UPPER EXTREMITY VENOUS EVALUATION

INTRODUCTION

The presence of upper extremity deep vein thrombosis was, for many years, neglected. It was thought that upper extremity deep vein thrombosis was more of a nuisance than a true medical entity. This perception has dramatically changed. It is now recognized that upper extremity deep vein thrombosis can account for at least 10% and as high as 30% of incident cases of pulmonary embolism. In addition, the increased used of chronic venous access catheters such as PICS (Peripherally Inserted Central venous catheters), and various tunneled catheters has been associated with an increased incidence of upper extremity deep vein thrombosis. This is compounded by the fact that many of these patients have a malignancy, a well recognized and major risk factor for the development of either lower or upper extremity thrombophlebitis.

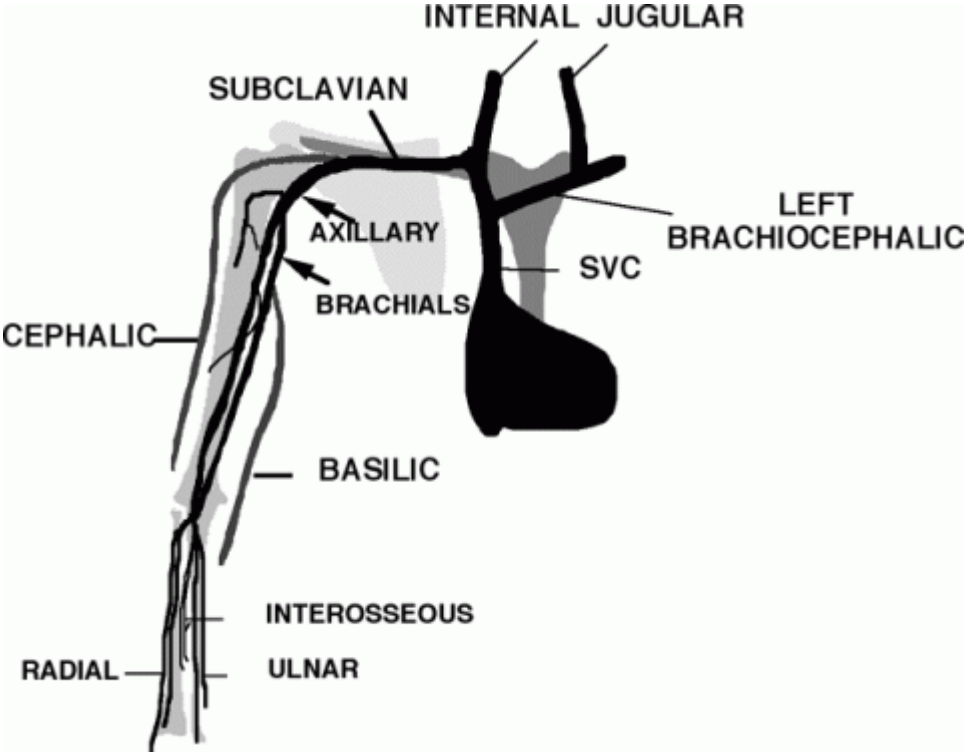

Figure 1: The deep system of the upper extremity veins consist of paired veins below the elbow (ulnar, radial and interosseous). These form the paired brachial veins that then join the basilic vein to form the axillary vein. The axillary vein continues as the subclavian. The subclavian vein then joins the internal jugular vein to form the brachiocephalic veins. From the left, the brachiocephalic must cross the midline so it can join the right brachiocephalic vein and form the superior vena cava.
The cephalic and basilic veins start from branches in the forearm and course one laterally (cephalic) and the other medially (basilic) to join the axillary vein at different levels.

There is greater variability in the venous anatomy of the upper extremity than there is in the lower

extremity. The number of venous branches and their respective lengths show great inter-individual differences in the arm.

As in the lower extremity, each deep vein accompanies an artery. The ulnar, radial and interosseous branches of the forearm are duplicated. They are rarely the site of spontaneous deep vein thrombosis. The brachial veins are also duplicated and rarely the primary site of thrombosis. Typically, idiopathic or spontaneous thrombosis occurs in the more proximal deep veins such as the axillary and subclavian veins. The internal jugular vein is a deep vein and is commonly the site of thrombosis. In addition to cases of spontaneous thrombosis, deep vein thrombosis is often associated with the insertion of pacemakers or chronic indwelling lines in the subclavian and internal jugular veins.

THE SUPERFICIAL VEINS

The two main superficial veins are the cephalic and basilic veins. They are the preferred access sites for the insertion of PICC (peripherally inserted central venous catheter) lines and the most common cause of thrombus formation. The likelihood of thrombosis is also closely related to instrumentation with a needle during blood draws. The thrombi caused either by blood draws or associated with PICC lines are likely to spread to the deep venous system.

The median basilic vein is the most commonly accessed site for blood draws. Spread of an early thrombus is likely to occur into the basilic vein and then continue into the axillary vein in the upper arm (figure 2). PICC placement also increases the likelihood of thrombosis. The most common site of placement is the cephalic vein (Figure 3). The forming thrombus can then involve the axillary vein since the cephalic vein joins the axillary vein at the level of the upper chest.

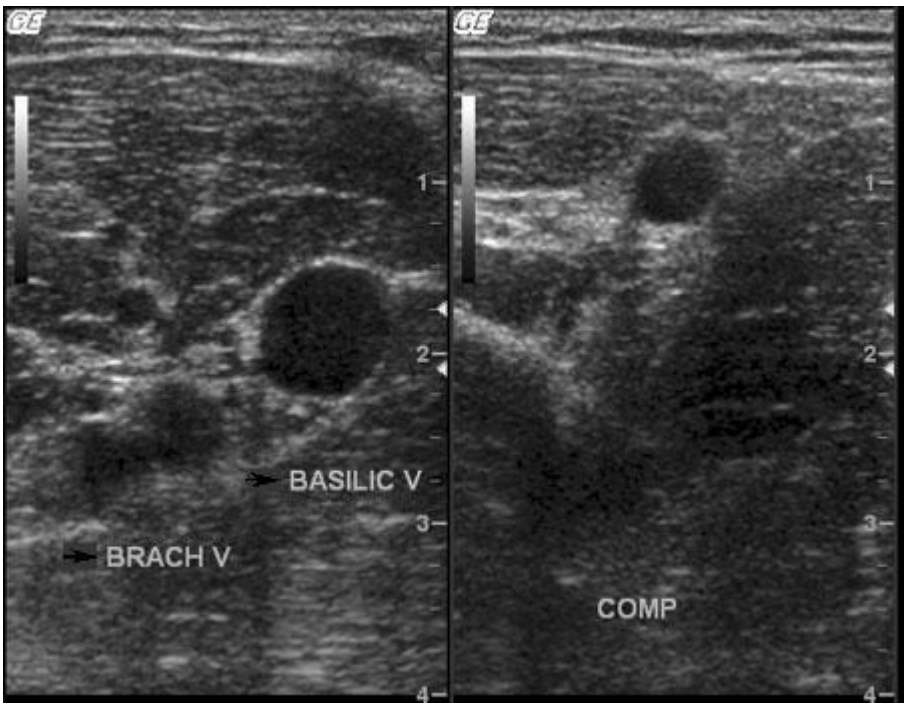

Figure 2: The basilic vein courses parallel with the duplicated brachial veins in the lower arm, just above the elbow. On this transverse image, the basilic vein is shown to be distinct from the brachial veins, and more importantly, distant from the brachial artery. The basilic vein continues and joins into the axillary vein. Typically, one or two of the brachial veins will also join with the basilic vein as it forms the axillary vein.

