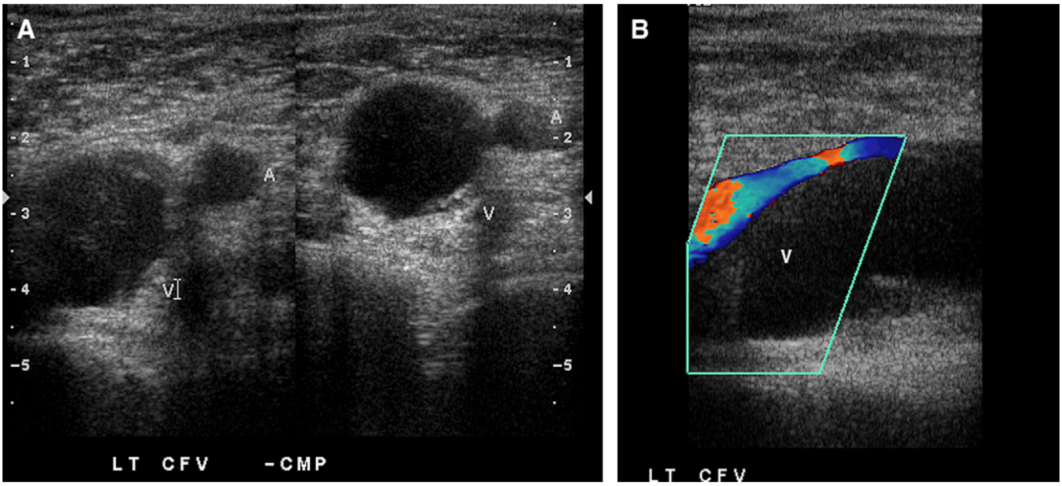

Fig. 12. (*A*) Transverse and (*B*) sagittal views of the left common femoral vein (*V*) demonstrate hypoechoic thrombus and expansion of the venous lumen.

a diagnosis that should be considered in the patient presenting with leg pain or swelling and a negative DVT US examination, particularly in someone who has persistent or recurrent symptoms. Patients who have venous reflux also may complain of leg soreness, burning, aching, throbbing, cramping, and/or muscle fatigue. Symptoms are typically exacerbated by standing or warm weather. The severity of a patient's symptoms is not necessarily related to the size or number of the varicosities or even to the amount of reflux, and significant symptoms may occur even before varicosities are clinically apparent. Over time, patients may develop chronic skin or soft tissue changes ranging from swelling and discoloration to inflammatory dermatitis and, ultimately, chronic, nonhealing leg ulcers.

Pathophysiology

Normally, venous blood flow is directed from the superficial venous system of the lower extremities through the GSV, SSV, and perforators toward the deep venous system and the heart by a series of competent, one-way valves as well as by contraction of the foot and calf muscles [48]. Valve failure is the primary cause of venous insufficiency. An incompetent valve results in retrograde flow of blood and venous pooling, which in turn dilates the more caudal veins, resulting in a cascading effect of sequential failure of more and more valves. The precise pattern of varicosities that develop depends on exactly which valves fail. Because the tributaries of the GSV and SSV are not protected by a fascial layer, they are more prone to dilate. Primary pump failure

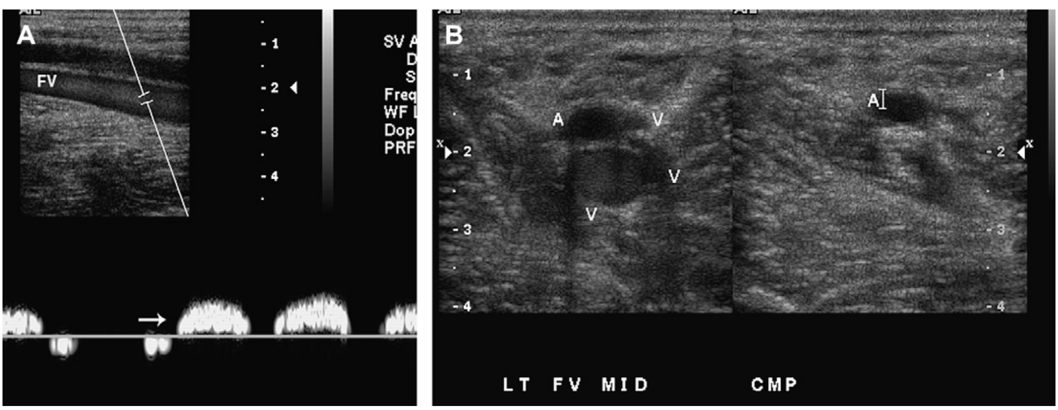

Fig. 13. Slow-flowing blood mimicking acute thrombosis. (*A*) Sagittal view of the left femoral vein demonstrates echogenic material in the venous lumen. Note venous waveform on duplex spectrum, however. (*B*) With transverse compression the venous lumen (*V*) collapses completely.

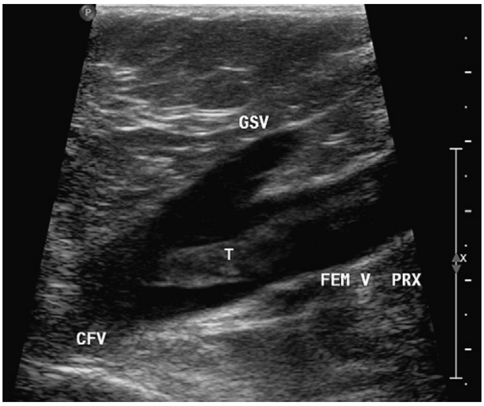

Fig. 14. Free-floating thrombus (*T*) in the left femoral vein (*FV*) extending into the common femoral vein (*CFV*).

of the calf muscles, venous obstruction from DVT, and extrinsic venous compression are much less common causes of venous insufficiency.

