Vascular Imaging for the Primary Care Provider

Venous and Arterial Disease

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KEYWORDS

• Peripheral artery disease • Vasospastic disorders • Leg swelling
• Abdominal aortic aneurysm • Carotid artery disease • Vascular ultrasound

KEY POINTS

- This article will outline the clinical utility and value of the vascular lab in the diagnosis, treatment, and surveillance of common vascular diseases.
- Peripheral artery disease (PAD) is often underdiagnosed which can lead to significant cardiovascular morbidity and mortality. The vascular lab is instrumental in early detection of PAD as well as the ongoing surveillance of known disease, which can benefit in the selection of optimal therapies including medical management and interventions.
- Lower extremity swelling is a common complaint in primary care patients, and the vascular lab can help differentiate between the numerous underlying etiologies for this issue. DVT protocol or venous insufficiency studies help facilitate the diagnosis utilizing these non-invasive diagnostic modalities.
- Screening for abdominal aortic aneurysms (AAA) is an important part of general health maintenance and can be accomplished using arterial duplex ultrasound. This imaging technique can also be utilized for expansion surveillance in patients with a known AAA, using consensus guidelines for imaging intervals.
- Carotid artery disease can be diagnosed and followed on a serial basis utilizing carotid artery duplex ultrasound. Screening for carotid artery disease is not recommended for asymptomatic patients.

APPROACH TO THE SUSPECTED PERIPHERAL ARTERY DISEASE PATIENT

Peripheral artery disease (PAD) most often describes lower extremity obstructive arterial disease with the most common etiology of atherosclerosis. PAD affects approximately 8 to 12,000,000 people in the US and over 200 million people worldwide.

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The incidence increases with age and the prevalence is nearly twice as high in the African American population as compared to the Caucasian population. PAD affects men and women equally, and is a strong predictor of mortality, myocardial infarction, and stroke. Risk factors for PAD include age, hypertension, hyperlipidemia, diabetes, advanced chronic kidney disease, tobacco use, and a genetic disposition. Unfortunately, PAD is often under diagnosed and undertreated, and this can lead to excessive morbidity or mortality. The diagnosis of PAD should be entertained in patients at high risk or who have clinical features suggestive of this disease. This can be challenging as the majority of patients can be asymptomatic. The minority of patients will relate classic symptoms of intermittent claudication or present with ischemic rest pain. Consideration for PAD is recommended in patients with symptoms of claudication, atypical leg pain, functional impairment, significant cardiovascular risk factors, abnormal lower extremity pulse exam, non-healing wounds, or ischemic rest pain.[1–5]

Patients with risk factors and clinical features suggestive of peripheral artery disease should undergo a comprehensive vascular evaluation. Physical examination should include bilateral brachial artery blood pressures, skin examination, and examination of peripheral arteries including palpation and auscultation. Patients with PAD have an increased risk of subclavian artery stenosis and should have bilateral brachial blood pressures obtained at the initial encounter. Pressure gradients greater than 15 to 20 mm Hg are abnormal and suggestive of subclavian (or innominate) stenosis. Skin changes in PAD include mottling, arterial ulcers, gangrene, limb or digital color changes, or coolness to touch. Peripheral pulses should be examined including auscultation for bruits, and palpation of femoral, popliteal, and distal pulses including the dorsalis pedal and posterior tibial artery.

Diagnostic imaging involves the vascular lab and guidelines for imaging include surveillance for patients with established vascular disease, post intervention or surgery imaging, at-risk individuals (age > 65, age 50–64 with risk factors for atherosclerosis, age <50 with diabetes + 1or more risk factor for atherosclerosis, patient with non-healing wound and ulcers) and in patients with suspected PAD based on history and physical examination findings listed above.

Functional assessment includes the ankle and toe brachial index, exercise ankle brachial index (ABI), and pulse volume recordings (PVRs).[6] The ABI can be easily utilized in the office setting to diagnose PAD. Blood pressure cuffs are placed on the upper arms and ankles bilaterally and inflated above systolic pressure. As the pressure is released, the onset of flow is detected by the placement of a Doppler probe over the brachial and both pedal arteries (posterior tibial and dorsalis pedis). The ABI of each leg is calculated by dividing the higher of the two pedal arteries by the higher of the arm blood pressure. An ABI of less than 0.9 is used to make the diagnosis of PAD. The ABI has a sensitivity of 68% to 84% and a specificity of 84% to 99% for the diagnosis of PAD. Limitations of the ABI include medial arterial calcification resulting in non-compressible arteries that may lead to false elevation (ABI > 1.4) or artificial normalization of ABI. The ABI may also be normal or borderline abnormal at rest in patients with aortoiliac disease Interpretation of PAD is based on numerical findings as outlined in **Table 1**.[7] **Fig. 1** depicts the technical aspects of obtaining an ankle brachial index.

Exercise ABI may be helpful in unmasking PAD (especially proximal aorta iliac inflow disease) with normal resting ABI's. Patients with PAD may be asymptomatic at rest and only develop claudication symptoms with exertion. In such individuals, it is possible for resting ABI measurements to be normal. As a result, when the suspicion for PAD is high and resting measurements are normal, it is recommended to obtain an exercise ABI. Usual protocols involve fixed treadmill settings of 2 miles per hour at a 2% grade for a maximum of 5 minutes. Pre- and post-exercise ABI can be obtained

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Table 1
Interpretation of ankle-brachial index (ABI)

Ankle-Branchial Index (ABI)	Interpretation
1.0–1.4	Normal
0.91–0.99	Borderline range
0.70–0.90	Mildly abnormal
0.40–0.69	Moderately abnormal
<0.40	Severely abnormal
>1.4	Incompressible vessels