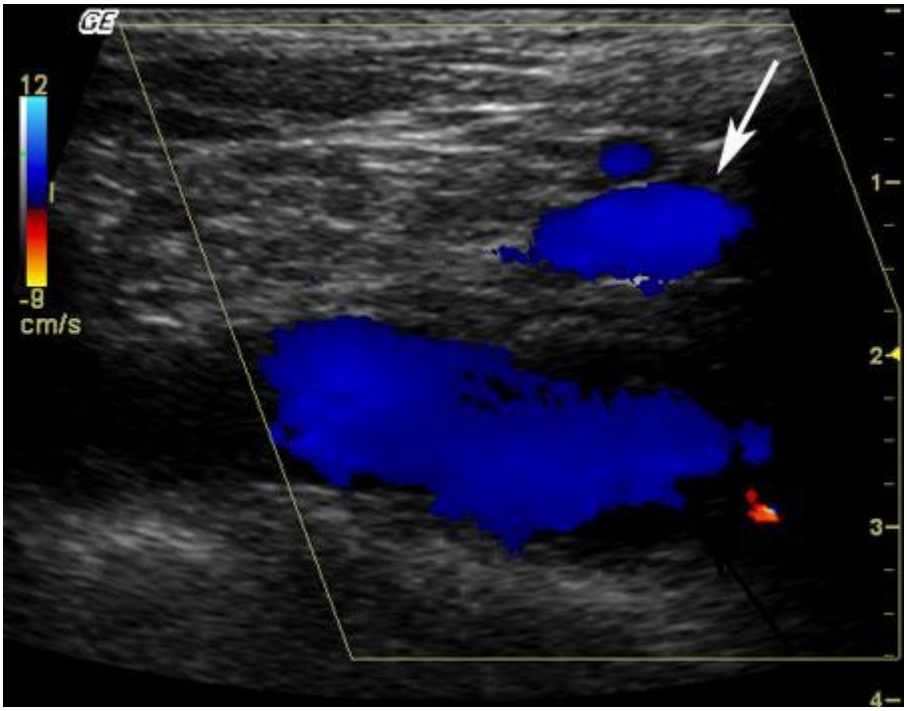

Figure 3: The cephalic vein is the other dominant superficial vein of the arm. On this image, the cephalic vein, on top (arrow), is nearing the axilllary vein and is about to join into it. The cephalic vein is a common site of superficial vein thrombosis, mostly due to the increased use of PICCs and their placement in this vein.

COMMUNICATING SYSTEM

There are no easily identifiable perforating veins routinely identified in the arm. Collaterals transiting through the fascia exist and become potential collateral pathways when a deep segment is occluded. With the exception of this, the major sites of communication between deep and superficial veins are the continuation of the basilic vein into the axillary vein and the cephalic into the more proximal axillary vein.

PATHOPHYSIOLOGY

The estimated 1 to 2% incidence of pulmonary embolism arising from upper extremity DVT has historically been underestimated. More recent estimates place the risk at 5 to 10%.

The site of a deep vein thrombus in the upper extremity that is considered to be a risk for pulmonary embolism is poorly defined. Empirically, it is believed that the more central thrombi, located in the subclavian and brachiocephalic veins, are the greatest threat for emboli to the lung.

The major risk factors associated with the development of upper extremity thrombophlebitis are the same as those for the lower extremity veins. These risk factors were originally described by Virchow more than two centuries ago: stasis, trauma to the endothelial lining of the vein wall and altered coagulation factors. The interaction of these three major risk factors explains in part the presentation of a patient with upper extremity thrombosis.

Idiopathic upper extremity deep vein thrombosis is believed to occur in the absence of any obvious risk factor. This is somewhat of a simplification. Even idiopathic thrombus can be associated with extrinsic compression of the vein, often at the crossing of the first rib and the clavicle (subclavian vein) and by the pectoralis muscle (axillary vein). This promotes the development of stasis and in some cases causes micro-traumas to the endothelial lining of the vein wall. Even if the term idiopathic is used, extrinsic compression of the vein likely plays is a significant role in the development of upper extremity deep vein thrombosis.

The risk of upper extremity thrombosis is linked to the existence of clear- cut anatomic factors. In these patients, extrinsic compression of the veins can be shown when different arm positions are adopted. The vein is compressed and the lining endothelium can be damaged at these anatomic "choke" points. This occurs typically for the subclavian vein at the crossing of the first rib with the clavicle and in the chest wall, at the crossing with the pectoralis muscle over the axillary vein (figure 4). Such patients have what is often called the Paget-Schroetter syndrome. More importantly, if aggressive therapy with thrombolytics is performed, the existence and location of the "choke" point dictates the need for further therapy. A patient with extrinsic compression of the subclavian vein between the first rib and the clavicle often need a surgical intervention such as resection of a portion of the first rib in order to decompress the vein.

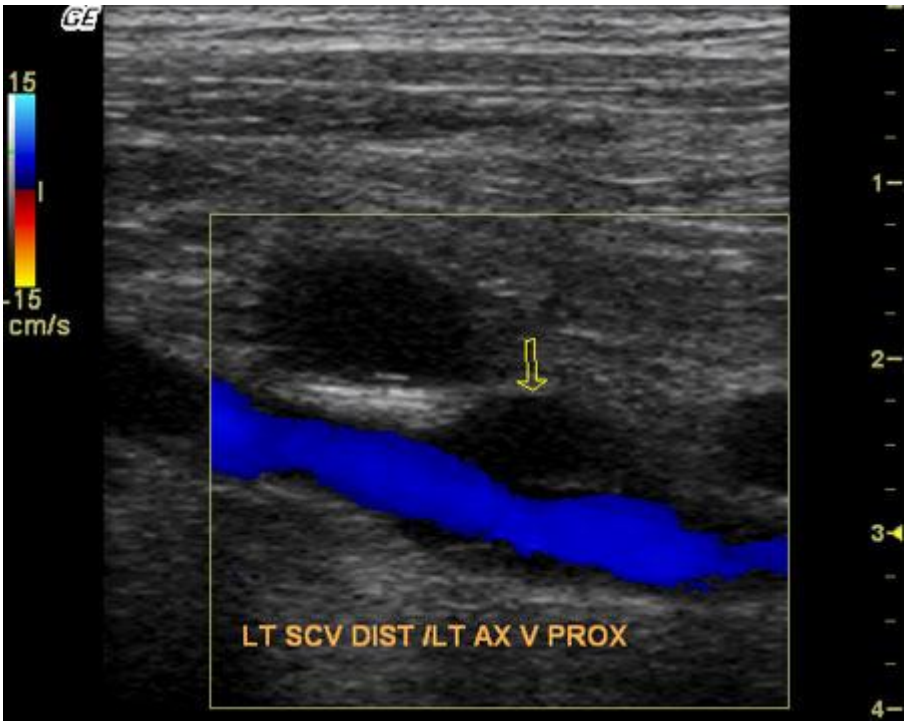

Figure 4: Early thrombus is forming at the site of a venous valve. This segment of the axillary vein just distal to the subclavian vein is the site of extrinsic compression by the pectoralis muscle. This compression can accentuate venous stasis as well as incite mechanical injury to the lining of the vein wall. The damaged endothelium then becomes a possible site for thrombus formation.

