How best to approach a patient with peripheral arterial disease (PAD) can be an intimidating and confusing task. There are few disease processes as variable in location, presentation, and severity as those seen in the vasculopathy. There are many etiologies to be considered simultaneously. Ironically, it is the variability of PAD that provides an opportunity to extract a comprehensive and tightly integrated history and physical examination that is nearly unparalleled in medicine. Similarly, there are few disease processes that offer the opportunity to impact so many aspects of the patient, literally from head to toe. This article, part of a series on PAD, focuses on the initial assessment and evaluation of the PAD, with pathophysiology, genetics, and treatment addressed in accompanying articles.

History

Each patient with PAD is unique (Table 1). Even though the pathophysiology, risk factors, location, and eventual treatment options for a patient often prove routine, the manner in which the patient presents is anything but predictable. We have all had the experience of taking a well-structured history (Table 2) from a textbook patient, presented to an attending physician or a colleague, and then watched and listened in disbelief as the confirmatory history is nothing at all similar to what was heard 15 minutes earlier. Unfortunately, patients do not often read the textbook. As demonstrated by McDermott and colleagues, the discomfort caused by PAD is more often atypical than typical. Descriptions such as “tired,” “giving way,” “sore,” and “hurts” are offered more often than “cramp.” This forces the healthcare provider to meticulously clarify the location, quality, and circumstances of the discomfort. It is not unusual that a patient reports ≥2 types and locations of discomfort. Careful consideration for pain from both orthopedic and neurogenic sources must be taken. Each symptom should be individually addressed and clarified. These symptoms must be taken in context with the lifestyle of each patient. Defining how the symptoms have an impact on vocation, activities of daily living, and social activities should be done with each evaluation. Comorbidities and risk factors for PAD are discussed elsewhere in this series and should be elicited from the patient when not already clear. The physical examination should focus on the clues gleaned from the history. Specific scenarios are discussed at the end of this article.

Claudication

The most common presentation of lower extremity PAD is claudication, from the French and Latin words for limping. The classic description of claudication is a cramp in a muscle group causing an alteration in gait that occurs at a reproducible distance when walking on a level surface and is relieved quickly and consistently by rest. The distal extremity usually becomes symptomatic before the proximal extremity does. The calves are affected more often than the thigh or buttock. In general, the location of the symptom is 1 joint below the flow-limiting stenosis. As noted above, on a flat, level surface, the distance and time to limiting symptoms is reproducible. Carrying a load, uneven ground, or an uphill grade shortens the distance to claudication owing to an increased workload. Conversely, slowing the walking pace decreases workload and increases the time, if not the distance, to the onset of symptoms. Stopping, standing upright, without the need to sit, bend, or lean, is consistent with arterial claudication. The need to sit or lean on an object, thus flexing the spine to obtain relief, suggests an orthopedic cause, such as spinal stenosis, but is not specific. In contrast, leaning on a shopping cart may flex the spine, decrease the workload of walking, off-load a joint, or any combination of thereof, and is therefore not specific.

Unconscious, slow adaptation by the patient in their daily life occurs often, resulting in asymptomatic PAD even though severe on objective testing. Simple questions like “What is the most strenuous thing you do in a typical week?” or “Did you have pain or need to stop coming in from the parking lot for your appointment?” can help clarify the functional status of the patient. Patients often note the onset of symptoms during a vacation or a yearly activity. This forces them out of their usual routine and thereby causes symptoms that the patient may relate as acute.