Risk factors for the development of venous insufficiency include any condition that distorts and/or weakens the venous valves or results in venous dilation or volume overload or increases hydrostatic pressure. Hence, risk factors include genetic predisposition, prior DVT, pregnancy, age, obesity, and occupations or activities that require prolonged standing, running, or lifting [49]. The incidence of varicose veins is nearly twice as high in women as in men [50], probably because of multiple factors including cyclic hormonal changes. Venous insufficiency also is more common in industrialized Western countries; the increased incidence is postulated to be largely secondary to either genetic predisposition and/or diet [51].

Venous anatomy of the superficial veins of the lower extremity

The anatomy of the deep venous system in the lower extremity has been described earlier in this article. One should note here, however, several features that help discriminate the deep venous system from the superficial venous system. The veins of the deep venous system in the thigh and calf are surrounded by muscles and a deep fascial layer that help propel blood back toward the heart by means of the primary muscle pump and also help prevent reflux by limiting dilatation of the deep veins. The deep veins also always accompany an artery. The superficial veins, on the other hand, are found in the subcutaneous tissues and do not accompany an artery. A thin superficial fascial layer surrounds the saphenous trunks but not their tributaries. Thus, major branches of the saphenous system are more prone to dilate and develop reflux.

There are three major subdivisions of the superficial venous system in the lower extremity: the GSV, the SSV, and the lateral subdermic venous systems. There is substantial variability in the anatomy of the superficial venous system and in the distribution of the communicating veins (perforators) between the deep and superficial venous system.

As mentioned previously, the GSV originates medially on the foot, courses anteromedially around the medial malleolus at the ankle, and ascends anteromedially up the calf and thigh in a relatively

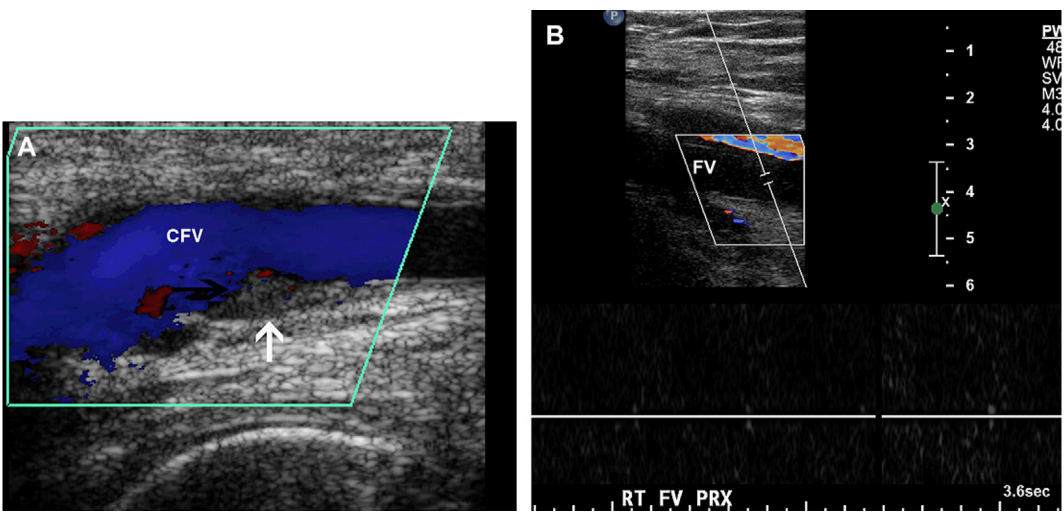

Fig. 15. (*A*) Color Doppler filling defect in the common femoral vein (*CFV*) caused by mural thrombus (*arrow*). (*B*) Absence of color flow and spectral waveform in completely thrombosed right femoral vein. Note color flow in adjacent femoral artery.