Patients who have PICC lines in place also have a higher risk of upper extremity venous thrombosis. Placement of the PICC causes local trauma to the vein wall (figure 5 a and b). In addition, the native vein diameter is small enough for the presence of the catheter to promote stasis and incite a superficial thrombophlebitis. Such a benign superficial thrombophlebitis can then spread to the more central veins and become a risk for pulmonary embolism. Larger catheters increase the likelihood that stasis can develop. In addition, such catheters are often used in patients with malignancy. The presence of malignancy in itself is associated with an increased susceptibility to develop thrombosis.

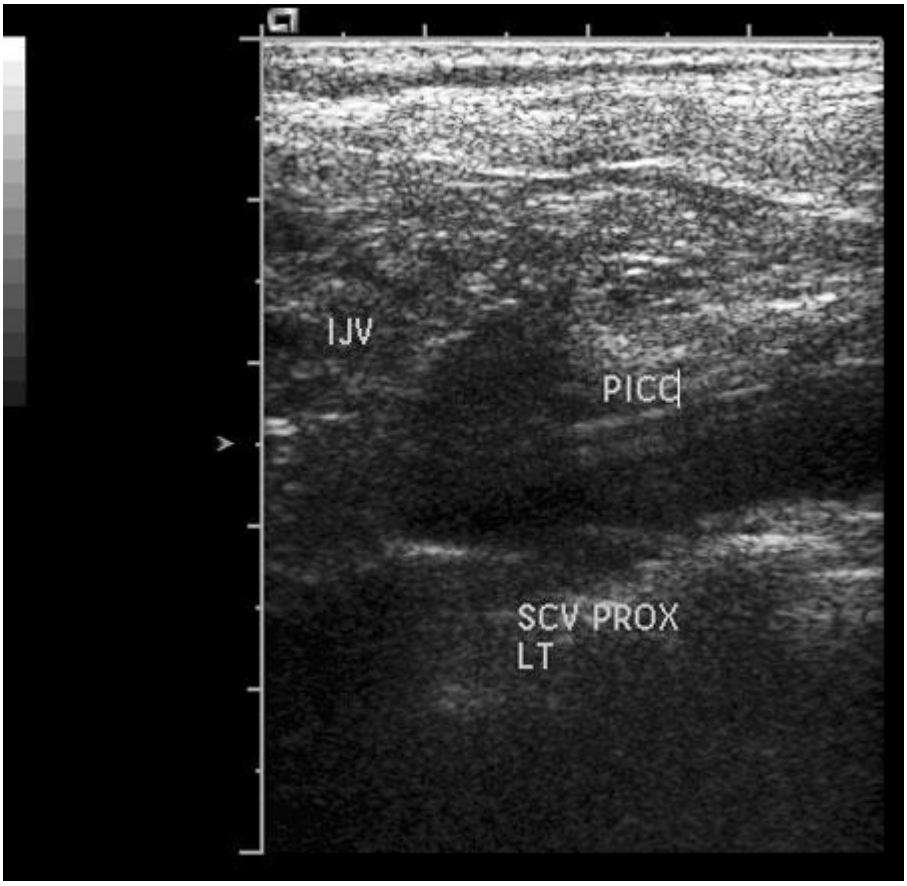

Figure 5a: A PICC will typically be placed in the cephalic, basilic or brachial veins. Placement in the basilic and cephalic veins is possible by palpation, specifically at the elbow. Ultrasound guidance is used for accurate placement. This is considered essential if the brachial vein is used since the brachial veins are in close proximity to the brachial artery. On this image, the upper portion of a PICC is seen as it transits towards a more central location and is shown at the level of the brachiocephalic vein.

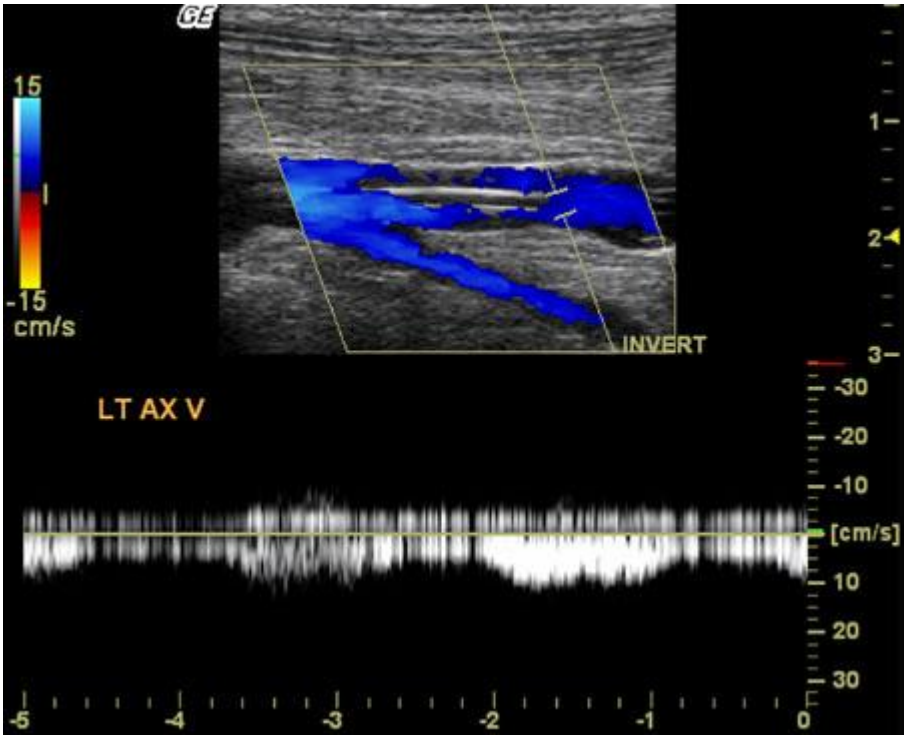

Figure 5b: This color Doppler image shows blood flow around the catheter. This is a typical finding since the PICC is normally smaller than the diameter of the vein. Absence of blood flow should be considered as possible evidence of a developing obstructive vein thrombosis. The small size of the cephalic vein

increases the likelihood of thrombus forming around the catheter.

The presence of stasis is also increased in patients with right heart failure or with central mediastinal masses compressing the central veins. Mediastinitis can also cause peri-vein fibrosis and narrow the vein, also resulting in stasis.
However, the general principle of deep vein thrombus formation remains venous stasis. As is the case in the lower extremity, stasis is typically seen at the site of a venous valve in the upper extremity vein.

TABLE 1: TYPES OF UPPER EXTREMITY VEIN THROMBOSIS

- Idiopathic: no predisposing risk factors
- Iatrogenic: catheter related
- Traumatic, including thoracic outlet syndrome and effort thrombosis
- Spontaneous in association with risk factors such as malignancy, congestive heart failure, or a mediastinal process

VENOUS ACCESS

Verification of vein location, size, and patency is increasingly needed in the hospitalized patient. This is made necessary in patients who have had previous long-term requirements for central access catheters. These patients have tunneled dialysis catheters or other tunneled catheters used to maintain long-term venous access for chemotherapy and total parenteral nutrition (TPN). Ultrasound evaluation is requested to confirm patency in order to determine suitability for placement of new venous lines, pacemakers, and dialysis catheters.
This approach is especially useful since catheter placement in these individuals is often difficult and requires ultrasound guidance in order to be successful.