Physical Examination

The vascular examination should focus on the symptoms presented by the patient during the history. However, a complete vascular examination is appropriate given the diffuse nature of the atherosclerotic process (Table 3). Vital signs including bilateral blood pressures, heart rate, height, and weight should be obtained as a routine. Systolic blood pressures should be within 10 to 15 mm Hg, and, when greater, formal simultaneous pressures should be obtained. Cardiac and pulmonary examinations should be performed to assess cardiac manifestations of
Table 1. Differential Diagnosis of Peripheral Arterial Disease

<table>
<thead>
<tr>
<th>Arterial Occlusive Disease</th>
<th>Nonvascular Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerosis</td>
<td>Orthopedic pain</td>
</tr>
<tr>
<td>Fibromuscular dysplasia</td>
<td>Degenerative joint disease</td>
</tr>
<tr>
<td>Thromboangiitis obliterans</td>
<td>Bursitis</td>
</tr>
<tr>
<td>(Buerger disease)</td>
<td></td>
</tr>
<tr>
<td>Vasculitis</td>
<td>Arthritis</td>
</tr>
<tr>
<td>Takayasu disease</td>
<td>Baker cyst</td>
</tr>
<tr>
<td>Giant cell arteritis</td>
<td>Fracture bony injury</td>
</tr>
<tr>
<td>Atheroembolism</td>
<td>Spinal stenosis</td>
</tr>
<tr>
<td>Peripheral embolism</td>
<td></td>
</tr>
<tr>
<td>Aortic/iliac aneurysm</td>
<td>Nerve root impingement</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Other</td>
</tr>
<tr>
<td>Popliteal aneurysm</td>
<td></td>
</tr>
<tr>
<td>Popliteal entrapment syndrome</td>
<td>Compartment syndrome</td>
</tr>
<tr>
<td>Thrombosis in situ</td>
<td>Myopathy</td>
</tr>
<tr>
<td>Radiation arteritis</td>
<td>Peripheral neuropathy</td>
</tr>
<tr>
<td>Trauma</td>
<td>Venous claudication</td>
</tr>
<tr>
<td>External impingement (mass effect)</td>
<td>Poststroke syndrome</td>
</tr>
<tr>
<td>Pseudoxanthoma elasticum</td>
<td></td>
</tr>
<tr>
<td>Endofibrosis of the iliac artery</td>
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</tbody>
</table>

atherosclerosis, arrhythmias responsible for thromboembolism, and the risks of potential invasive or surgical therapy.

Inspection: Skin Findings

Dependent rubor and elevation pallor are often seen in severe PAD. Patients with suspected moderate to severe PAD should have their feet elevated by the examiner, and the time to onset of pallor, when present, should be recorded. In the setting of a microatheroembolic event, livedo may be seen at the plantar surface weeks to months after the event (Figure 1). The patient is then returned to the seated position with venous refilling time recorded. Venous refilling time is not accurate in the presence of severe varicose veins. The presence or absence of hair is not a sensitive or specific indicator of PAD, but when asymmetric and associated with other findings, is strongly suggestive.

Tissue loss most commonly presents as a wound or ulcer at the foot, but may also be noted with cracking or fissures in the heel. In PAD, wounds are most often distal, at the toes or metatarsals, and are dry in appearance. They tend to occur over pressure points and often begin after an event that required more walking than normal or a change from their usual footwear. Similarly, an innocuous injury, such as a bump by a dishwasher door, may lead to a nonhealing wound even though other symptoms are unchanged or absent.

The toes and the area between the toes should be examined routinely in patients with PAD for irritation or ulcers. Kissing toe ulcers (between the toes), or pressure ulcers over the surfaces of the lateral and medial metatarsal heads, are common after the purchase of new footwear. This is true regardless of socioeconomic status and especially in those with diabetes mellitus. Patients with diabetes mellitus have the additional problem of neuropathy, resulting in not only diminished sensation, but also the loss of normal nervous input to the musculature. Swan-necking of the toes may be seen, which leads to irritation and ulceration at the toe tips and at the dorsal surface of the toe(s) even when shoes are well fit for length. A deep toe box is required to avoid this potentially limb-threatening situation.