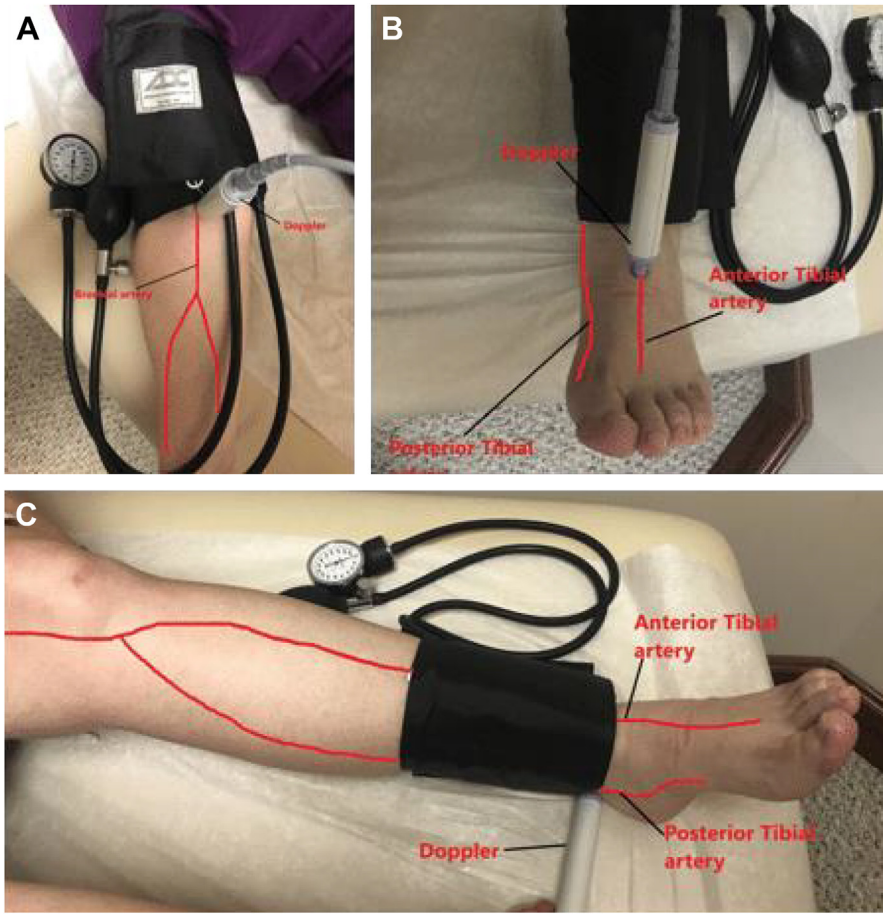

Fig. 1. Performing an ABI. (*A*) Measurement of blood pressure at the level of brachial artery. (*B*) Pulse volume recording at the area of anterior tibial artery and posterior tibial artery by doppler ultrasound. (*C*) Measurement of blood pressure at the level of lower leg. (Photographs courtesy of G. Jay Bishop M.D.)

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with an abnormal result defined as a decrease in the post exercise ankle pressures and/or ABI result by greater than 20%. Claudication symptoms can be reproduced with exercise and quantified regarding time and distance as well. Fig. 2 depicts an abnormal exercise ABI study with a drop in ankle pressures and ABI greater than 20%, consistent with exercise induced peripheral artery disease.

Pulse volume recording (PVR) is a non-invasive physiologic imaging modality that is helpful in the care of the patient with suspected or known PAD. This test refers to the graphic representation of the change in volume of the pulse contour in a specific segment of the extremity during the cardiac cycle. They are typically obtained by using BP cuffs placed at the high thigh, low thigh, calf, ankle, midfoot, and toe and may be used in

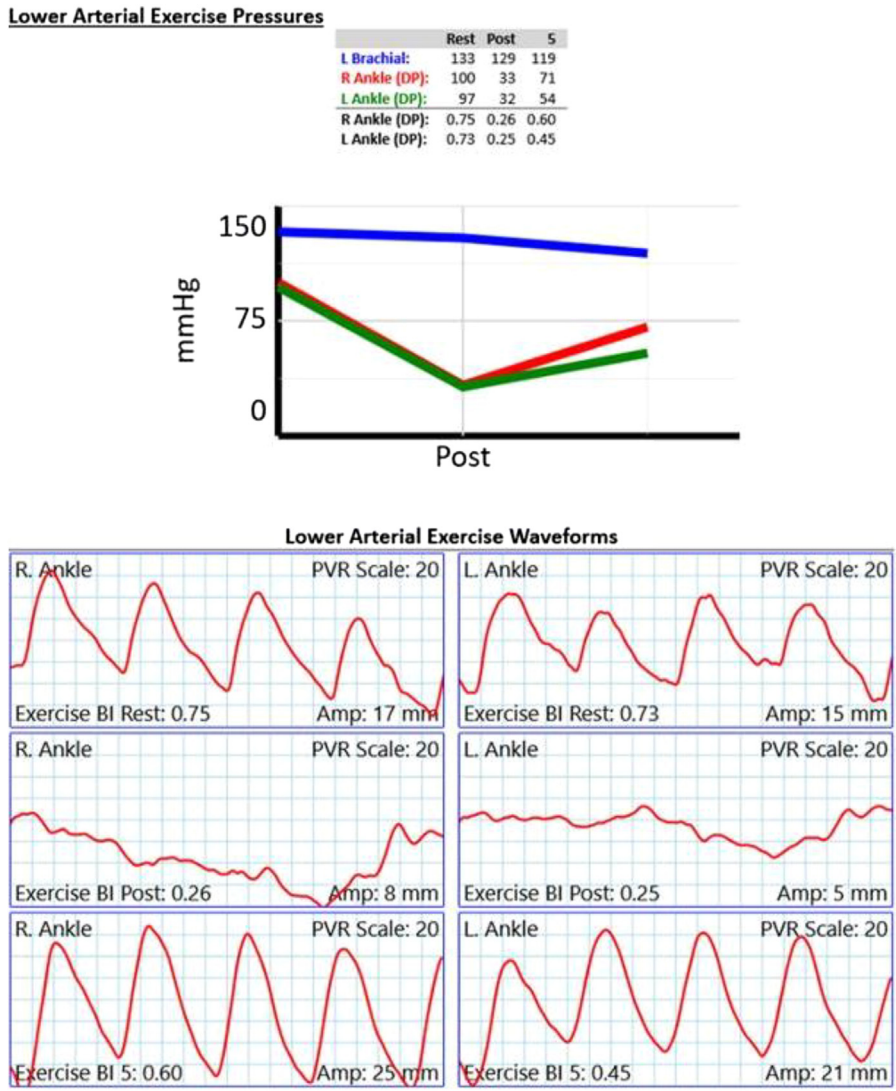

Fig. 2. Exercise ankle brachial index study – pre- and post- Exercise.Ankle pressure and ABI. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

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conjunction with segmental pressures. PVRs are usually paired with the ABI as a single test at most vascular labs as PVR can help localize anatomic segments of the disease (in contrast to the ABI). PVRs also have the advantage in patients with noncompressible vessels as they can still yield diagnostic information through review of the pulse volume contours and amplitude. They also can localize diseases including aorta iliac inflow, femoral/popliteal, infrapopliteal, and small vessel disease. However, despite their ability to localize disease, PVR measurements do not yield any information about specific lesion characteristics (ie, length, occlusion) and have decreased sensitivity for distal disease when inflow disease is present. Abnormal PVR findings include decreased amplitude, flattened peaks, absent dicrotic notch, and decreases in segmental pressure gradients between cuffs greater than 20 mm Hg which aid in the diagnosis of arterial occlusive disease. Examples of a normal PVR study are noted in **Fig. 3**.