DIAGNOSTIC CRITERIA: COMPRESSIBILITY

This remains the more accurate criterion for making the diagnosis of acute venous thrombosis of either the deep or superficial veins.
All of the upper extremity veins are, unfortunately, not accessible to this maneuver. The basilic, cephalic, brachial, and jugular veins are readily accessible (figure 2). The axillary and subclavian veins are poorly accessible over portions of their course through the arm and chest wall. The axillary vein specifically in the upper arm as it transits in the axilla since it can separate from the close proximity to the artery in the deep axilla (figure 6). The compression maneuver will nevertheless be effective in confirming the absence of a thrombus in the vein.

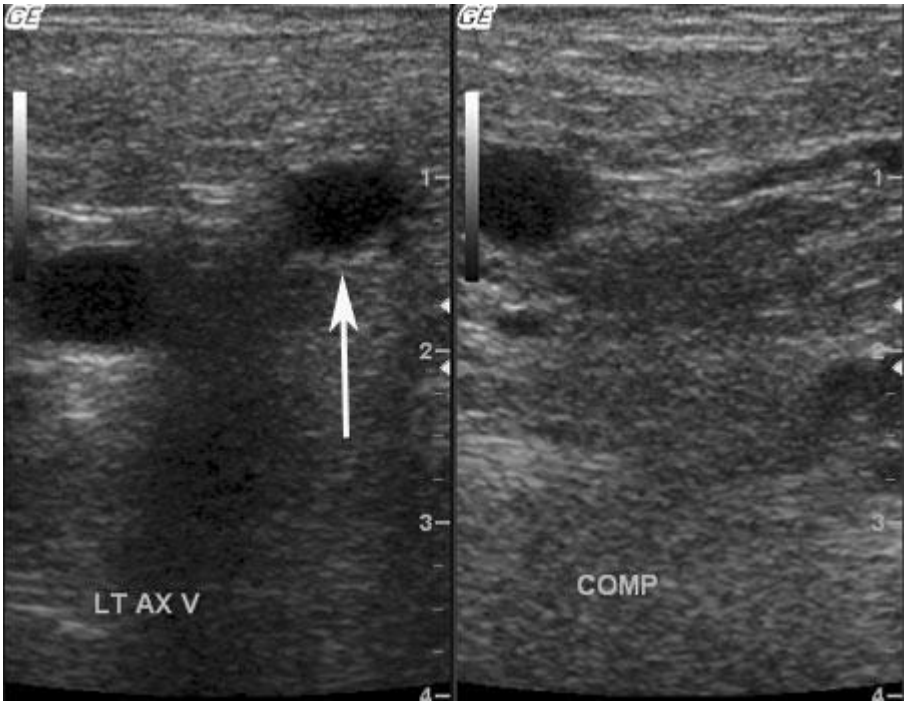

Figure 6: The axillary vein (arrow) can separate from the the artery in the deep axilla. The compression maneuver will nevertheless be effective in confirming the absence of a thrombus in the vein.

DIAGNOSTIC CRITERIA: BLOOD FLOW EVALUATION

The response to flow augmentation is less reliable in the upper extremity than it is in the lower extremity veins. There is also marked variability in the color Doppler and spectral waveforms that mimic arterial signals.

Normally, strong pulsatility should be transmitted from the right atrium to the superior vena cava, the brachiocephalic and then the jugular, subclavian and axillary veins.

These veins will typically have waveforms with a pulsatile appearance. Forward flow into the atrium causes a sharp increase in blood flow velocity that decreases as the atrium fills (figure 7a). Blood flow will then reverse as the ventricle contracts and some of the blood in the atrium can flow retrograde. Proper positioning in a supine position may be necessary to increase venous pressure sufficiently to demonstrate the normal pulsatile blood flow pattern (figure 7b).

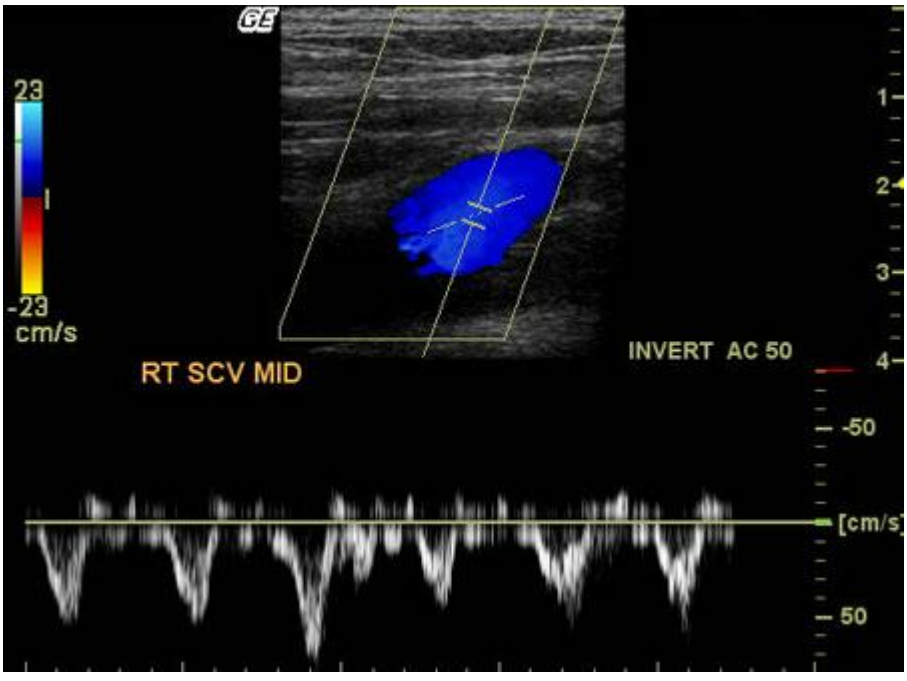

Figure 7a: The Doppler velocity tracings from the central veins (subclavian, internal jugular and subclavian) will typically have a pulsatile appearance. Forward flow into the atrium causes a sharp increase in blood flow velocity that decreases as the atrium fills. Blood flow will then reverse as the ventricle contracts and some of the blood in the atrium can flow retrograde.