Palpation

Palpation of arteries is performed routinely for all vascular patients. American Heart Association/American College of Cardiology guidelines recommend grading on a scale of 0 to 3, with 0, absent; 1, reduced; 2, normal; and 3, bounding. \(^1\) Trans-Atlantic Inter-Society Consensus (TASCII) suggested slightly different scale, and many institutions have a scale that is traditionally used. \(^4\) It is important to document the scale used to examine the patient so that a provider from another institution understands the nomenclature. In the lower extremity, the femoral, popliteal, posterior tibial, and dorsalis pedis arteries should be palpated. The popliteal pulse may be difficult to isolate and should be examined with the patient both seated and supine. Absence of the ankle pulses and a femoral bruit has been shown to be sensitive for PAD. \(^5\) The aorta should be palpated and size estimated. In the obese patient, the aorta should not be easily palpable. The iliac arteries should not be palpable in any type of body habitus and when they are, they should be considered aneurysmal and appropriate imaging should be arranged. \(^6\)

Examination of the upper extremity should include simultaneous palpation of the contralateral radial and ulnar arteries to detect any volume difference or delay in the pulse. In the young patient with a pulseless extremity or in the older patient with symptoms of giant cell arteritis, a very thorough examination of the upper extremities, head, and neck should be performed. The carotid artery should be examined in all patients with PAD, and, when indicated, the subclavian artery should be examined as well.

Palpation should include feeling for temperature differences between limbs and at multiple levels of each limb. A definite level of cold is often present in critical limb ischemia. Atrophy of the calf or at the fat pad of the heel may also occur.
Simultaneous palpation of these areas can help the examiner determine whether early, unilateral tissue loss is present before the presence of a wound.

**Auscultation**

Auscultation of the vessels should be performed in all patients presenting with PAD. It is often helpful to auscultate vessels while palpating a radial artery. This allows better discrimination of subtle bruits from ambient noise, such as bowel sounds, and the determination of whether the bruit extends into diastole, which suggests a higher degree of stenosis than a bruit that is present in systole only. The carotid artery is routinely auscultated. A previously unheard or undefined carotid bruit(s) may be further investigated. A femoral bruit may be tracked proximally to the pelvis (overlying the iliac arteries) and to the aorta (in general, the aortic bifurcation is at the level of the umbilicus) to help determine the site of stenosis. When an aortic bruit is present, further differentiation is required. A bruit that changes with respiration suggests dynamic celiac artery compression by the arcuate ligament. An abdominal bruit that lateralizes to the left or to the right suggests, but is not diagnostic for, renal artery stenosis.7

**Testing for PAD**

The type of testing will depend on the question to be answered. In general, the ankle brachial index (ABI) can establish the diagnosis and severity of PAD in the office setting. The ABI is the most appropriate test for screening as well. Duplex ultrasound can further define the location and degree of stenosis in the vast majority of patients. For those with symptoms during walking, but normal ABI, standardized treadmill testing is appropriate. Further imaging is appropriate in patients with critical limb ischemia (ie, tissue loss or ischemic rest pain) and those with lifestyle or employment limiting claudication that require more detailed imaging for planning revascularization. Tests of walking function are not only used for clinical diagnosis, but can also help assess response to treatment in research studies8,9 and the clinical setting.10

**Office-Based Testing**

**The Ankle Brachial Index**

The ABI is just 1 possible segmental pressure that may be measured. Segmental pressures are the arterial closing and opening pressure at a specific anatomic location, such as the

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Table 3. The Vascular Examination by Location and Method

