This chapter reviews vascular imaging. The main points of this chapter are:

1. CT angiography has largely supplanted catheter angiography for diagnostic vascular studies.
2. Catheter angiography may still be used in those situations where therapy is necessary or detail beyond the current resolution of less invasive methods is necessary.
3. Specific recommendations for imaging vary with the anatomic location and clinical situation.

Note that the book has already covered many examples of vascular imaging: Chapter 1 discussed renovascular hypertension; Chapter 2 discussed scrotal varicocele; Chapter 3 discussed cerebral artery aneurysm and malformation; Chapter 10 discussed pulmonary embolism; and Chapter 11 discussed coronary artery disease. This still leaves a number of topics, which this chapter addresses, along with the promise in Chapter 4 (see page 47) to address vascular imaging in the evaluation of neurologic symptoms including TIA and stroke.

In general, the gold standard for vascular imaging has long been catheter angiography. However, diagnostic catheter angiography has largely been replaced, at least for screening purposes or initial diagnosis, by other methods. In general, vascular ultrasound is now widely used for screening and follow-up examination (where applicable), supplemented by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA) when more definitive evaluation is required or when ultrasound cannot be used. CTA has the advantages of higher spatial resolution and is less subject to motion artifact. MRA using flow techniques can be performed without contrast material, although contrast-enhanced MRA tends to provide better images. Catheter angiography nowadays is often used to confirm (or disprove) results obtained from noninvasive vascular imaging, and to allow intravascular intervention.

Beyond these generalizations, specific recommendations for vascular imaging vary by body part, and will be covered below, first addressing the arterial tree and then the venous side of circulation. Although this is a book on the radiology of symptoms, vascular imaging is performed relatively frequently in asymptomatic individuals for screening purposes, and the chapter will cover this topic as well.
ARTERIAL STUDIES

Intracranial aneurysms

As noted above, Chapter 3 discussed symptomatic intracranial aneurysms. What about asymptomatic intracranial aneurysms? Such asymptomatic intracranial aneurysms may be discovered either incidentally, during imaging for unrelated symptoms, or because of screening. In both cases, current recommendations are to monitor small aneurysms annually for two to three years. If the aneurysm grows in this interval, referral for consideration of intervention is appropriate; if it is stable, extending the monitoring interval to two to five years is appropriate. “Small” in this context means less than 10 mm, although some authorities use 7 mm. Of course, such screening must take into account the age and general medical condition of the patient with more aggressive management of younger and healthier patients.

Regarding screening for asymptomatic aneurysms, there is no role for such studies in the general population. For subsets of the general population with a higher risk of aneurysm, recommendations are generally not to screen: for example, recommendations are not to screen for genetic syndromes known to be associated with intracranial aneurysms, and not to screen patients with a single first-degree relative with an intracranial aneurysm which has bled. Patients with two (or more) first-degree relatives with bleeding intracranial aneurysms, however, should be screened. The role of screening in adult polycystic kidney disease (which is associated with increased risk of intracranial aneurysms) is unsettled.

Aortic arch, carotid vessels, and intracranial vasculature: screening and in patients with ischemic symptoms

As noted in Chapter 4, in patients with neurologic symptoms, a facilitated work-up including MRI of the brain and either US, CTA, or MRA of the carotid arteries is recommended to evaluate for a causative lesion (Figure 1). The main purpose of this facilitated work-up is to find stenotic arterial lesions which will benefit from carotid endarterectomy. Current recommendations call for carotid endarterectomy (CEA) for patients with 70-99% stenosis, and for men (but not women) with 50-70% stenosis; recommendations for women are different because clinical trials have shown less benefit for CEA compared to medical treatment in women with <70% stenosis. The facilitated work-up is important, because the shorter the delay between the symptoms and surgery, the better the outcome, with the best outcomes when the surgery is performed within two weeks of the neurologic event. If ultrasound is the initial study for documentation of stenosis, an MRA or CTA is typically performed for confirmation of results, since US tends to overestimate stenosis and does not provide for the direct visual assessment of the stenosis as well as CTA or MRA (Figure 2). For symptomatic disease with less 50% stenosis, annual follow-up is recommended to document stability, with referral for further evaluation in the event of disease progression.