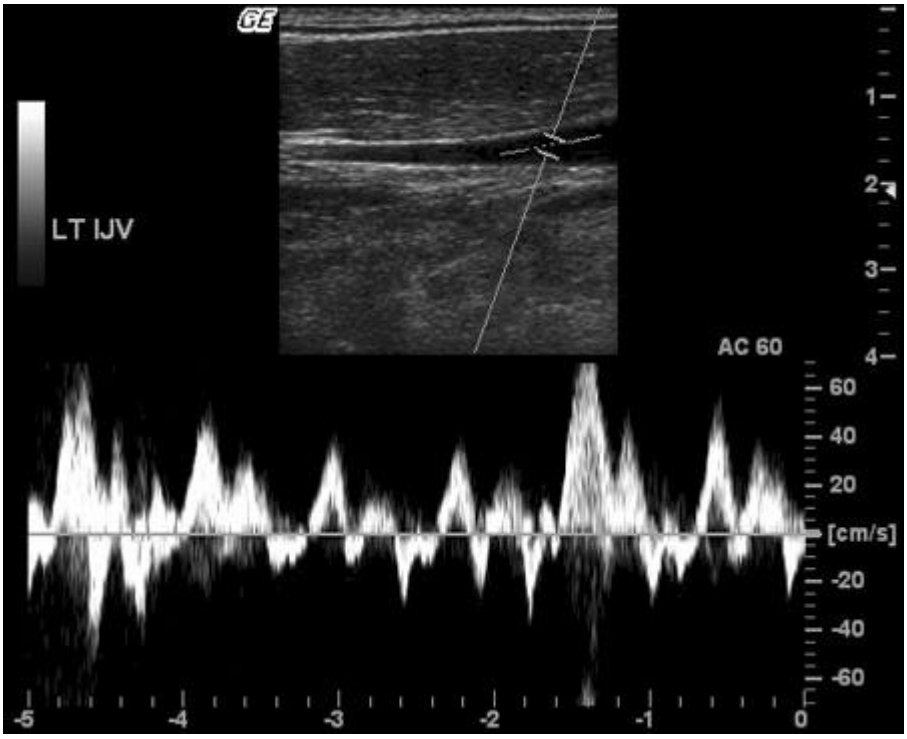

Figure 7b: This pattern of blood flow can be difficult to perceive when the jugular vein is not distended. They can be accentuated when the vein is poorly distended (note the partly collapsed internal jugular vein) and because of changes due to breathing (changes intrathoracic pressures affect the blood flow patterns). This causes the pattern of blood flow to become more complex.

Loss of pulsatility, especially if it is unilateral suggests central venous obstruction, most often at the brachiocephalic vein. Bilateral loss of pulsatile signals suggests central obstruction of the superior vena cava or of both brachiocephalic veins (figure 8).

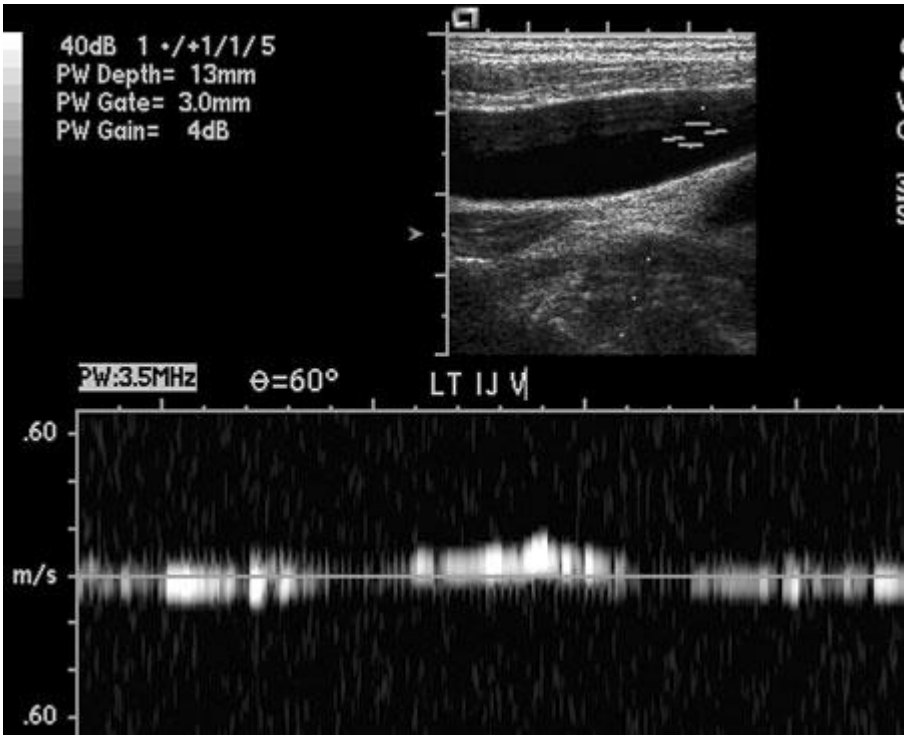

Figure 8. This waveform in the internal jugular vein shows evidence of central venous obstruction. The pulsatile waveform due to changes in the cardiac cycle are no longer being transmitted due to an obstructed brachiocephalic vein.

On occasion, a maneuver that is indicative of central venous obstruction is the response of the vein to rapid inspiration (or a "sniff"). The vein diameter decreases in response to rapid inspiration since the negative intra-thoracic pressure promotes blood flow into the chest. A normal response can exclude obstruction of the more central veins (brachiocephalic veins and superior vena cava). However, patients with very chronic central venous obstructions can have large collaterals veins and regain normal response to this respiratory maneuver.

When compared side by side as diagnostic criteria, loss of pulsatility seems to perform with greater accuracy than evaluations of venous dynamics due to respiratory phasicity.

DIAGNOSTIC CRITERIA: THROMBUS VISUALIZATION

Detection of echogenic signals may be the only evidence of non-obstructing or mural thrombus in the more central brachiocephalic, subclavian and jugular veins. It is often seen in association with an indwelling catheter or as a non-obstructing adherent thrombus.

Ultrasound images of deep vein thrombosis of internal jugular vein typically show a distended vein that contains echogenic signals (figure 9). This type of venous thrombosis is often completely asymptomatic.

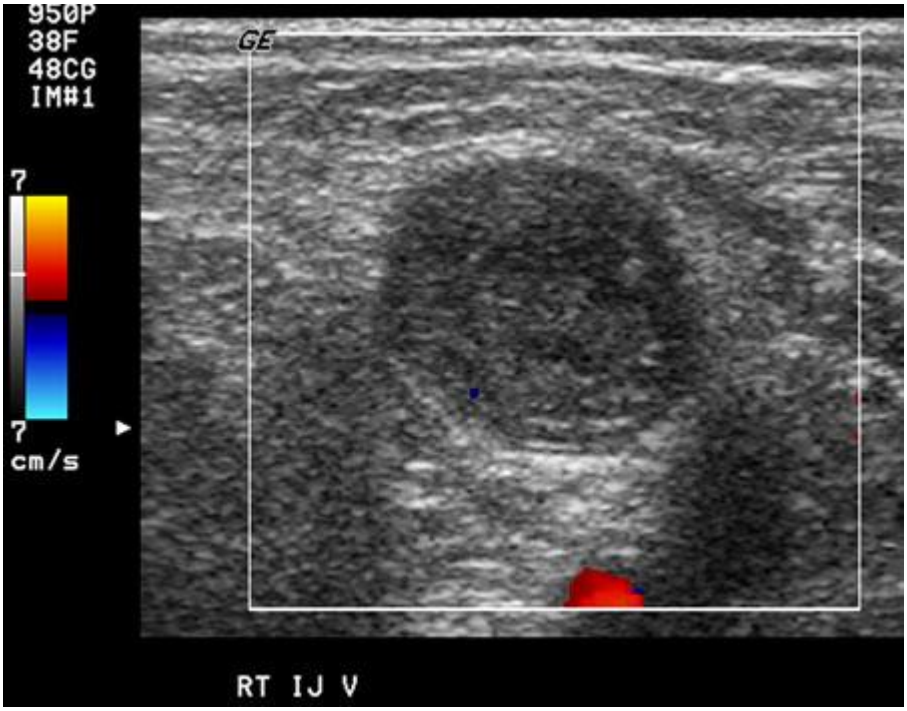

Figure 9: This internal jugular vein is distended and contains echogenic signals consistent with acute deep vein thrombosis. This type of venous thrombosis is often completely asymptomatic.