Anatomic assessment can be obtained utilizing *arterial duplex ultrasound*. This noninvasive modality is a useful adjunct to non-invasive physiologic testing. This test is typically obtained for a more focused evaluation of the lower extremity arterial system. This imaging option utilizes a combination of B-mode ultrasound (US) imaging and spectral Doppler analysis. Doppler complements the standard qualitative US imaging by allowing waveform analysis and assessment of peak systolic velocities (PSV). Using the concept that the velocity of blood flow increases as it flows through a stenotic lesion, peak systolic and end-diastolic velocities are measured and used to estimate the severity of stenosis. Arterial flow characteristics can be normal or abnormal.

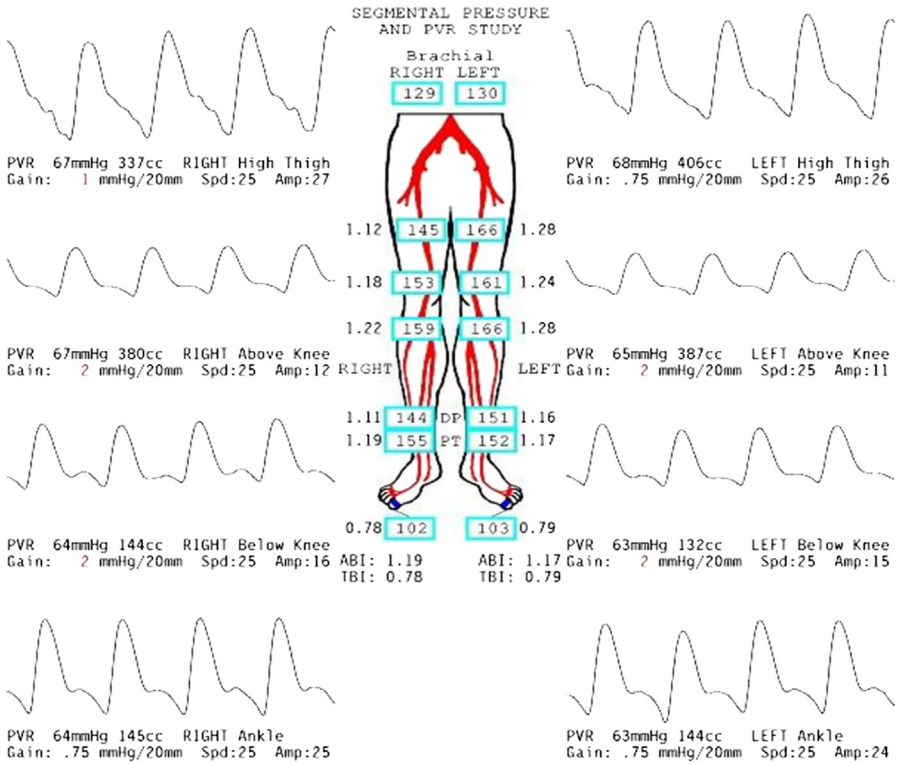

Fig. 3. Normal pulse volume recording study. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

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This modality is useful for the anatomic visualization of lesions and for surveillance after stenting or bypass grafting. Arterial duplex ultrasonography can indicate the level and severity of occlusions, patency of stents or bypass grafts, evaluate AV fistulas for dialysis, and screen for pseudoaneurysms and other post-interventional or post-surgical complications. A class IIa recommendation is given for routine surveillance using duplex ultrasound following infrainguinal revascularization with a goal of early identification of high-grade stenosis (PSV > 300 cm/s) and impending graft failure (PSV < 40 cm/s).[7] Duplex image of normal flow through the right superficial femoral artery is noted in **Fig. 4**. **Table 2** outlines the diagnostic criteria for PAD utilizing peak systolic velocities and velocity ratios. These velocity criteria are less reliable when interrogating the infra-popliteal arteries.[8] **Fig. 5** depicts arterial duplex ultrasonography of a patent popliteal stent with gray scale and color flow images.

The approach to the patient suspected of peripheral artery disease includes utilization of the vascular lab for optimal diagnostic value. **Fig. 6** provides guidance on the diagnostic approach to the patient with suspected PAD. AHA/ACC consensus guidelines give a Class I recommendation for patients with history or physical examination finding suggestive of PAD, the resting ABI, with or without segmental pressures and waveforms is recommended to establish the diagnosis. A IIa recommendation is given in patients at increased risk of PAD but without history or physical examination finding suggestive or PAD, measurement of resting ABI is reasonable.[9]

APPROACH TO THE SUSPECTED VASOSPASTIC DISORDER

Vasospastic disorders are conditions that occur when blood flow is disturbed related to arterial or arteriolar spasms. This can result in mild color changes to severe ischemic pain and necrotic changes. This can involve several vascular conditions,

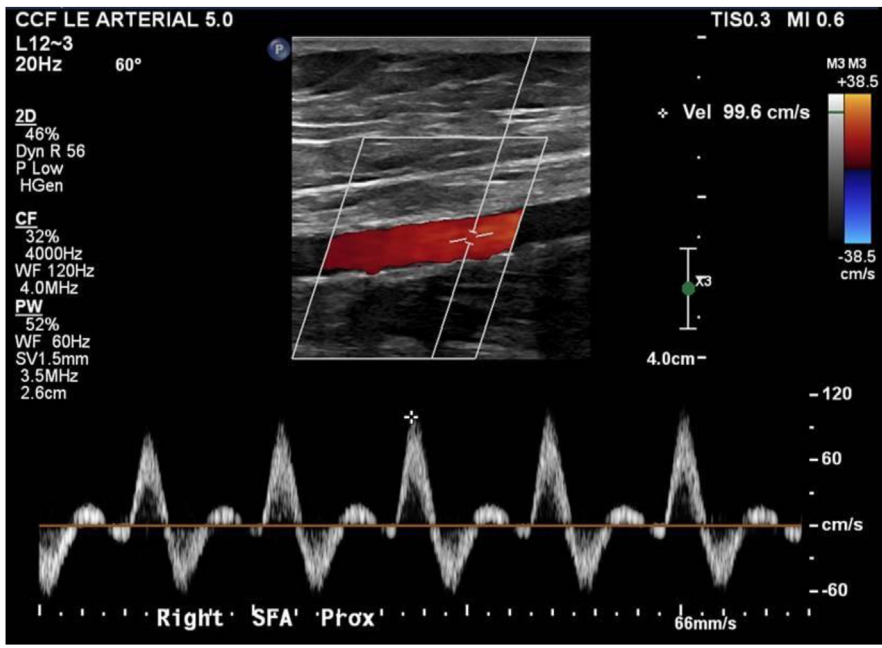

Fig. 4. Duplex arterial ultrasound of superficial artery showing normal flow and velocities. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

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Table 2
Diagnostic criterial for lower extremity arterial duplex scan for peripheral arterial disease (PAD)

Degree of Stenosis	Peak Systolic Velocity (cm/s)	Velocity Ratio
<20%	<150	<1.5
20%–49%	150–200	1.5–2.0
50%–80%	200–300	2.0–4.0
>80%	>300	>4.0
Occlusion	No flow detected in lumen	N/A

Adapted from Hodgkiss-Harlow KD & Bandyk DF. Semin Vasc Surg 2013;95-104.

including Raynaud's phenomena (that can be primary or secondary), acrocyanosis, peripheral cyanosis, pernio, and erythromelalgia.[10] Comprehensive history, physical examination and vascular laboratory testing are necessary to identify and assess the underlying etiology.