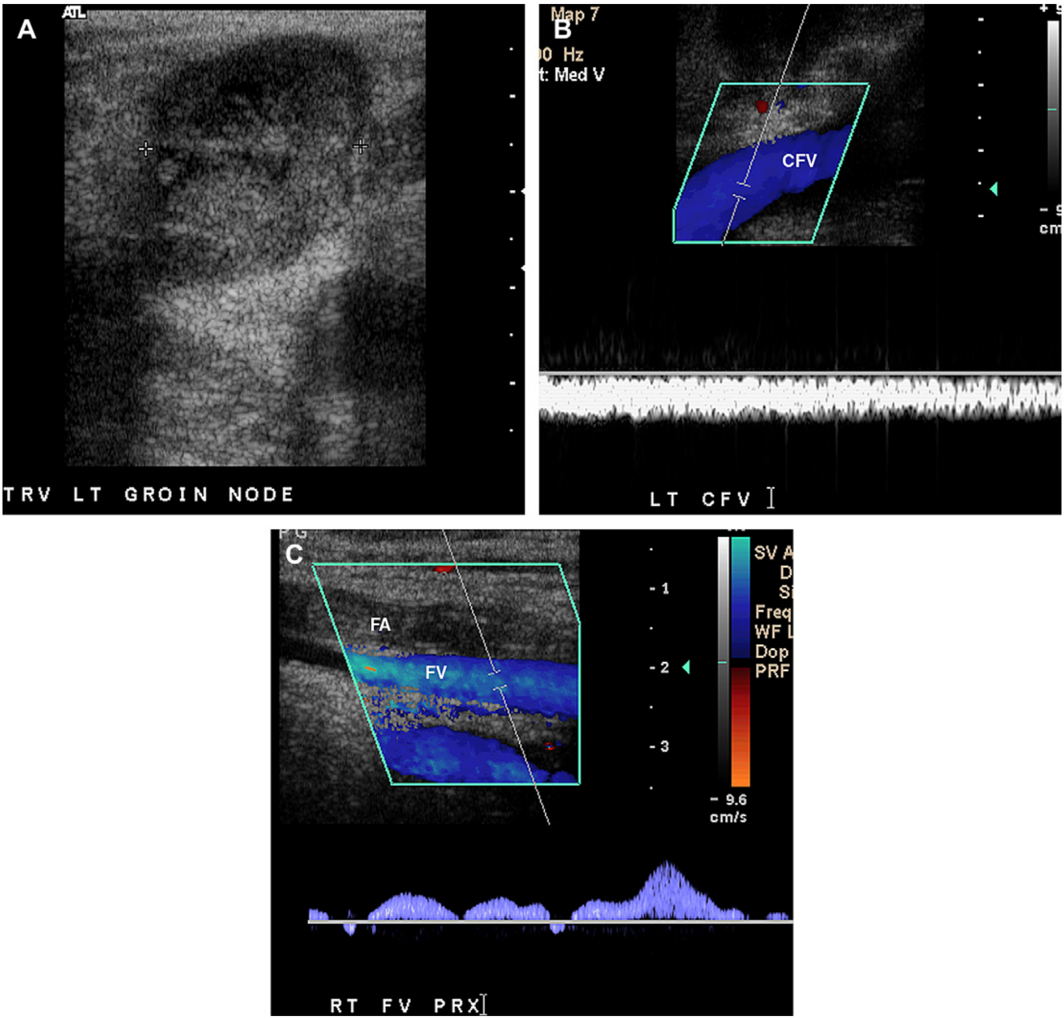

Fig. 16. False-positive clinical findings mimicking DVT. (*A*) Groin adenopathy (*between calipers*) causing leg swelling and pain. (*B*) Dampening of flow in adjacent patent left common femoral vein (*CFV*). (*C*) Right femoral artery thrombosis (*FA*) causing pain. Note normal flow in adjacent femoral vein (*FV*).

straight line, joining the CFV medially near the groin (see Fig. 1A). There always is a terminal valve at the saphenofemoral junction and usually one or two subterminal valves within 1 to 2 cm of the terminal valve. The GSV is typically 3 to 4 mm in diameter in the upper thigh and is contained in an oval fascial envelope resulting in a characteristic appearance in cross-section that has been compared with an "Egyptian eye" (Fig. 24) [52]. The most important branches of the GSV in the thigh are the posteromedial and anteromedial thigh veins, which arise in the upper to mid thigh. The lateral and medial cutaneous femoral branches, the external circumflex iliac vein, the superficial epigastric vein, and the external pudendal vein are all found near the saphenofemoral junction and terminal valves; the later three branches course superiorly (see Figs. 1A and 24). These branches, although they may be large, are contained by the oval echogenic fascial envelope. It is estimated that 60% of patients who have varicosities have reflux in the GSV system.

The SSV originates laterally on the dorsum of the foot, passes posterior to the lateral malleolus at the ankle, and ascends the calf posteriorly, like the seam in an old-fashioned stocking (see Fig. 1B). The exact point of termination of the SSV into the deep system is quite variable. In most people, the SSV terminates directly into the PV near the popliteal crease, but the SSV may join the GSV in the lower thigh through the vein of Giacomini or even may empty directly into the FV in the thigh. The SSV also is surrounded by a fascial layer in the upper calf and typically is not wider than 3 mm in diameter. The SSV runs close to the tibial and sural

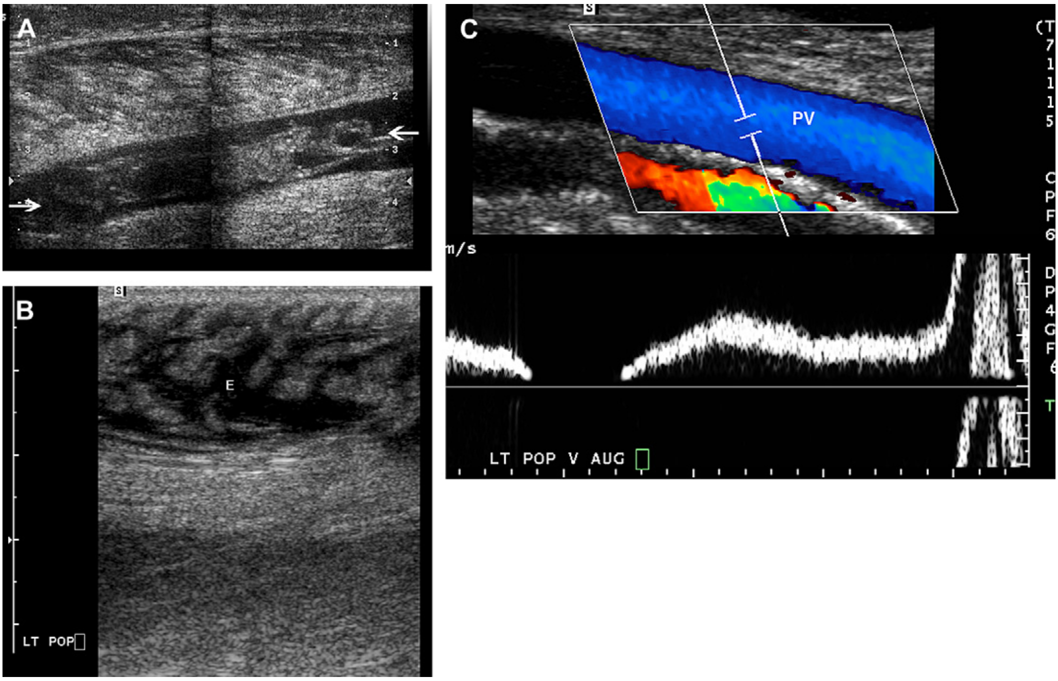

Fig. 17. False-positive clinical findings mimicking DVT. (*A*) Large calf hematoma (*between arrows*) secondary to a muscular tear. (*B*) Diffuse soft tissue edema (*E*) of the lower extremity. (*C*) The underlying popliteal vein (*PV*) was patent.

nerves, which may explain the pain associated with varicosities arising from the SSV.