The lack of symptoms is a major reason why previously instrumented patients will present with evidence of a previous thrombosis of the internal jugular vein (figure 10a). Typically, the vein will not be visualized or will show up as a small non-compressible structure. On occasion, the vein can become recanalized (figure10b). The arm veins may not distend with acute deep vein thrombosis as is the case in the lower extremity. This may be due to the trauma of multiple blood draws or catheter placements (figure 11).

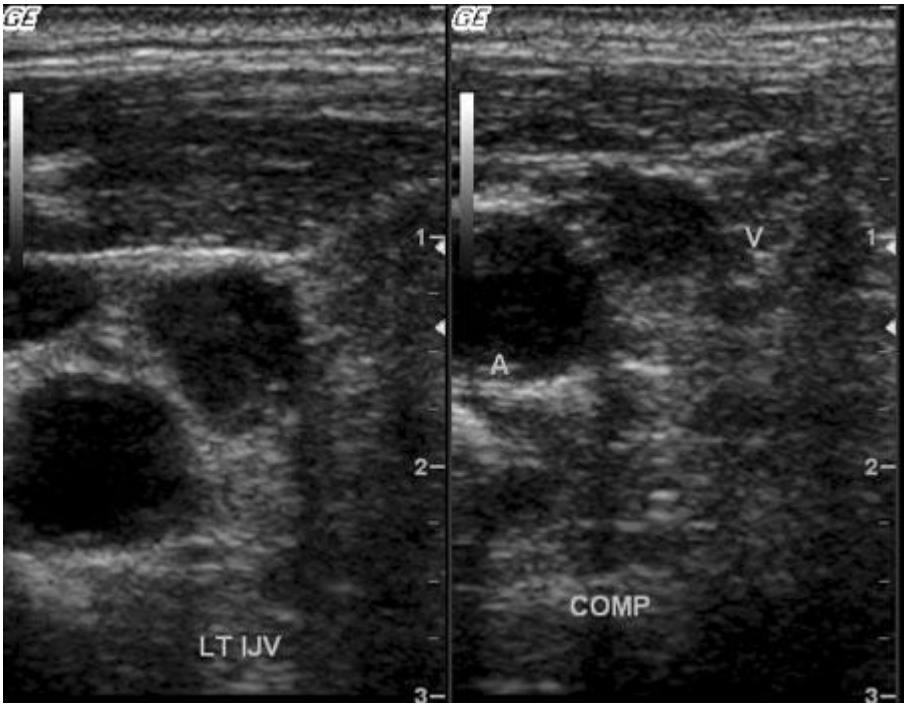

Figure 10a: This internal jugular vein is not distended and contains mostly low echogenic signals. The vein is poorly compressible. This is evidence of a previous deep vein thrombosis in a patient who had a previous indwelling venous catheter. This finding is consistent with a chronic or subacute deep vein thrombosis.

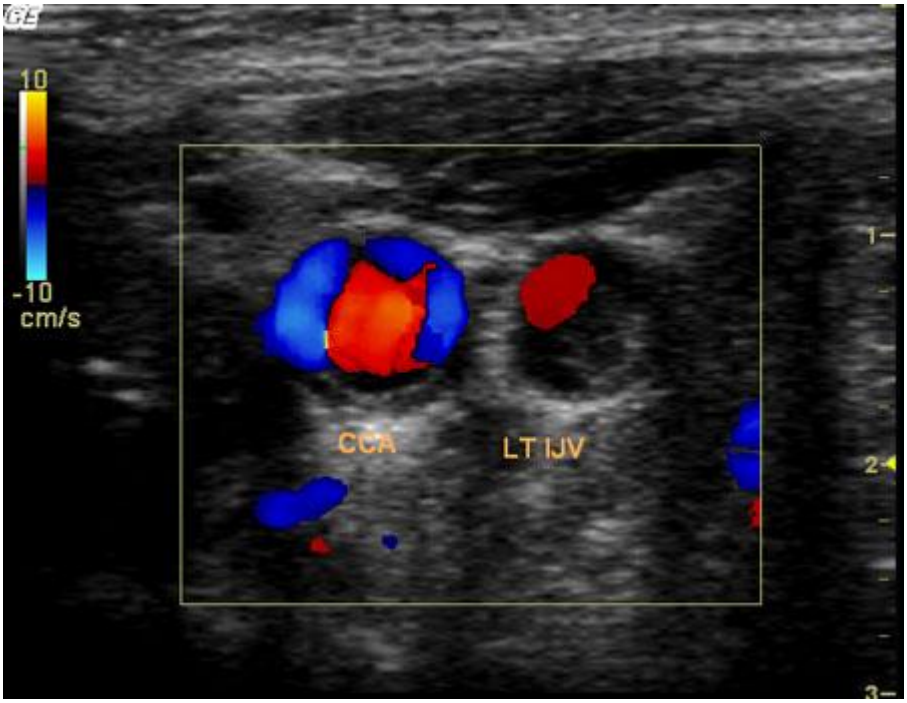

Figure 10b: A color Doppler image from the same patient shows a vein shows low echogenic thrombus and a recanalized lumen. Such recanalized venous segments may be difficult to perceive without the concurrent use of color Doppler imaging.

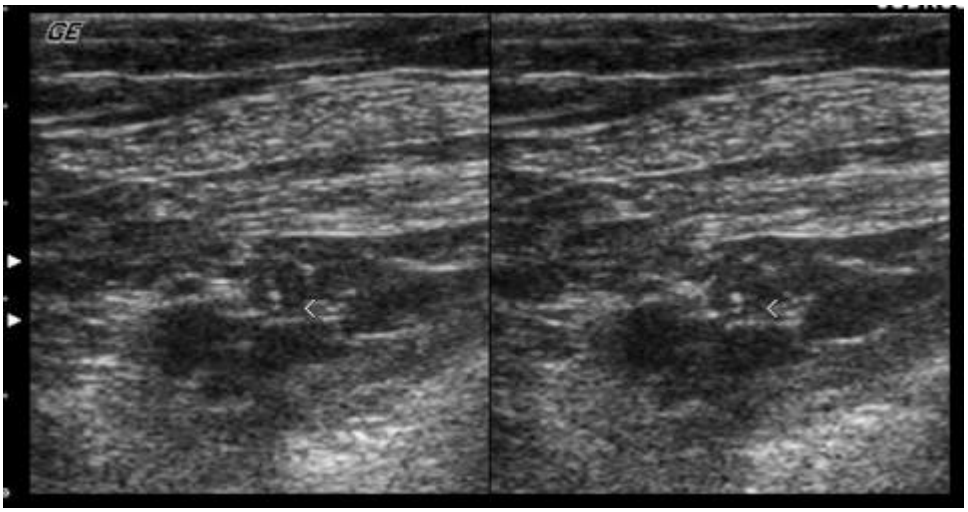

Figure 11: This brachial vein (arrowhead) contains echogenic signals. The vein is non-compressible. The source of the echogenic signals is actually a PICC whose placement was responsible for the upper extremity thrombosis.