Raynaud's is caused by dysregulated constriction of the precapillary arterioles that lead to changes in skin color, swelling, and paresthesia. It generally affects the fingers and toes but can also affect other sites such as the nose, ears, and nipples.[11] Furthermore, Acrocyanosis is due to a decrease in the amount of oxygen delivered to the tissues of the extremities, and the precise mechanism remains elusive. Potential pathophysiological disturbances include abnormal arteriolar tone, alteration of microvascular responsiveness with capillary and venular dilation and stasis, and abnormal sympathetic nervous activity.[12] In contrast to Raynaud's, the bluish discoloration of acrocyanosis is persistent rather than episodic and is not associated with discomfort.

Noninvasive vascular laboratory testing can be used to document the presence and severity of vasospasm, assess for improvement with warming, establish an individual patient baseline and monitor improvement with therapy. Digital waveforms and thermal provocation studies are integral strategies in the approach to the patient with a suspected vasospastic disorder. Extremity segmental pressures include digital systolic pressures, sometimes with a temperature challenge. Digital waveforms can be mild, moderate dampening, or severely abnormal with flattening waveform. The measurement of cold recovery time is a classic test that utilizes photoplethysmography (PPG) or laser Doppler to monitor digital blood flow. Pulse volume recording can

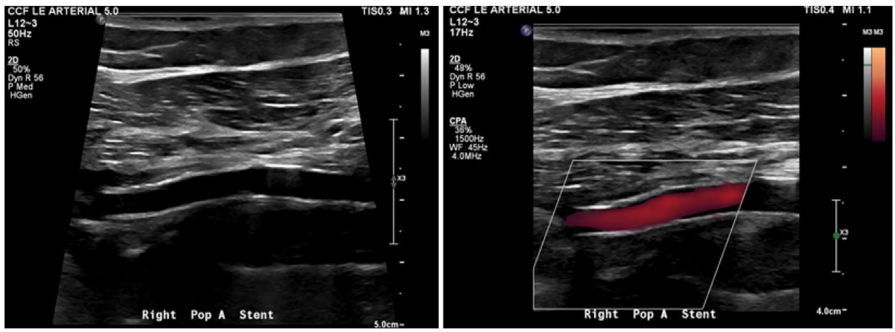

Fig. 5. Gray scale and Duplex ultrasound imaging of patent popliteal artery stent. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

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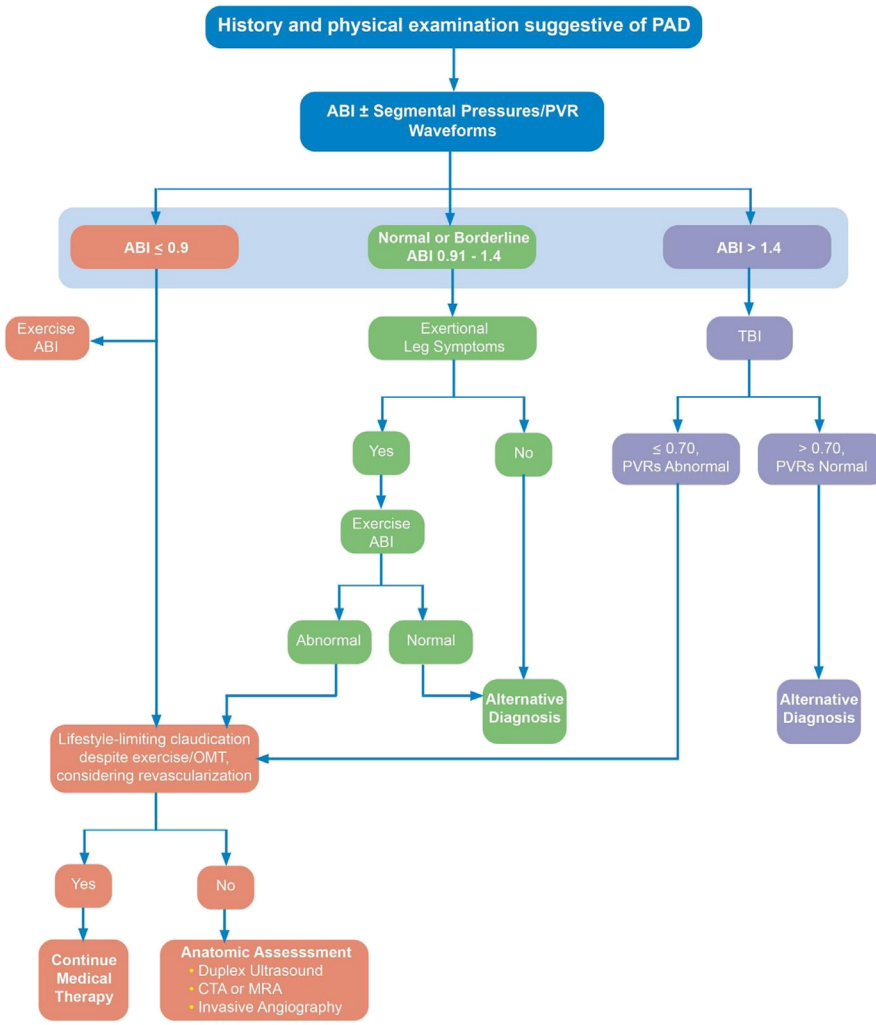

Fig. 6. Diagnostic pathway PAD. ABI, ankle-brachial index; CTA, computed tomography angiography; MRA, magnetic resonance angiography; PAD, peripheral artery disease; PVRs, pulse volume recordings. (*Adapted from* 2016 AHA/ACC Guideline on the Management of Patients with Lower Extremity Peripheral Artery Disease.)

detect severe small vessel disease, fixed occlusive disease, ischemia, and/or vasospasm. PPG waveforms can be normal, reduced, or absent pulsatility. Doppler flow studies with a thermal challenge can be employed to evaluate pathologic response to heat or cold. Thermal provocation with warming is usually preferred to minimize patient discomfort if baseline digital waveforms are abnormal. **Fig. 7** provides guidance on the diagnostic approach for vasospastic disorder.