The lateral subdermic venous system consists of a group of small veins found laterally above and below the knee. These veins have variable communications with the deep system. Reflux in the lateral subdermic venous system typically results in a fine telangiectasic web of superficial varicosities posterolaterally above or below the knee. Such spider-vein varicosities often occur in younger women (probably resulting from a genetic predisposition) and typically are not associated with truncal varicosities or significant pain and swelling.

Numerous short, perforating veins direct flow from the superficial to the deep system. The exact number and location of these perforators is quite variable. In general these veins are short, straight, and run perpendicular or at a slight angle from the GSV or SSV toward the deep venous system. The most common named groups are the Hunterian perforators (near the medial aspect of the upper adductor canal); Dodd's perforators (in the lower

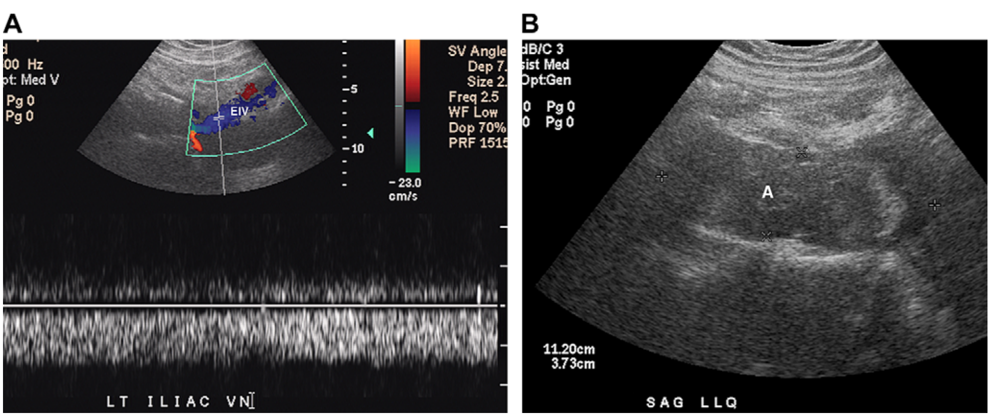

Fig. 18. Clinically false-positive results for DVT caused by groin adenopathy. (*A*) Dampened flow in the left external iliac vein (*EIV*) caused by (*B*) large groin adenopathy (*A*) from prostate cancer (between calipers).

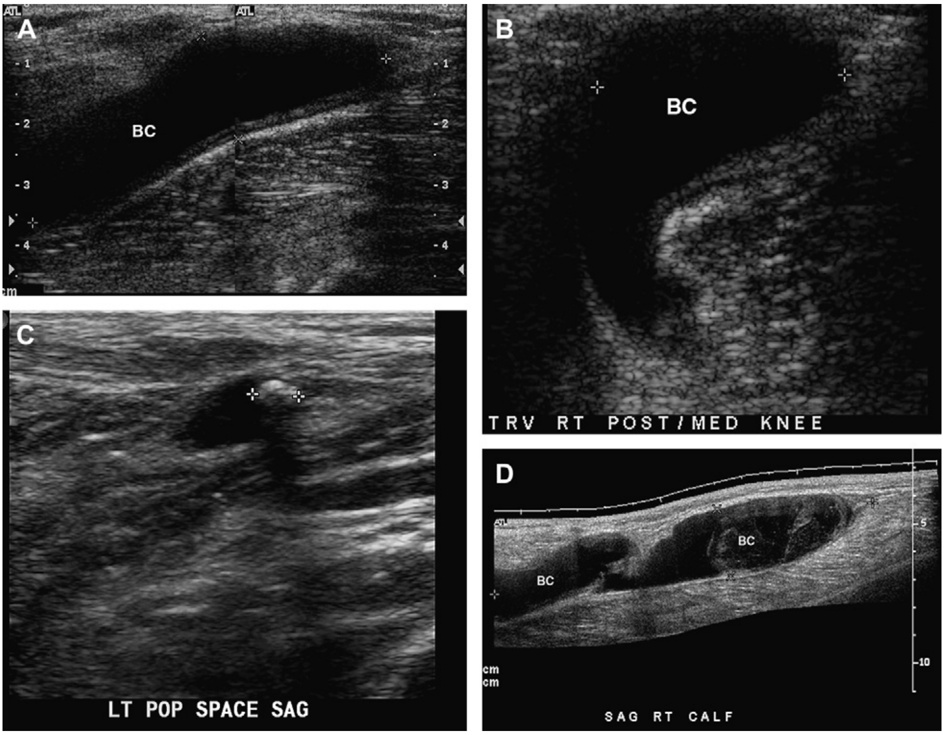

Fig. 19. Baker's cysts. (*A*) Sagittal and (*B*) transverse images of a simple Baker's cyst (*BC*). (*C*) Sagittal view of a complex Baker's cyst (*BC*) with calcifications (between calipers). (*D*) Sagittal view of a ruptured Baker's cyst (*BC*) extending into the lower calf (between calipers).

medial thigh); Boyd's perforators (in the mid calf); and Crockett's perforators (in the lower posteromedial calf) (Fig. 25). When incompetent, these perforators dilate focally and may cause "blow outs," point tenderness, or even ulceration where they join the superficial system.