<table>
<thead>
<tr>
<th>Location</th>
<th>Inspection</th>
<th>Palpation</th>
<th>Auscultation</th>
<th>Maneuvers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td>Arcus senilis telangiectasias</td>
<td>Temporal arteries</td>
<td>Carotids</td>
<td>Thoracic outlet</td>
</tr>
<tr>
<td>Neck</td>
<td>Venous distention</td>
<td>Carotids</td>
<td>Subclavian</td>
<td>As indicated</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Jugular venous pressure</td>
<td>Cardiac</td>
<td>Cardiac</td>
<td></td>
</tr>
<tr>
<td>Chest</td>
<td>Venous plethora or collaterals</td>
<td>Percussion</td>
<td>Pulmonary</td>
<td></td>
</tr>
<tr>
<td>Abdomen</td>
<td>Distension</td>
<td>Aorta</td>
<td>Lateralization or respiratory</td>
<td>Respiratory maneuvers</td>
</tr>
<tr>
<td></td>
<td>Venous plethora</td>
<td>Liver span</td>
<td>variation if bruit</td>
<td></td>
</tr>
<tr>
<td>Pelvis</td>
<td>Distension, scars</td>
<td>Iliac</td>
<td>Lateralization or respiratory</td>
<td>Hip range of motion</td>
</tr>
<tr>
<td></td>
<td>Venous plethora</td>
<td>Trochanteric bursa</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leg</td>
<td>Asymmetric hair growth</td>
<td>Femoral, popliteal,</td>
<td>Femoral artery</td>
<td>Elevation pallor</td>
</tr>
<tr>
<td></td>
<td>Varicosities</td>
<td>Size discrepancy</td>
<td></td>
<td>Dependent rubor</td>
</tr>
<tr>
<td>Foot</td>
<td>Wounds</td>
<td>Knee crepitus or</td>
<td></td>
<td>Sensory examination</td>
</tr>
<tr>
<td></td>
<td>Varicosities</td>
<td>mass</td>
<td></td>
<td>Separate toes to inspect</td>
</tr>
<tr>
<td></td>
<td>Tinea pedis, cracking</td>
<td>Dorsalis pedis and</td>
<td></td>
<td>Capillary refill</td>
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<tr>
<td></td>
<td>Telangiectasias</td>
<td>posterior tibial</td>
<td></td>
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<tr>
<td></td>
<td>Venous stasis</td>
<td>arteries</td>
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<td></td>
<td>Cellulitis</td>
<td>Edema</td>
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<td></td>
<td>Livedo</td>
<td>Thickness</td>
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<tr>
<td>Upper</td>
<td>Venous plethora</td>
<td>Brachial</td>
<td></td>
<td>Allen test</td>
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<tr>
<td>extremity</td>
<td>Telangiectasias</td>
<td>Radial, ulnar</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Figure 1. Examples of elevation pallor (A), dependent rubor (B), and thromboembolism (C) of the left foot.
brachial, thigh, calf, or ankle level. A drop of 15 to 20 mm Hg between segments indicates a pressure-limiting stenosis between levels. Because the upper extremities are often spared by atherosclerosis, the segmental pressure can be normalized to the brachial pressure creating an index by dividing by the greater of the 2 brachial pressures. In the case of the ABI, there are 2 arteries at the ankle to be considered, the posterior tibial and the dorsalis pedis. The greater of the 2 is divided by the greater arm pressure providing the ABI. The ABI at rest is both sensitive and specific for PAD, and, as demonstrated in multiple population studies, it is a good marker for cardiovascular morbidity and mortality. The ABI is an appropriate screening test in patients >65 years of age who have suspected PAD, or those >50 with a history of tobacco use or diabetes mellitus.\textsuperscript{13}

The Coalition on Ankle Brachial indices proposed guidelines for the interpretation of ABI that were adopted by the American College of Cardiology/American Heart Association Vascular Diseases writing group (Table 4).\textsuperscript{12} Although based primarily on epidemiological rather than physiological evidence, these guidelines are a useful and appropriate standard. An ABI of <0.9 is abnormal and indicates PAD. A normal ABI is defined as 1.0 to 1.4. Until recently, patients with an ABI of 0.90 to 0.99 had been considered normal. This range is now considered borderline PAD, because evidence shows an increased risk of cardiovascular morbidity and mortality in this group. The ABI is not reliable in the setting of calcified vessels that become either non- or poorly compressible, most commonly seen in patients with diabetes mellitus or renal failure. In these patients, the ABI is >1.4, and, in epidemiological studies, the ABI is associated with increased cardiovascular morbidity and mortality in comparison with normal ABI. The toe brachial index is usually a more reliable indicator of obstructive PAD in this setting.\textsuperscript{13} The toe brachial index is performed by placing a small occlusion cuff on the great toe with a photoplethysmography sensor on the tip of the toe. Limitations include the absence of the toe or the presence of a wound. Other alternatives to the ABI in the setting of noncompressible vessels include the continuous-wave Doppler signal, pulse volume recording (PVR), perfusion pressure testing, and duplex ultrasound. Recently, a statement on how to obtain accurate and reproducible ABIs was published that gives preference to manual pressure readings with continuous-wave Doppler signals over automated devices.\textsuperscript{12}