The purpose of the vascular lab in evaluating the upper extremities to identify, localize and quantify obstruction of the arteries supplying the circulation to the arms and fingers. This may not be offered in all vascular labs. The basic procedural protocol includes placing blood pressure cuffs on the upper or lower extremity and the middle

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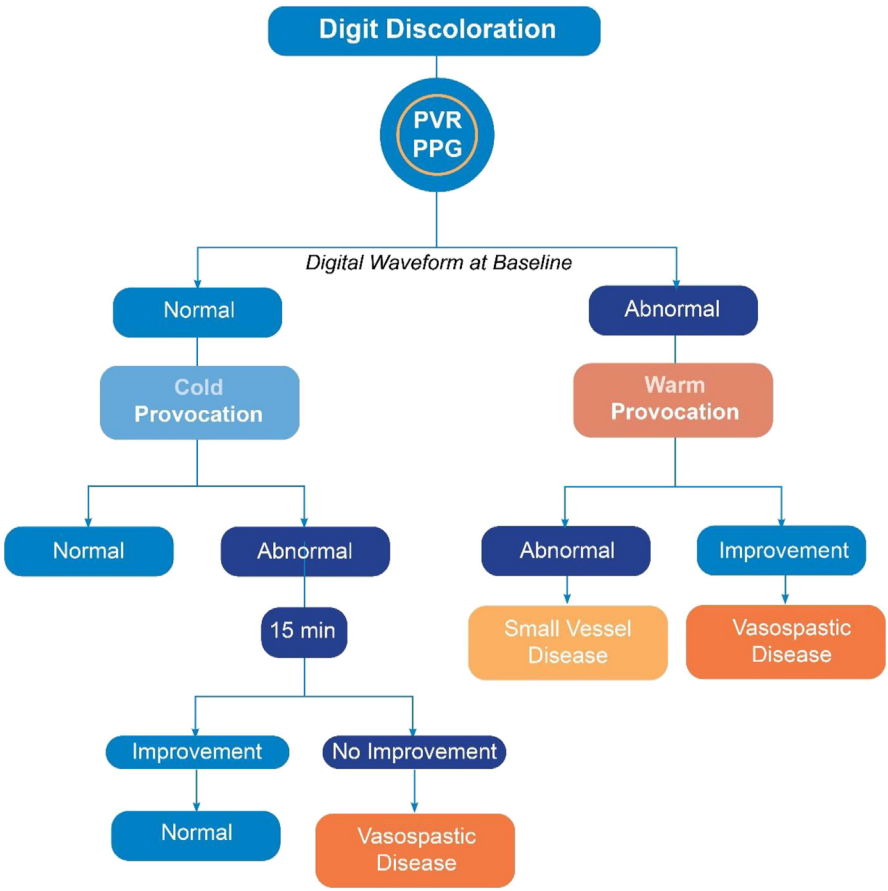

Fig. 7. Approach to digit discoloration.

digit at the base. Digital waveforms can be taken with a PPG sensor taped to the tip of fingers. Doppler waveforms are recorded in bilateral radial and ulnar arteries.[13] **Fig. 8** depicts an abnormal PPG recording with significant improvement after warming provocation.

APPROACH TO THE SWOLLEN LEG

Lower extremity swelling is a common presentation for primary care providers and the emergency department. Identification of the underlying etiology can be challenging but is important, as limb swelling may have a significant impact on the patient. Lower extremity swelling is usually a result of an increase in the interstitial fluid that exceeds the physiologic capacity of lymphatic absorption and drainage.[14] The swelling can be acute or chronic, as well as primary or secondary. A practical diagnostic approach to efficiently identify the causes of swelling includes a complete history and physical examination, and appropriate vascular ultrasound laboratory testing. The basic ultrasound examination for vascular and non-vascular etiologies is low cost and non-invasive. The vascular lab is helpful in evaluating the deep and superficial lower

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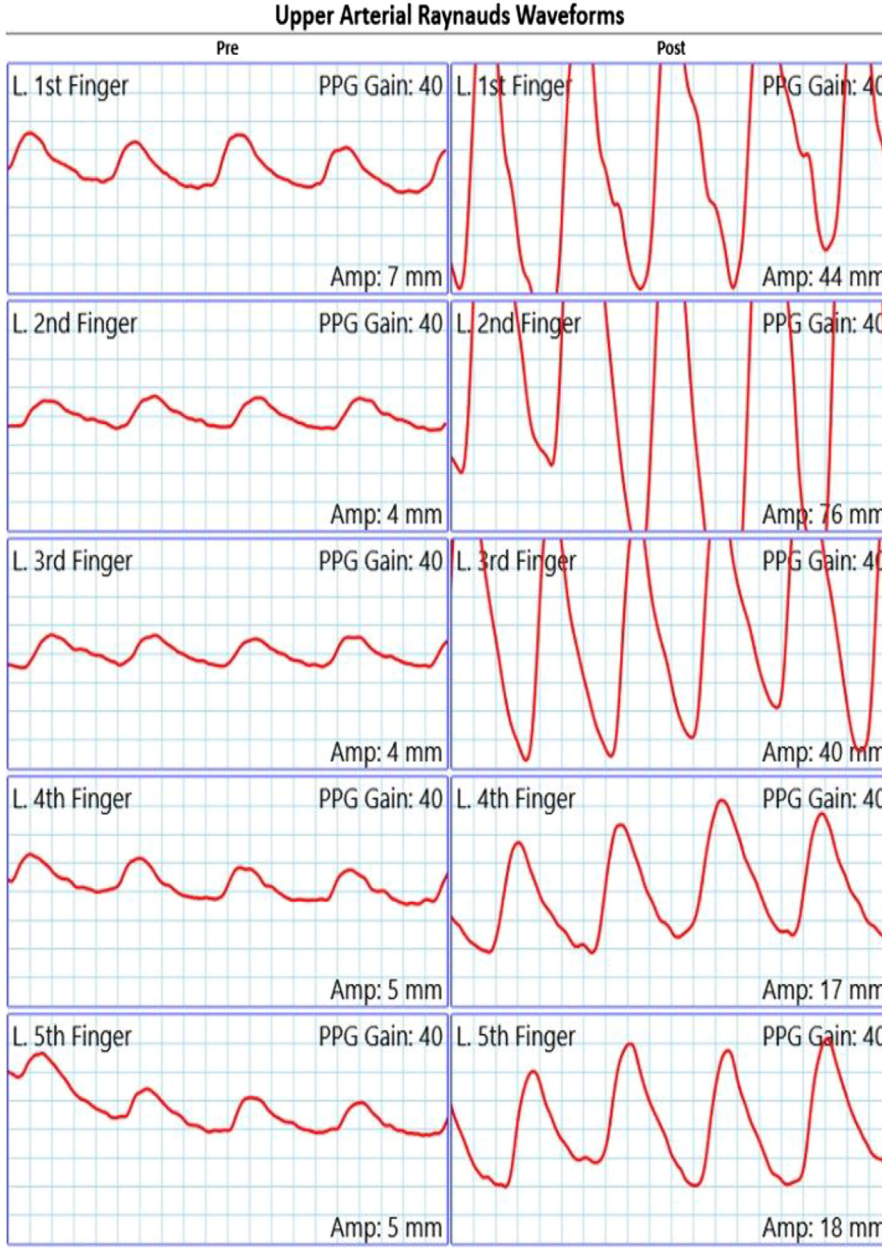