Method of examination

Designing a treatment plan for a patient who has varicose veins requires identification of the highest as well as the lowest point of reflux. In addition, the source of reflux must be identified for every

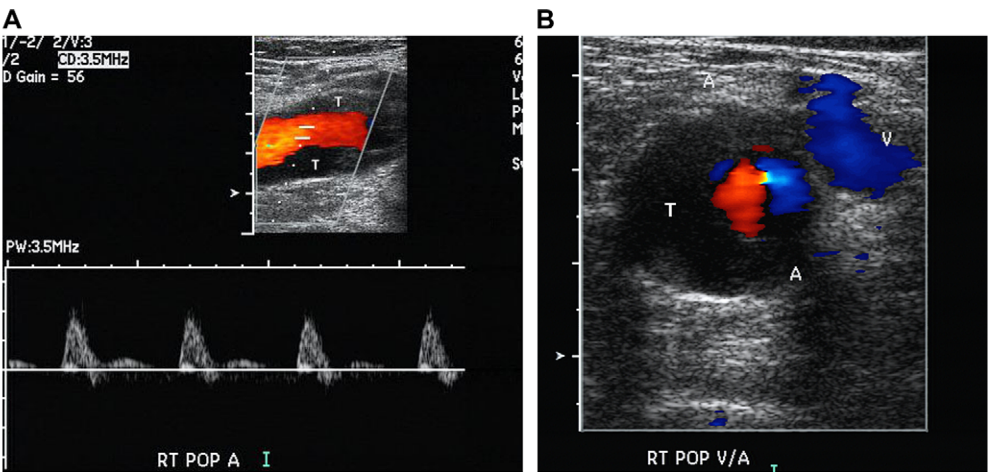

Fig. 20. Popliteal artery aneurysm. (*A*) Sagittal and (*B*) transverse color Doppler US of a partially thrombosed popliteal artery aneurysm (*A*) causing pain and calf swelling. Note absence of flow in the thrombosed portion (*T*).

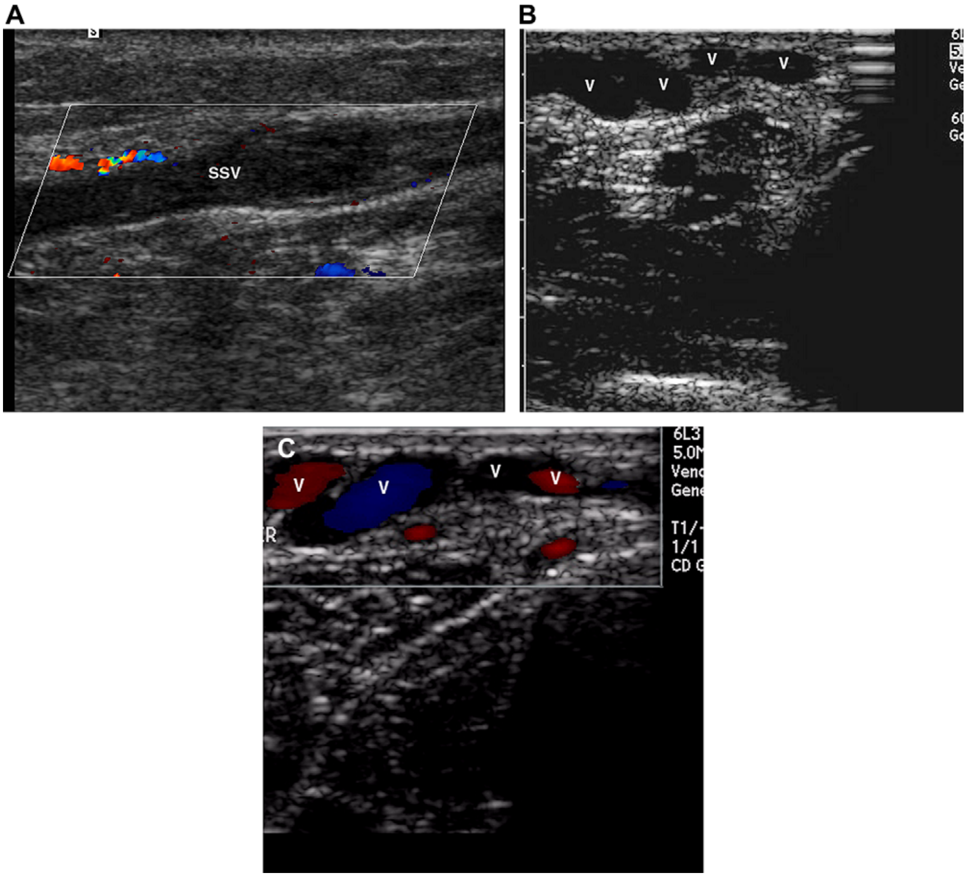

Fig. 21. (*A*) Sagittal view of a thrombosed small saphenous vein (*SSV*) mistaken for popliteal vein thrombosis. Note the very superficial location of the small saphenous vein and absence of an accompany artery. (*B*) Gray-scale image and (*C*) color Doppler US of superficial varicosities (*V*) causing pain and swelling, thus clinically mimicking DVT.

varicosity. This identification is accomplished by a combination of visual inspection and Doppler US. US examination can be quite lengthy in a patient who has numerous varicosities because patterns of reflux are highly variable and often are quite complex, with multiple sources of reflux.

The Doppler US examination for reflux begins with a standard US examination of the deep system performed with the patient in the supine position to exclude DVT. The patient then stands, and the legs are inspected visually to identify all varicose veins, because the examination should be tailored to address all varicosities in question. In general, reflux in the GSV tends to involve the medial (inner) thigh, whereas reflux in the SSV causes varicosities in the posterior calf. Telangiectastic varices developing laterally around the knee typically are secondary to reflux in the lateral subdermic venous system. Because not all symptomatic varicosities are visible, the examiner should remember to ask the patient where it hurts.