Pulse Volume Recording

PVR measures the volume and flow pattern into a limb. A cuff is inflated to a known pressure, usually 60 mm Hg. Arterial inflow displaces the air, generating an increase in pressure that is recorded over time. A normal PVR has a rapid upstroke to a peak in systole, with a dicrotic notch and subsequent descent during diastole (Figure 2). When arterial stenosis is present, the upstroke becomes slurred, the peak becomes flattened, and the dicrotic notch is lost. In severe stenosis or occlusion, the waveform is sinusoidal or even lost. One advantage to the PVR is that, unlike the ABI, it is independent of arterial calcification.\textsuperscript{14}

Continuous-Wave Doppler

Continuous-wave Doppler is most often used as part of the ABI test. The probe is placed over the artery to be measured, and the loss of and return of flow in the vessel is recorded as the signal is lost and returns, respectively. Unlike duplex ultrasound, the continuous-wave Doppler probe sends and receives constantly. There is no depth selection possible, and, therefore, a true velocity within the vessel cannot be obtained. Nonetheless, the same qualitative analysis of a normal triphasic signal can be heard and captured graphically if instrumented to do so. This allows approximation of the level of disease, similar to PVR. A normal continuous-wave Doppler is triphasic, with a rapid upstroke or forward flow, a downstroke to below baseline as flow reverses, and finally a short period of forward flow is seen again. When stenosis is present and as it increases, the reversal of flow is lost and the upstroke may be delayed. With greater stenosis, the upstroke becomes smaller and further delayed (parvus et tardus), sinusoidal, and eventually absent (Figure 3). Continuous-wave Doppler devices are readily available and portable – easily carried in a coat pocket. Because both arterial and venous Doppler signals may be obtained with the continuous-wave Doppler, it is a powerful bedside tool for the physician.\textsuperscript{15}

Vascular Laboratory Testing

Exercise Testing

Although the ABI at rest has been shown to be an excellent screening tool to assess the risk of cardiovascular morbidity and mortality for patients with claudication, a normal resting ABI does not exclude the presence of PAD. Stein et al\textsuperscript{16} demonstrated that nearly one-third of patients with claudication symptoms who had normal PVRs and a normal ABI at rest had a positive treadmill exercise test (ABI <0.90 postexercise). The exercise ABI test is performed after segmental pressures at rest have been obtained. There is no agreed upon protocol for lower extremity arterial exercise testing. Both fixed and variable speeds and degrees of incline are used. Plantar pedal flexion in the office setting and chemically induced stress have been used as well.\textsuperscript{17,18} Although not mandatory, ECG monitoring during exercise is often done. Regardless of the protocol used, the patient is exercised until the onset of symptoms, often to maximal tolerated symptoms or until the protocol is completed. Following exercise, the patient

| Table 4. ABI Guidelines (AHA/ACC 2011)\textsuperscript{11} and the TBI |
|------------------|------------------|------------------|------------------|
| ABI             | Grade           |                  |
| >1.40           | Noncompressible |                  |
| 1.0–1.39        | Normal          |                  |
| 0.90–0.99       | Borderline      |                  |
| <0.90           | Abnormal        |                  |
| TBI             |                  |                  |
| >0.70           | Normal          |                  |
| <0.35 or < 30 mm Hg | Severe         |                  |