Fig. 8. Photoplethysmogram (PPG) tracing with improvement with warming provocation. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

extremity venous system for the presence of thrombosis, reflux, or even suggestive evidence for more proximal stenosis or high systemic venous pressure by waveform abnormalities. Additional vascular ultrasound images can be obtained, particularly the Inferior Vena Cava (IVC), and common iliac veins, assessing unusual vessel sites

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like the visceral vessels by recording patency of the vessels by color and waveform Doppler. Dedicated venous insufficiency studies allow evaluation for venous incompetency in both deep and superficial veins, along with perforator veins and varicose veins if present. When encountering masses or cysts adjacent to the vessels, more detailed images are acquired, obtaining the size, and describing the consistency, in addition to the presence of vascularity by color Doppler. Documenting the presence of subcutaneous edema is usually not required but possibly mentioned in the final ultrasound report.

DVT protocol duplex venous ultrasound determines the presence or absence of thrombosis in the veins of the legs, identifies the exact location and extent of any identified thrombus and allows qualitative analysis of its characteristics. Acute DVT is characterized by a dilated vein, the presence of intraluminal echoes (usually echo lucent), and thrombus characteristics include spongy, organized, and poorly attached to the vein wall. The inability to coapt the walls of the vessel on compression maneuvers with the ultrasound probe is the most important aspect of diagnosing acute DVT. In contrast with non-acute and chronic DVT, the thrombus appears more echogenic, has a rigid texture, and is more attached to the walls. Other echotexture and acoustic properties include irregular borders, focal venous wall thickening, large collateral veins, normal or atrophic size veins, the presence of reflux, and recanalization of prior DVT. When assessing for thrombus, interrogation of the superficial veins and evaluating for superficial vein thrombophlebitis is an integral part of the imaging study. **Fig. 9** depicts an abnormal compression for the femoral vein indicating a noncompressible vein and a deep vein thrombosis.

When assessing for suspected venous insufficiency, a specific venous duplex test must be ordered, commonly known as a venous insufficiency or venous reflux study.

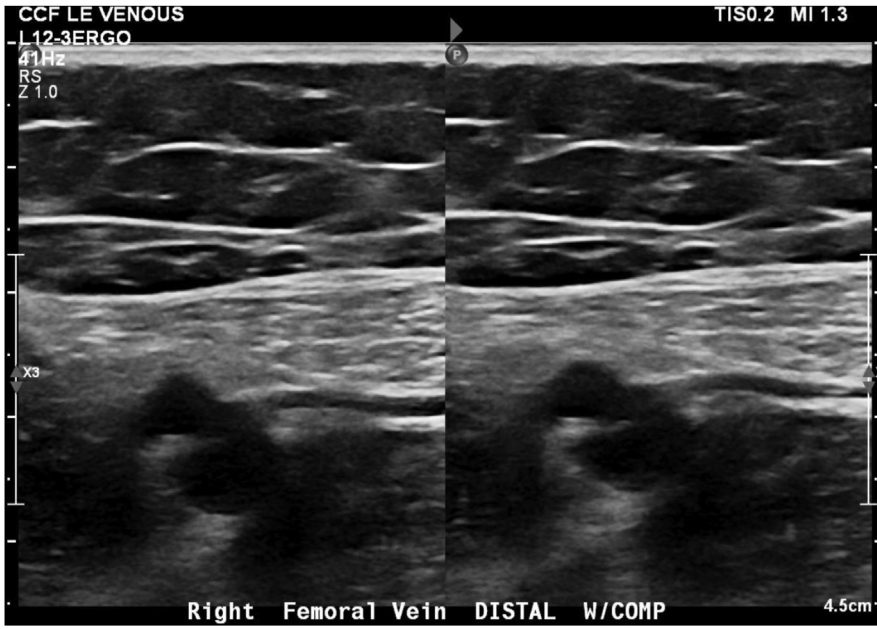

Fig. 9. Abnormal compression of the femoral vein indicating an acute DVT. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

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This duplex imaging of the extremity is performed to assess the deep and superficial venous system for the presence of deep or superficial venous incompetence and to document the location and severity of disease. These are time consuming and labor-intensive studies and should usually be ordered based on specific symptoms of leg heaviness, aching, swelling, throbbing, or itching, also known by the mnemonic HASTI. It is advisable only to interrogate the affected limb; these tests are usually ordered when contemplating any potential role for venous intervention. Positive findings of valvular incompetency are defined as retrograde (reflux) venous flow with Valsalva or augmentation maneuvers with durations of greater than 1 second for deep veins and greater than 0.5 seconds for superficial veins. Negative valvular incompetency demonstrates no retrograde venous flow with Valsalva and augmentation maneuvers or retrograde flow of less than 1 second of deep veins or less than 0.5 seconds of superficial veins. **Fig. 10** depicts a venous insufficiency study of the small saphenous vein with abnormal reflux greater than 0.5 seconds.[15] Air plethysmography (APG) is another tool to evaluate for venous insufficiency. APG is a non-invasive test that measures venous hemodynamics that quantify volume changes in the lower extremity with certain techniques.[16] Although APG has been clinically validated it is not widely used and not available in all vascular laboratories.

APPROACH TO ABDOMINAL AORTIC ANEURYSM AND CAROTID ARTERY STENOSIS

Abdominal aortic aneurysms (AAA) and Carotid artery Stenosis (CAS) are two common conditions primary care physicians will see in their clinical practice. The vascular lab plays an integral part in the care of patients with these vascular disorders, specifically with the appropriate use of aorta and carotid artery duplex ultrasound. We briefly outline screening recommendations, disease monitoring recommendations and the importance of risk factor modification in these patients.