Doppler assessment of reflux always should be performed with the patient in the standing position. Because the examination can be time consuming, it may be helpful to have a walker, table, or counter to help support the patient as well as a stool for the examiner. Either color or pulse Doppler can be used, and three different maneuvers can be attempted to elicit reflux:

1. Augmentation: The leg is squeezed from below, directing flow toward the heart. If blood flows back toward the feet for more than 0.5 seconds following augmentation, the examination is positive for reflux (Fig. 26), although some examiners require a full second of reflux before considering the examination to be positive. Transient retrograde flow is normal and serves to push back the valve leaflets, causing them to close.
2. Valsalva maneuver: Reflux can be assessed in the thigh after the Valsalva maneuver because the resultant increased intra-abdominal pressure causes retrograde flow through incompetent

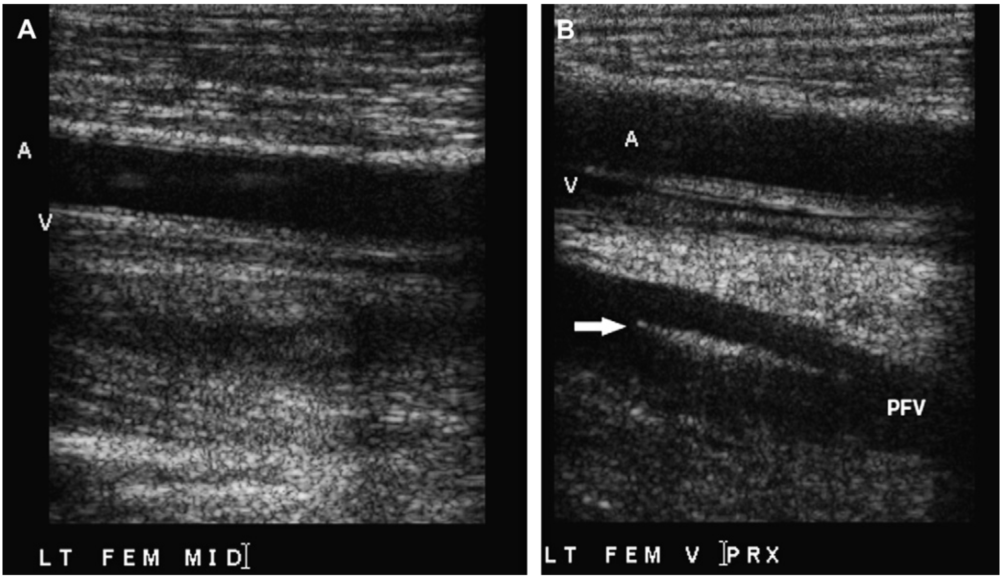

Fig. 22. DVT. (*A*) Thickened irregular vessel walls in a patient who has chronic DVT (*V*). (*B*) Linear fibrin strand in left profunda femoris vein (*PFV*), a sequela from prior DVT (*arrow*).

valves (Fig. 27). Although this technique is sensitive, it works well only in the upper thigh. If the terminal and/or subterminal valves in the GSV are competent, the Valsalva maneuver will not detect more distal points of reflux.

3. Direct or retrograde compression: Direct compression above the point of Doppler interrogation propels blood toward the feet in the setting of an incompetent valve. Although it is easiest to assess for reflux in the longitudinal plane using color or pulse Doppler interrogation, with experience the examination can be performed more efficiently in the transverse plane with color Doppler while angling the transducer toward the head or feet (Fig. 28).

In addition to Doppler interrogation, gray-scale imaging can be helpful. Often the valves themselves can be visualized directly in the GSV. If the valve leaflets are scarred, thickened, or retracted and do not meet in the midline, reflux is likely. Similarly, if the GSV measures more than 6 mm in diameter, reflux is also likely.

The standard or minimum US examination should assess for reflux in the deep venous system at the levels of the CFV (above and below the saphenofemoral junction), FV (upper, mid, and low),

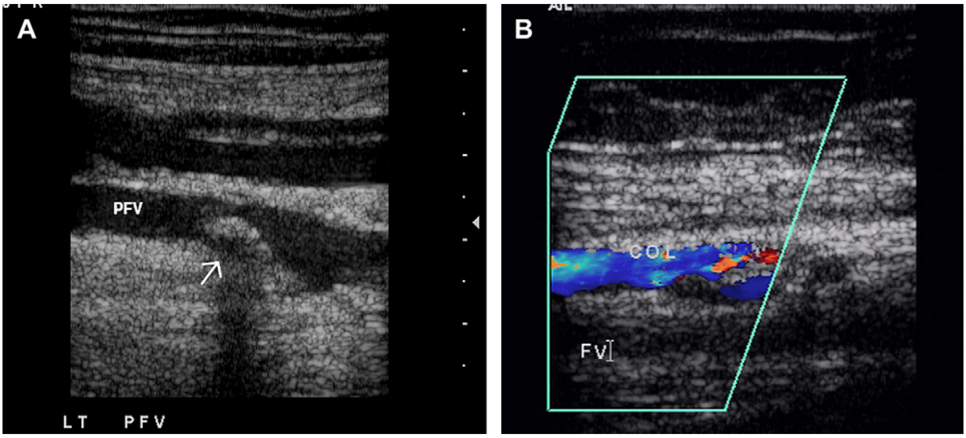

Fig. 23. Chronic DVT. (*A*) Echogenic, calcified thrombus in the left profunda femoris vein (*PFV*) (*arrow*). (*B*) Collateral vessels (*COL*) in a patient who has chronic thrombosis of the right femoral vein (*FV*).