\textsuperscript{11} ABI = higher ankle pressure/higher arm pressure at each leg, ABI indicates ankle brachial index; ACC, American College of Cardiology; AHA, American Heart Association; and TBI, toe brachial index.
quickly lies down and pressures at the higher arm and both ankles are taken within 1 minute. If normal at rest, an abnormal Doppler signal at the common femoral artery postexercise indicates that proximal PAD is present. The time and distance to the onset of symptoms with standardized testing is more reliable than the patient’s estimate of walking ability. This makes

Figure 2. A, Pulse volume recordings in the upper extremity. The right arm is normal. The left arm is abnormal with a slurred and diminished upstroke, loss of dicrotic notch, and a sinusoidal appearance. Note the segmental pressures at the brachial, radial, and ulnar location are also presented and lower on the left versus the right. B, Pulse volume recording of the lower extremities with segmental pressures. Note the progressive loss of amplitude, slurred upstroke, and minimal deflection at the metatarsal level on the left lower extremity. The dicrotic notch is absent because of tachycardia. BP indicates blood pressure; DP, dorsal pedis; L, left; PT, posterior tibial; PVR, pulse volume recording; R, right; UA, upper arm; and VPR, volume-pressure relationship.
exercise testing an excellent way to follow both known PAD and those who have undergone revascularization.

**Perfusion Testing**

Tissue perfusion testing can be performed by several methods. Transcutaneous oximetry assesses the partial pressure of oxygen in mmHg (TcpO₂). It is most commonly used in wound care centers or as part of the evaluation for or during hyperbaric oxygen treatment. The study is performed with the patient supine for baseline values. The likelihood of wound healing is good when the TcpO₂ is >40 mmHg, and unlikely when <20 mmHg. An increase in TcpO₂ in response to high-flow oxygen is used when evaluating for hyperbaric oxygen therapy. Interpretation of a TcpO₂ study is challenging when the patient has an acute condition that alters microvascular physiology such as edema, acute local infection, and vasoconstriction during sepsis, which will all lower the TcpO₂ values. In contrast, cellulitis with normal vasculature and supplemental oxygen therapy may increase the TcpO₂ values. Recent exercise TcpO₂ has been shown to be reproducible for the assessment of regional blood flow impairment induced by exertion. Near infrared spectrometry and laser Doppler flux are other emerging, although not widely used, tools for the assessment of deep tissue and skin perfusion, respectively.

**Vascular Imaging**

Direct imaging of the arterial system is an integral part of the evaluation of PAD for many patients and is required to accurately plan how best to approach those who are candidates for revascularization. Catheter-based contrast angiography remains the gold standard for vascular imaging. The resolution and applicability to all vascular beds has not yet been matched by other modalities. Unlike catheter-based angiography, both computerized tomography angiography (CTA) and magnetic resonance angiography (MRA) obtain imaging of the surrounding structure including the vascular wall. This is helpful for evaluation in patients with aortitis, aneurysms with laminated thrombosis, and retroperitoneal fibrosis. CTA and MRA both offer postimage acquisition processing that allows the creation of 3-dimensional images. Duplex ultrasound is most often available in the office setting and is discussed first.

**Arterial Duplex Ultrasound**

Duplex ultrasound is the most common testing modality used in vascular laboratories. Duplex ultrasound incorporates 2-dimensional gray-scale ultrasound to provide an image of arterial structures and pulse-wave Doppler to sample velocities of blood flow at a specific location within the artery lumen. It spans the gap between static imaging and hemodynamic evaluation. The reliability, portability, and range of studies that can be performed with duplex ultrasound make it indispensable for arterial evaluation. Unlike continuous-wave Doppler and PVR, anatomic location of the stenosis is possible because direct imaging of the vessel is performed. An estimate of stenosis is possible by the duplex image, the
Doppler waveform, and the velocity changes within the vessel. Changes in the Doppler waveform are similar to those seen in continuous-wave Doppler, but the velocity is able to be estimated because the source of the signal sampling is known. Turbulent flow results in spectral broadening of the Doppler signal as well. A parvus et tardus waveform with marked diastolic flow is seen in severe stenosis (Figure 4). The ability to localize and detect changes makes duplex ultrasound an excellent tool for following the site of endovascular or surgical procedures. Duplex arteriography may be used in place of contrast angiography when contraindications are present. However, it is not an appropriate screening tool and difficult, at best, to use in conjunction with exercise. It is not within the scope of this article to include a complete review of vascular duplex testing, but many excellent texts are available.