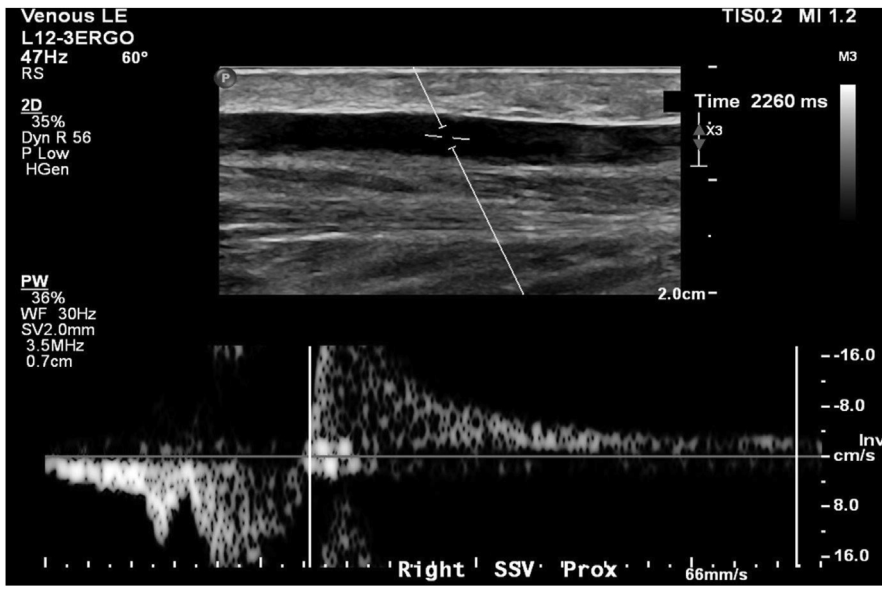

Fig. 10. Venous insufficiency study demonstrating reflux in the small saphenous vein. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

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ABDOMINAL AORTIC ANEURYSMS (AAA)

Screening

Abdominal aortic aneurysms (AAA) are typically defined as aortic enlargement greater or equal to 3 cm in diameter. AAAs are often asymptomatic and are found through screening, incidentally on imaging done for other indications, or at the time of rupture. Since they are often asymptomatic, screening high-risk populations is deemed an evidence-based approach to find AAA earlier in the disease course. In 2019, the United States Preventive Services Task Force (USPSTF) updated their recommendations for AAA screening.[17]

- B recommendation (moderate net benefit) - For men aged 65 to 75 who have ever smoked, it is recommended to perform a one-time screening for AAA with ultrasonography
- C recommendation (small net benefit) – For men aged 65 to 75 who have never smoked, screening for AAA can be recommended selectively based on the patients' medical and family history, risk factors and patients' preferences/values.
- D recommendation (potential harms > benefits) - Do not screen women aged 65 to 75 who have never smoked.
- I recommendation (insufficient evidence) - the evidence for screening in women aged 65 who have smoked is currently insufficient to determine net benefit.

The 2022 American College of Cardiology/American Heart Association (ACC/AHA) Aortic Disease guidelines differ slightly from USPSTF guidelines.[18] The ACC/AHA guidelines recommend AAA ultrasound screening in men > to 65 years of age who have ever smoked and in men or women who are greater than 65 years of age and who are first-degree relatives of patients with AAA (Strength of Recommendation – Strong, Class 1). In addition, they state it is reasonable to consider ultrasound screening in women greater than 65 years of age who have ever smoked. (Moderate, Class 2a recommendation) The guidelines also state that one could consider ultrasound screening in men or women less than age 65 if they have multiple risk factors or a strong family history of AAA. (Weak, Class 2b recommendation) Lastly, the guideline states that there is no benefit to repeating ultrasound screening in men or women greater than 75 years of age who are asymptomatic and have had a negative initial ultrasound screen.

The 2018 Society of Vascular Surgery (SVS) guidelines call for a one-time screening ultrasound in all men and women aged 65 to 75 with the history of tobacco use (Level 1 recommendation, quality of evidence High). (19) In addition, the SVS states that ultrasound screening for AAA in patients who have first degree relatives with an AAA should be considered. (Level 2 recommendation, quality of evidence low).

In summary, The USPSTF, ACC/AHA and SVS all agree that it is appropriate to screen men age greater than 65 who have smoked. The ACC/AHA and SVS guidelines also include women who have smoked and patients with a strong family history of AAA in their screening recommendations.

Abdominal aortic aneurysms are also identified incidentally on imaging done for other indications, whether this be computed tomography (CT) studies, ultrasound studies, or magnetic resonance imaging (MRI) studies. The prevalence rate of incidental AAA has been estimated at 1% to 2%.[19,20] There is some data to suggest that incidentally found AAAs are often not monitored appropriately during follow up.[19] Appropriate protocols should be in place when AAAs are found incidentally to alert the ordering physician or primary care physician about the need for follow up.

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Surveillance

Once discovered, it is important to follow surveillance guidelines to monitor AAA expansion. Repairs are usually recommended when an AAA is greater than 5.5 cm or for aneurysms that have expanded greater than 1 cm in a year. The interval between screening tests is determined by the size of the initial aneurysm. The ACC/AHA guidelines recommend the following monitoring intervals.[18]

Male Patients

- 3 to 3.9 cm in diameter: Ultrasound (US) every 3 years (Strong, Class 1 recommendation)
- 4 to 4.9 cm in diameter: US every 12 months (Strong, Class 1 recommendation)
- Greater than 5.0 cm in diameter: US every 6 months (Strong, Class 1 recommendation)

Female Patients

- 3 to 3.9 cm in diameter: Ultrasound (US) every 3 years (Strong, Class 1 recommendation)
- 4 to 4.4 cm in diameter: US every 12 months (Strong, Class 1 recommendation)
- Greater than 4.5 cm in diameter: US every 6 months (Strong, Class 1 recommendation)

If ultrasound images are suboptimal (eg, secondary to body habitus), then surveillance CT is recommended.

The Society of Vascular Surgery Guidelines recommend the following monitoring intervals for both male and female patients.[21]

- 3.0 to 3.9 cm in diameter – US every 3 years (level of recommendation – Weak; quality of evidence – low)
- 4.0 to 4.9 cm in diameter – US every year (level of recommendation – Weak; quality of evidence – low)
- 5.0 to 5.4 cm in diameter – US every 6 months (level of recommendation – Weak; quality of evidence – low)

The SVS recommends a referral to a vascular surgeon at the time of the initial diagnosis of an aortic aneurysm. They also recommend consideration of an elective repair once the AAA is > 5.5. cm in men and between 5.0 and 5.4 cm in women. (19) **Fig. 11** depicts an infrarenal abdominal aortic aneurysm measuring 5.04 cm in the transverse view.