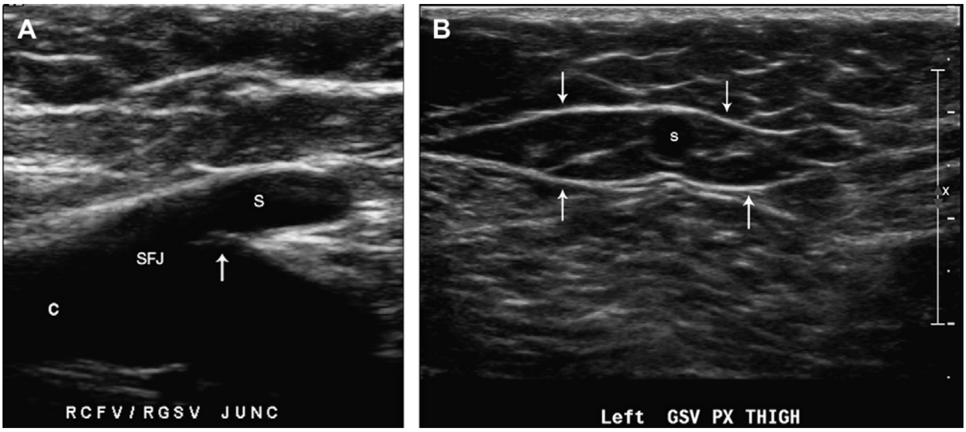

Fig. 24. Normal greater saphenous vein. (*A*) Longitudinal image at the saphenofemoral junction (*SFJ*). Note the junction of the more superficial and smaller greater saphenous vein (*S*) with the deeper and larger common femoral vein (*C*). The greater saphenous vein runs medial to the femoral vessels and joins the common femoral vein in the groin. Arrow indicates terminal valve. (*B*) Transverse image of the greater saphenous vein (*S*) within its surrounding echogenic elliptical fascial sheath creating an appearance similar to a stylized "Egyptian eye" (*arrows*). Tributaries of the greater saphenous vein pierce this fascial envelope and run more superficially in the subdermal fat and connective tissue.

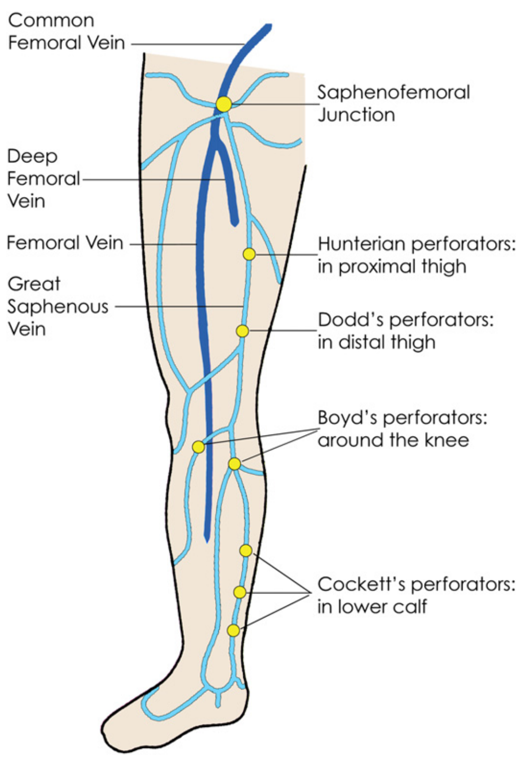

Fig. 25. Major named perforators between the superficial and deep venous system in the lower extremity. (*Adapted from* Weiss RA, Feied CF, Weiss MA. Vein diagnosis and treatment. New York: McGraw-Hill; 2001.)

and PV as well as in the GSV, from the saphenofemoral junction to the knee including the origin of the major tributaries and the PV-SSV junction. Full examination of the SSV and GSV below the knee need not be performed unless varicosities are noted in the calf. All varicosities should be traced back to the origin of reflux. In this way, reflux in major branches of the GSV or SSV or reflux originating

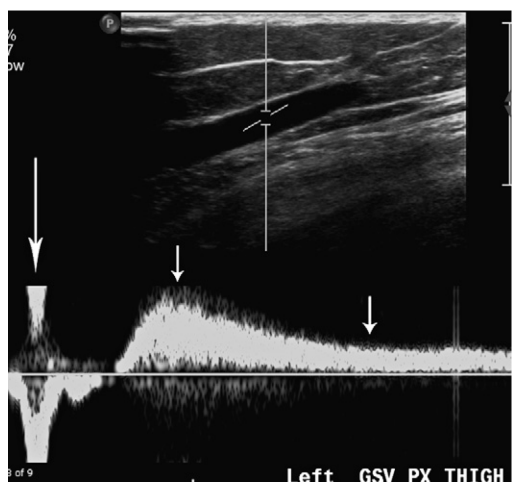

Fig. 26. Reflux in the greater saphenous vein following augmentation. Note sharp, short increase in volume and velocity of blood flow directed toward the head when the leg is squeezed from below (*long arrow*). Subsequently, there is prolonged reversed flow of blood toward the feet lasting well over several seconds (*short arrows*).

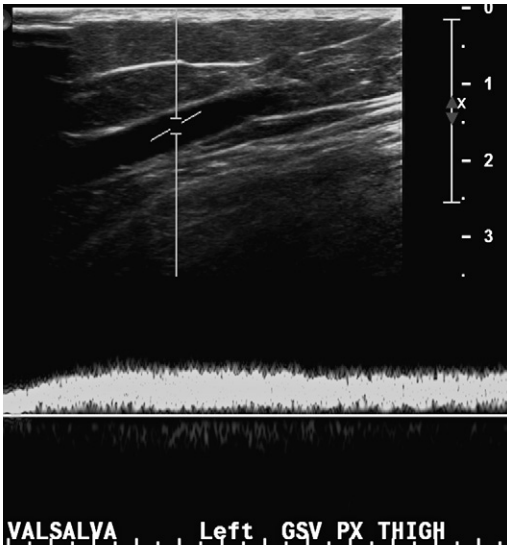

Fig. 27. Reflux in the greater saphenous vein following a Valsalva maneuver. Prolonged reflux (ie, reversed flow in the greater saphenous vein), is noted when the patient increases intra-abdominal pressure by performing a Valsalva maneuver.

from incompetent perforators may be identified even in the absence of truncal reflux in the GSV and SSV.