TABLE 2: DIAGNOSTIC CRITERIA THAT CAN BE APPLIED TO THE VARIOUS SEGMENTS OF THE UPPER EXTREMITY VEINS

Vein	Compressibility	Flow	Echogenicity
Brachials	+++	+	+/-
Axillary (distal)	++	+	+/-
Axillary (middle)	+ - ++	+	+/-
Axillary (proximal)	+++	++	+/-
Subclavian D1/3	+	++	+/-

Subclavian M1/3	-	++	+/-
Subclavian P1/3	-	++	+
Jugular	+++	++	++
Brachiocephalic	-	++	-

The diagnostic criteria used for the diagnosis of acute DVT in the lower extremity veins can be applied to the upper extremity veins. The relationship of the subclavian vein to the clavicle forces some change in the way these diagnostic criteria are applied. The internal jugular vein, the lateral portion subclavian vein, the axillary vein axilla) and more peripherally located veins can be evaluated with compression ultrasound. The subclavian vein below the clavicle and medial to it cannot be compressed (figure 12). Color Doppler imaging and gray scale imaging are used for evaluating the proximal subclavian since the vein cannot be compressed. The innominate (right brachiocephalic) and left brachiocephalic veins are evaluated by relying on gray scale images and the presence of echogenic signals associated with thrombi and detecting alterations in the flow patterns within the vein lumen with the aid of color Doppler signals (figure 13). This approach requires great care while setting the sensitivity and velocity scale of the color Doppler signals. Relatively hypoechoic thrombi can easily be missed if the gain settings are set too high and the velocity scale too low.

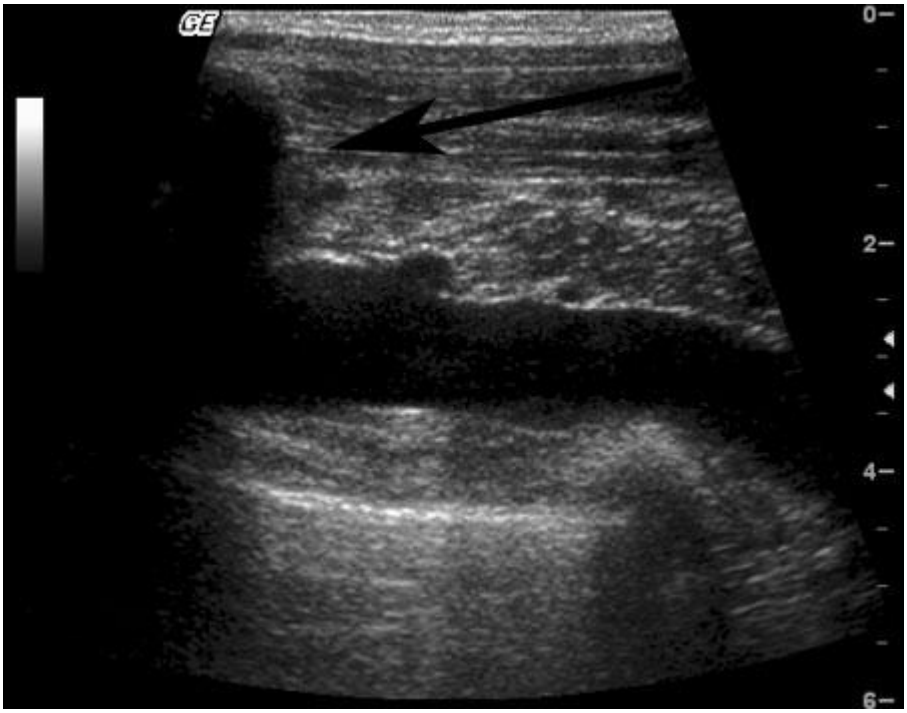

Figure 12: The portion of the subclavian vein cannot be imaged as it crosses under the clavicle (black arrow). Color Doppler evaluation cannot be used either. Doppler waveform analysis can help confirm the absence of an obstructing thrombus (figure 7a and 8). Smaller, non-obstructing thrombi could be missed since they would not perturb any blood flow patterns.

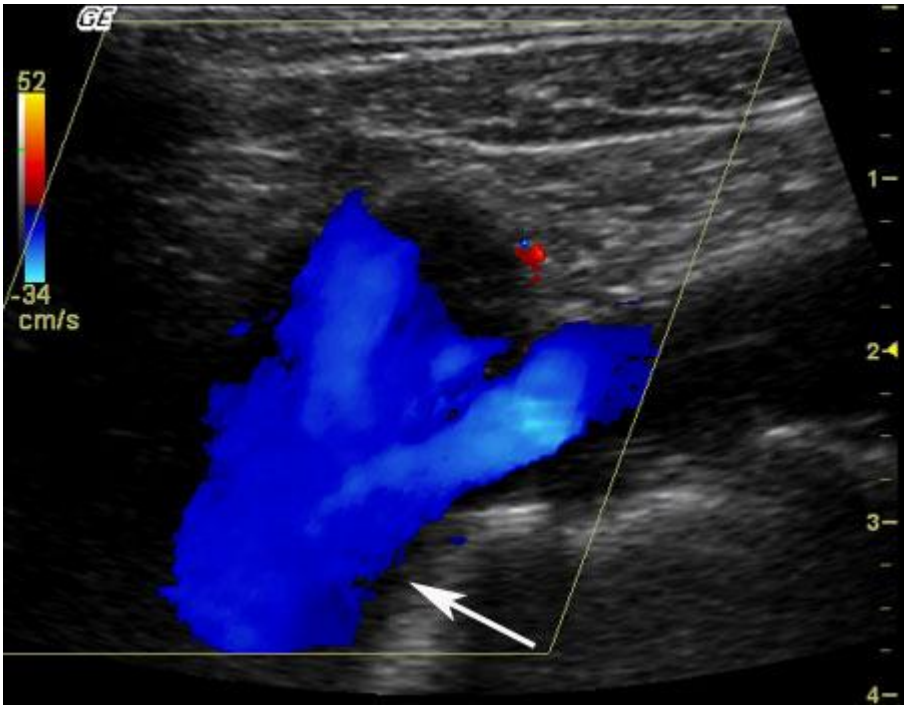

Figure 13: Color Doppler imaging is, however, very useful in evaluating the brachiocephalic junction (arrow), confirming the absence of localized thrombus. Care must be given to afjust the gain of the Doppler settings since the color Doppler signals can paint over relatively echolucent (hypoechoic) thrombus.

Doppler ultrasound can document the presence of a total occlusion but lacks diagnostic accuracy for detecting partly obstructing thrombus. Flow augmentation is often difficult to elicit in the upper extremity veins. Adjunctive images showing intact blood flow signals through the arm veins are, as is the case for the leg, reassuring evidence of the absence of thrombi (figure 14).