Risk Factor Modification

Risk factors for abdominal aortic aneurysm are similar in scope to risk factors for coronary artery disease. Strong risk factors include smoking history, older age, male sex, and a positive family history of AAA. (18) Additional risk factors include hypertension, hyperlipidemia, white race, inherited vascular connective tissue disorder and atherosclerotic cardiovascular disease.[18] Although outcome data is limited regarding medical management/risk factor modification on AAA disease progression, the primary care physician should focus on helping the patient control modifiable risk factors (such as smoking, hypertension, and hyperlipidemia), which will also improve overall cardiovascular health. The ACC/AHA guidelines recommend antihypertensive medication in patients with AAA who have an average systolic blood pressure greater than 130 mm Hg or an average diastolic blood pressure greater than 80 mm Hg. (Strong, Class 1 recommendation).[18] The SVS guidelines does not recommend administering beta-blocker therapy for the sole purpose of reducing the risk of AAA expansion and rupture (Level

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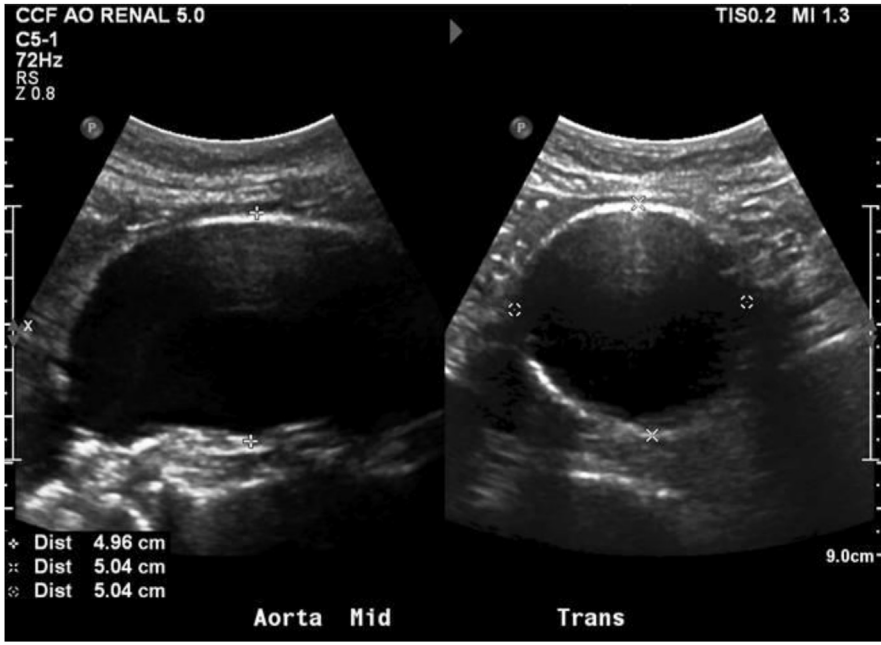

Fig. 11. Abdominal Aortic Aneurysm measuring 5.04 cm.in transverse diameter. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

of recommendation – Strong, Quality of evidence – moderate).[21] In patients with AAA who have evidence of aortic atherosclerosis, the ACC/AHA guidelines recommend moderate to high intensity statin therapy. (Strong, Class 1 recommendation).[18] Smoking sensation is recommended in all patients with AAA.

APPROACH TO CAROTID ARTERY STENOSIS (CAS)

Screening

The USPSTF and SVS guidelines do not recommend screening for asymptomatic carotid stenosis in the general population.[22,23]

- USPSTF – Recommends against screening for asymptomatic carotid stenosis in the general population. (D recommendation)
- SVS – recommends against screening in asymptomatic patients without symptoms or significant risk factors for carotid disease (Grade 1(strong); quality of evidence: B (moderate))

The SVS guidelines state that screening in asymptomatic patients at increased risk for carotid stenosis should be considered, particularly in patients willing to consider intervention if significant stenosis is found. (Grade 2(weak); quality of evidence: B (moderate)).[23] This high-risk group includes patients with peripheral artery disease, coronary artery disease, previous radiotherapy to the neck and patients with evidence of previous cerebral infraction on brain imaging. The presence of a carotid bruit on physical exam is a common indication for testing. However, the presence of severe carotid disease in patients with a bruit and no other risk factors is only 2%.[22–26] The recommended screening test is a carotid artery duplex ultrasound performed in an accredited vascular lab. **Fig. 12** depicts right internal carotid artery stenosis.

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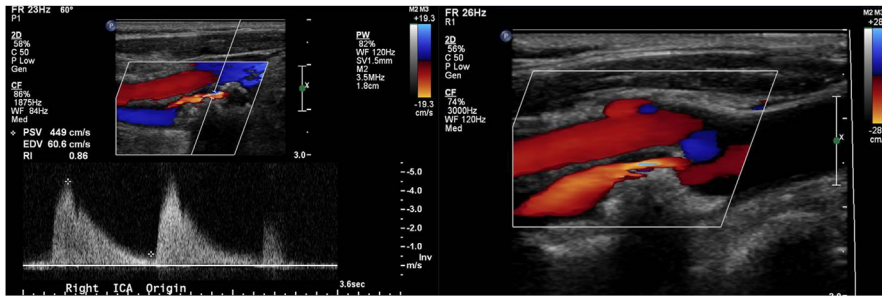

Fig. 12. Carotid Artery Stenosis right internal carotid artery with elevated peak systolic velocities. (Images courtesy of Cleveland Clinic Foundation Vascular Lab.)

Surveillance

Once carotid artery stenosis is diagnosed, a common question arises about the frequency of follow-up ultrasound examinations to document disease progression. Strong evidence-based guidelines to answer this question are lacking and recommendations are based on expert opinion. Patients found to have moderate stenosis (50% to 79%) should be followed up initially at 6-month intervals to detect disease progression that may require intervention.[27] Patients with less than 50% carotid stenosis can be followed initially annually.[27] Patients with severe stenosis (>79%) should be referred to a vascular specialist for further evaluation and management.

Risk Factor Modification

All patients diagnosed with carotid artery stenosis should be evaluated and treated with risk factor modification. Medical therapy has narrowed the gap between the medical and surgical treatment of carotid artery disease in reducing the risk of stroke. In patients with carotid artery stenosis, the annual risk of ipsilateral stroke is estimated to be 0.5%.[28] Moderate carotid artery stenosis is often more closely associated with myocardial infarction than stroke.[29] The core components of risk factor modification include lifestyle modification (eg, diet, exercise), blood pressure control, intensive lipid lowering, Diabetic control, B vitamin supplementation in patients with elevated homocysteine levels and smoking cessation.[28,30,31]

CLINICS CARE POINTS

- The best screening tool for peripheral artery disease (PAD) is the ankle brachial index (ABI).
- The PVR (pulse volume recording with segmental pressures) study is another first line study in the suspected PAD patient. Lower arterial duplex examinations are usually reserved for anatomical identification of known disease and surveillance of arterial interventions.
- If the ABI is normal and suspicion for PAD is still present, an exercise ABI can be helpful to unmask PAD.
- PAD is not just a leg disease, but a brain and heart disease as well given the association with stroke and MI.
- The mnemonic for venous insufficiency symptoms is HASTI (heaviness, aching, swelling, throbbing, and itching).
- Venous duplex ultrasound available for the legs include DVT protocol and a more complete venous insufficiency study.

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- Surveillance regimens for AAA and carotid artery disease are important strategies in patients with known vascular pathology to document stability or progression.
- Patient history, examination, as well as the vascular lab can be helpful in diagnosis thermally provoked vasospastic disease such as Raynaud's.

DISCLOSURE

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