**Computerized Tomography Angiography**

With the development of multislice computed tomography scanners that increased the resolution of the arterial system, CTA has become a first-line study for PAD (Figure 5). Arterial calcification is well seen with this technique, allowing for accurate planning of procedures. However, dense calcification and arterial stents, and other implanted objects, as well, such as hip and knee replacement, interfere with the signal of the arterial system owing to the dense artifact. Furthermore, intravenous contrast and radiation exposure are present. Young patients who will require repeated imaging, those with previous high levels of radiation exposure, and women who are pregnant or at risk of pregnancy must be carefully considered before a CTA.

**Magnetic Resonance Angiography**

Similar to the improvement in CTA, MRA techniques have improved markedly in the past decade (Figure 6). Study protocols with more rapid sequencing and efficiency of cardiac gating have allowed greater spatial and temporal resolution to be obtained. MRA has proven very useful in vasculitides, such as Takayasu arteritis, for evaluating stenosis attributable to wall thickening, and it avoids ionizing radiation in this young patient group. MRA may also be timed for venous phase imaging at the same setting as the arterial imaging. However, MRA is not compatible with most implantable devices such as pumps, defibrillators, or pacemakers. MRA is not suitable for imaging the patency of metallic stents, because there is a metal artifact void that obscures the stent and lumen. Furthermore, in patients with renal failure at risk for nephrogenic systemic fibrosis owing to gadolinium administration are contraindicated for MRA. A number of noncontrast protocols for MRA have been reported, but their availability is not yet universal.

**Catheter-Based Angiography**

Although it is used much less than in the past, catheter-based angiography remains the gold standard of vascular imaging. Neither CTA nor MRA have yet reached the resolution of conventional angiography. This fine level of detail may be required in the setting of vasculitis, Buerger disease, or an arteriopathy related to a connective tissue disorder. Diagnostic maneuvers such as vasodilator administration and popliteal or thoracic outlet maneuvers can also be done in real time. Unlike CTA and MRA, catheter-based angiography may be planned with a limited scope, reducing contrast load, and in patients at risk of renal complications. Intravascular ultrasound may be used to limit contrast or better define the disease process when unclear on imaging alone. Perhaps the main advantage of invasive angiography over all other imaging techniques is the option to convert to an intervention at the same setting. This is especially true in acute limb ischemia.
Contrast-induced nephropathy (CIN) and nephrogenic systemic fibrosis are the Achilles heel of vascular imaging. CIN is an acute renal failure induced by iodinated contrast that is usually reversible. An age of >75 years, hypotension, class 3 or 4 congestive heart failure, diabetes mellitus, administration of contrast >100 mL, and a glomerular filtration rate <30 are all risks for the development of CIN. It is important to remember that the estimation of glomerular filtration rate is only accurate in patients with a stable creatinine. A patient with an acute elevation in creatinine should be retested before proceeding with contrast administration. There are a number of pre- and post-exposure protocols to reduce the risk of CIN that have been evaluated, but no clear strategy has proven most effective.

Nephrogenic systemic fibrosis is rare, but the risk factors are similar to those of CIN. Gadolinium is poorly soluble and renally excreted. When not cleared adequately, it can be deposited in tissues, and a fibrotic process may occur. Patients with a glomerular filtration rate <30 are at risk for nephrogenic systemic fibrosis. There are data suggesting that same-day hemodialysis following the administration of gadolinium may reduce the risk of nephrogenic systemic fibrosis. However, this is not an encouraged practice and should be used only when no other alternatives are possible.