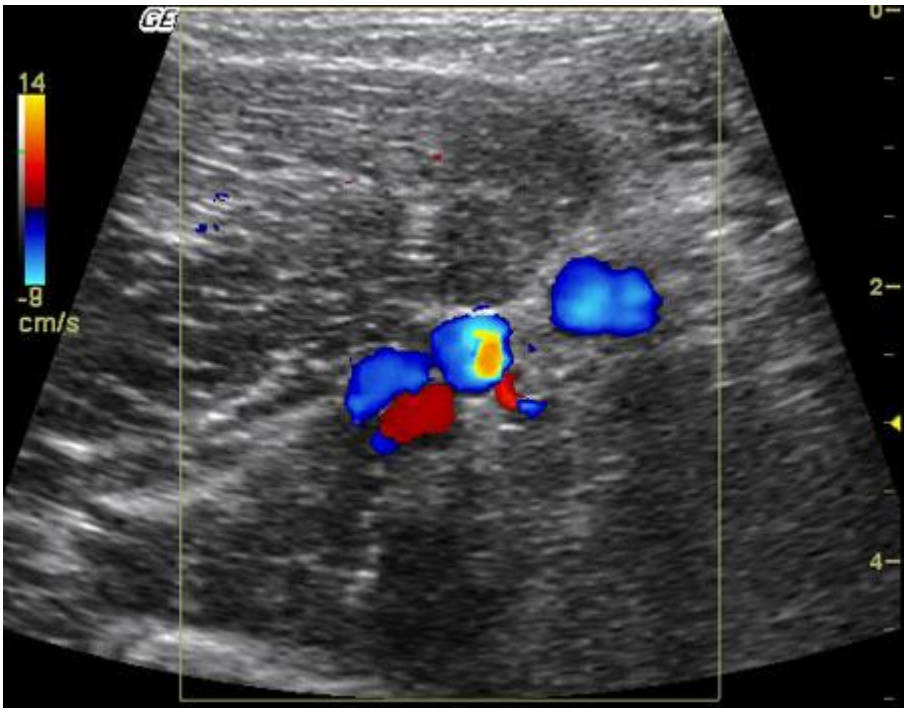

Figure 14: Color Doppler imaging can also be used to confirm patency of the vein segments that can also be compressed. Such images are reassuring of the absence of any thrombus. The brachial artery sits in between both brachial veins, as shown on this image.

Echogenic signals in the vein of a patient who has recently had a catheter removed may represent a fibrin sheath (figure 15). This is a sheath that forms around the catheter as it sits in the vein. The fibrin sheath and normally fragments as the catheter is removed. It is not considered to be a serious risk to the patient's well being. It is considered a self limited process and rarely associated with clinically detectable pulmonary embolism.

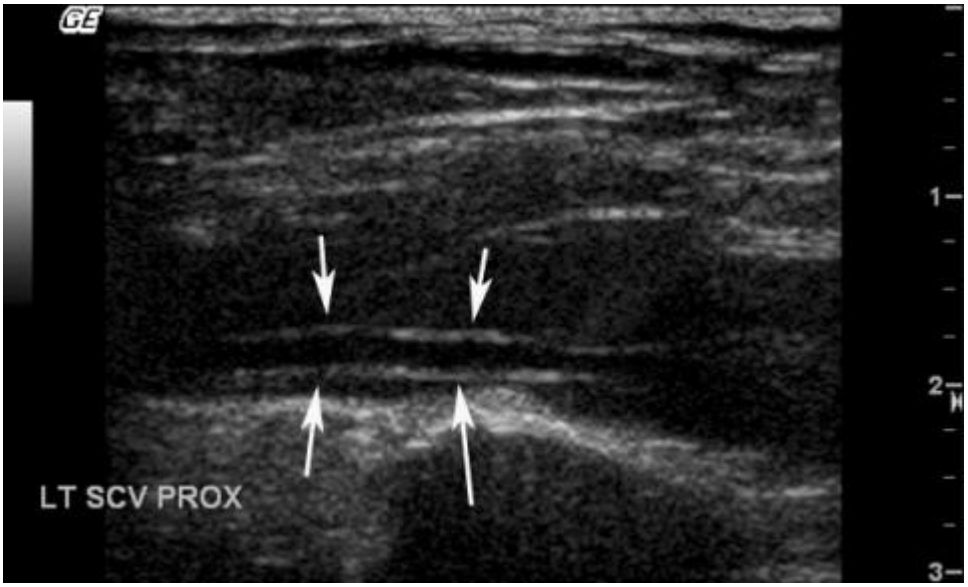

Figure 15: This patient recently had a catheter removed. A fibrin sheath remains in the vein lumen. Fibrin sheaths can be of various lengths. They are not believed to represent any risk to the patient.

SUMMARY

Upper extremity ultrasound can be used to make the diagnosis of deep and superficial vein thrombosis. It offers a practical means of evaluating patency for planned catheter insertions. It can be used to diagnose possible catheter related complications. As with any Doppler imaging technique, an understanding of the basic physiology is very useful in insuring the performance of a high quality examination.

REFERENCES

- Baarslag HJ, van Beek EJ, Koopman MM, Reekers JA. Prospective study of color duplex ultrasonography compared with contrast venography in patients suspected of having deep venous thrombosis of the upper extremities. Annals of Internal Medicine 2002; 136:865-72.
- Burbridge SJ, Finlay DE, Letourneau JG, Longley DG. Effects of central venous catheter placement on upper extremity duplex US findings. JVIR 1993; 4: 399-404
- Falk, RL, Smith DF. Thrombosis of upper extremity thoracic inlet veins: Diagnosis with duplex Doppler sonography. AJR 1987; 149:677-682.
- Grassi CJ, Polak JF. Axillary and subclavian venous thrombosis: Follow-up evaluation with color Doppler flow US and venography. Radiology 1990; 175:651-654.
- Haire WD, Lynch TG, Lieberman RP, Lund GB, Edney JA. Utility of duplex ultrasound in the diagnosis of asymptomatic catheter-induced subclavian vein thrombosis. J Ultrasound Med 1991; 10: 493-496
- Hood DB. Kuehne J. Yellin AE. Weaver FA. Vascular complications of thoracic outlet syndrome. American Surgeon. 63:913-7, 1997
- Hubsch PJS, Stiglbauer RL, Schwaighofer WAM et al. Internal jugular and subclavian vein thrombosis caused by central venous catheters: Evaluation using Doppler blood flow imaging. J Ultrasound Med.

1988; 7:629-636.

- Knudson G, J., Wiedmeyer, D, A., Erickson, S, J., Foley, W, D., Lawson, T., L, Mewissen, M, W., Lipchik, E, O. Color Doppler sonographic imaging in the assessment of upper-extremity deep venous thrombosis. AJR 1990; 154: 399-403
- Luciani A, Clement O, Halimi P, et al. Catheter-related upper extremity deep venous thrombosis in cancer patients: a prospective study based on Doppler US. Radiology 2001; 220:655-60
- Patel MC, Berman LH, Moss HA, McPherson SJ. Subclavian and internal jugular veins at Doppler US: abnormal cardiac pulsatility and respiratory phasicity as a predictor of complete central occlusion. Radiology 1999; 211:579-83.
- Prandoni P. Bernardi E. Upper extremity deep vein thrombosis. Current Opinion in Pulmonary Medicine. 5:222-6, 1999
- Prandoni P. Polistena P. Bernardi E. Cogo A. Casara D. Verlato F. Angelini F. Simioni P. Signorini GP. Benedetti L. Girolami A. Upper-extremity deep vein thrombosis. Risk factors, diagnosis, and complications Archives of Internal Medicine. 157:57-62, 1997
- Prescott SM and Tikoff G. Deep venous thrombosis of the upper extremity: A reappraisal. Circulation 1979; 59:350-355.
- Zell L, Kindermann W, Marschall F, Scheffler P, Gross J, Buchter A. Paget-Schroetter syndrome in sports activities--case study and literature review. Angiology 2001; 52:337-42.