Summary

DVT of the lower extremities is a common clinical entity and, if left untreated, may result in chronic disability producing the postphlebitic syndrome or potentially life-threatening and devastating outcomes such as PE. Compression US now is recognized as the most appropriate primary initial imaging modality for evaluating patients at risk for a peripheral DVT, and its accuracy in diagnosing acute or chronic DVT is well established in patients who have lower extremity symptoms. The CFV, FV, and PV are examined routinely. The need for evaluation of the calf veins remains controversial, however. US also is helpful in evaluating patients who have chronic venous changes such as the postphlebitic syndrome and venous insufficiency.

The advantages of US include the noninvasiveness of the examination, lack of ionizing radiation, lack of need for intravenous nephrotoxic contrast media, relatively low cost, and portability so that even the most critically ill patients in the ICU can be

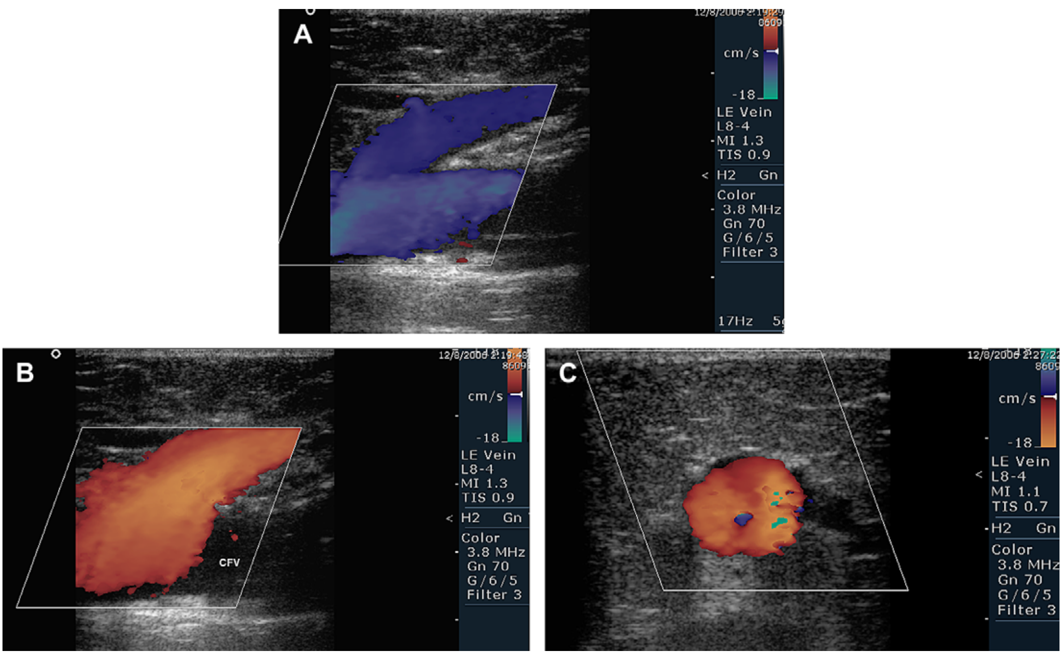

Fig. 28. (*A*) Color baseline examination reveals blood in the common femoral vein and greater saphenous vein heading away from the transducer toward the head (and therefore is color-coded blue). (*B*) Color reflux following augmentation (or the Valsalva maneuver): blood flow in the greater saphenous vein is toward the feet and away from the transducer (and therefore is color-coded red). Note no blood flow is noted in the deeper common femoral vein (*CFV*) because competent valves in the common femoral vein prevent reflux of blood toward the feet. (*C*) Imaging in the transverse plane with the transducer angled toward the feet demonstrates blood flowing away from the transducer (ie, refluxing toward the feet and therefore color-coded red).

examined. A thorough knowledge of regional vascular anatomy, equipment sensitivity, and scanning techniques is required. Despite its limitations in less well-visualized areas such as the iliac and calf veins, US is the most appropriate initial study to perform in patients suspected of harboring a lower extremity DVT. If US studies are nondiagnostic or equivocal, further evaluation with multidetector CT or MR venography may occasionally be necessary.

The signs and symptoms of venous insufficiency overlap with those of DVT. Hence, in a patient presenting with leg pain and swelling, the possibility of venous insufficiency may be overlooked by both clinician and the radiologist because of the overriding and more immediate concern of excluding DVT, a more serious and potentially life-threatening problem. Thus, in the acute setting, Doppler US examination of the lower extremities typically is performed solely to assess for DVT. The clinician and radiologist, however, should remember that venous insufficiency is a common cause of false-negative US examination of the lower extremities and should consider this diagnosis in patients who have leg pain and/or swelling and a negative Doppler US, particularly if symptoms are recurrent or if varicosities are visible. Consideration then should be given to referral for a more extensive Doppler evaluation of the lower extremities to evaluate for reflux.

Although more than 60% of varicosities are caused by reflux in the GSV and its major branches, other tributaries and perforators should be examined based on the patient's symptoms, visual inspection, and physical examination (focal tenderness, bulging, ulcers). If the point of origin of reflux cannot be identified by following the GSV or SSV distally, the examiner should begin at the varicosity and follow it proximally, being careful to identify the highest point of reflux. The examiner should remember that there can be complex patterns of reflux and multiple sites of origin involving tributaries and perforators with or without truncal reflux. It is important to make the diagnosis of chronic venous insufficiency, both to diagnose the cause of the patient's symptoms and because new, minimally invasive percutaneous techniques such as ambulatory phlebectomy, sclerotherapy, and endovascular thermal ablation have revolutionized treatment of this common, debilitating problem.

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