Clinical Scenarios

Pseudoclaudication

Pseudoclaudication is most often attributable to lumbar spinal stenosis or nerve root impingement. Symptoms mimic those of an arterial etiology with the exception that discomfort often, but not always, starts proximally and progresses distally. In contrast to arterial claudication, symptoms may occur when simply standing, such as waiting in line, shaving, cooking, or brushing one’s hair. Symptom relief requires sitting or bending to open the canal or relieve the nerve root impingement. When a bench or chair is not available, patients often lean against a wall, a tree, or even sit on the ground. Although rarely offered, patients will often say they cannot sleep flat on their back, but they have to be in a fetal position or in a chair with the spine bent. Coexistence of PAD and spinal stenosis is common. Functional testing is often required to determine the relative components contributing to the symptoms. A simple test to perform in office is to have the patient stand with both feet flat, without leaning on an object, and to record the time to onset and location of symptoms. This can be directly compared with the symptoms during exercise testing.

Orthopedic Pain

The coexistence of arthropathy and PAD is common. Ankle, knee, and hip discomfort may be confused with or additive to symptoms caused by PAD. Clues such as a previous injury or localized swelling at a joint can provide insight into the abnormalities. If the physical examination or risk factors for PAD are positive, noninvasive testing is appropriate. Because the symptoms in this population are often exertional, exercise treadmill testing is often the best selection.

Buttock and Thigh Claudication

Claudication at the buttock and thigh attributable to PAD is difficult to diagnose. Orthopedic and lumbar spinal stenosis may cause the symptoms. Isolated internal iliac stenosis without adequate collateralization may also cause regional blood flow impairment and may be present in patients with previous aorto-bi-iliac or aorto-bi-femoral revascularization. Exercise testing with postprocedure pressures or Doppler testing is not sufficient in this setting. Recently, exercise TcPO2 and near infrared spectroscopy testing have been used to define the diagnosis.

Critical Limb Ischemia

Critical limb ischemia is a clinical diagnosis, based on the presence of ischemic rest pain or tissue loss. Testing is supportive, but does not define the presence or absence of critical limb ischemia. The clinical evaluation is similar to claudication, but it needs to be expedited. When critical limb ischemia...
is in the setting of a wound, the viability of the toe, foot, or limb needs to be established. The presence of gas gangrene or extensive necrosis such that a usable limb is unable to be salvaged can be determined by physical examination. X-ray or MRI of the bone are often needed to determine whether osteomyelitis is present. When the limb is nonviable, amputation is often the best option.

Ischemic rest pain shares many of the symptomatic features of peripheral neuropathy. The quality of the discomfort is often burning or sharp, but pain is exacerbated by elevation of the feet, even when simply supine. Hanging the foot over the bed, sleeping in a chair with the legs dependent, or, paradoxically, walking around the room provide some relief by using gravity to assist with blood delivery (clinically apparent as dependent rubor). Edema is often present secondary to the foot being in a dependent position, and a deep vein thrombosis may be suspected. The foot is often exquisitely tender to palpation. When palpable, the pulses are often absent. However, the edema and discomfort often make palpation quite difficult. A hand-held Doppler is appropriate to use at the bedside to avoid delaying diagnosis and treatment.12

Acute Limb Ischemia

When acute limb ischemia is present (caused by injury, embolus, thrombosis in situ, or complication from catheterization of closure device), urgency is required to reestablish blood flow and salvage the limb. The classic presentation is the 5 Ps: a pulseless, painful, pale, polar (cold), and paralyzed (weak) limb. Imaging should be obtained to identify the location and process. In addition to the imaging studies previously discussed, transesophageal echocardiography is often appropriate to assess a cardiac source for the embolism. When thought to be embolic, proceeding directly to the endovascular or surgical suite for thrombolysis or embolectomy is appropriate. How to best image and to revascularize depends on the facilities available at each institution. Increasingly, angiography is used in the operating room to guide treatment.13

Disclosures

None.

References


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