In general, expert groups including the United States Preventative Services Task Force, the American Heart Association, the American Stroke Association, and the American Society of Neuroimaging recommend against screening the general population for carotid stenosis. Bruits of the carotid are a better indicator of general vascular disease (e.g., coronary artery disease, lower extremity arterial disease, and contralateral carotid disease) than they are of ipsilateral carotid stenosis (Figure 3). In men between 40 and 75 years of age where study of the carotids after auscultation of a bruit or a community screening program (done on a self-referral basis) has discovered a stenosis, CEA should at least be considered in those with a stenosis of over 70% (Figure 4).
Figure 1. Less than 50% carotid artery stenosis in a 58 year old man with transient facial droop and aphasia. A. Axial diffusion weighted MR image demonstrates restricted diffusion in the left hemisphere adjacent to the ventricles (arrow) compatible with an acute cerebral infarction. B. CT angiogram through the left carotid shows an axial slice at the level of the proximal internal carotid artery (arrow) with axial images arrayed around the vessel from proximal to distal, with reconstructed long axis views at perpendicular projections on the right side of the panel, showing less than 50% stenosis. C. 3D maximum intensity projection MR angiogram confirms less than 50% stenosis of the left internal carotid artery.
Figure 2. 50-70% stenosis of the right internal carotid artery in an 84 year old man with slurred speech and decreased strength in the right arm and hand. A. Axial diffusion weighted MR image shows a focus of restricted diffusion in the right hemisphere (arrow) compatible with an acute infarct. B. Right carotid ultrasound study shows peak systolic velocity (PSV) and internal carotid artery – common carotid artery ratio (IC/CC) compatible with only mild stenosis. C. Ultrasound at the
level of the bifurcation showing extensive atherosclerotic plaque making visual estimation of stenosis difficult. D. CT angiogram through the right carotid bifurcation (arrow) shows 50-70% stenosis along the proximal internal carotid artery.

Figure 3. Carotid stenosis on the left side in an 83 year old woman with a right carotid bruit. A. Carotid US study (right carotid artery) shows normal peak systolic velocity (PSV) and internal carotid artery to common carotid artery PSV ratio (IC/CC) on the side of the bruit. B. Carotid US study (left carotid artery) shows abnormal elevated peak systolic velocity and internal carotid artery to common carotid artery PSV ratio compatible with greater than 70% stenosis on the side opposite the bruit.

Figure 4. Severe left internal carotid artery stenosis in a 73 year old woman with an abnormal community screening result. A. Carotid US study shows dense calcification and severe stenosis of the proximal internal carotid artery. This was a diagnostic study done on the basis of the abnormal screening exam. B. Axial CT from a CT angiogram shows greater than
90% stenosis of the left internal carotid artery, with a trickle of flow (white arrow) along the periphery of a calcified arterial wall (black arrow). Other axial images and windowing confirmed that the lumen was open along the medial aspect.

**Thoracic aortic aneurysm**

Patients with symptomatic thoracic aortic aneurysms are generally direly ill because of rupture or dissection of the aneurysm. These patients typically come to the emergency room with severe chest pain. They will undergo emergent CT study to differentiate thoracic aortic dissection from pulmonary embolism (see pages 159-160). Most cases of dissection require emergent, hopefully life-saving, intervention. Some chronic thoracic aortic aneurysms may cause ongoing chest pressure (Figure 5).

Asymptomatic thoracic aneurysms are generally discovered during chest imaging for unrelated symptoms: for example, echocardiography, done for evaluation of cardiac function or valvular anatomy or chest x-ray examination for evaluation of cough and fever. Chest x-rays cannot differentiate aneurysmal dilatation of the aorta from tortuosity of the aorta, and so patients with an abnormal aortic contour need further evaluation with either chest CTA or MRA to define the aortic arch.

For patients with incidentally discovered, asymptomatic thoracic aneurysms, CTA or MRA allows evaluation of such important anatomic features as whether the aneurysm involves the ascending aorta, the descending aorta (with or without extension into the abdomen), the aortic arch and great vessels or some combination thereof. CTA or MRA also allow measurement of the size of the aneurysm, which is critical information since the risk of rupture is directly related to the size of the aneurysm: one study found that the five year risk of rupture was 0% for aneurysms less than 40 mm, 16% for aneurysms between 40 and 59 mm, and 31% for aneurysms greater than 60 mm. Another study found that aneurysms over 60 mm had an annual 16% risk of dissection, rupture, or death. Growing aneurysms also have an increased risk of rupture, even if smaller than 60 mm. As a result, general indications for surgery include: development of symptoms; diameter greater than 50 mm for ascending aortic aneurysms and 60 mm for descending aortic aneurysms, growth of greater than 10 mm per year, and evidence of dissection. These indications lead to the following recommendations for follow-up of known thoracic aneurysms: 1) a repeat study in 6 months from aneurysm discovery, and, if stable; 2) annual studies thereafter to document stability. Sequential scanning using the same imaging modality (and even the same center or equipment, when possible) will likely yield the best results (Figure 6). When and if the above thresholds are crossed, surgical consultation is indicated.
Chapter 12  Vascular Imaging  Page 175

Figure 5. Enlarging thoracic aortic aneurysm in an 84 year old man with chest pressure. A. PA chest x-ray shows a dilated, tortuous aortic aneurysm (arrows). B. PA chest x-ray taken 18 months later (when the patient had a sensation of increasing chest pressure) shows that the aneurysm is larger (arrows).

Figure 6. Stable thoracic aortic aneurysm in a 67 year old man with a known thoracic aortic aneurysm. A. Axial contrast-enhanced chest CT shows a thoracic aortic aneurysm which measured 51 mm. Annual studies were performed in follow-up. B. Axial contrast-enhanced chest CT done three years later shows an identical appearance and measurement. Interval annual studies (not shown) showed similar results.

Abdominal aortic aneurysm (AAA)

As with thoracic aortic aneurysms, patients with symptomatic abdominal aortic aneurysms are often direly ill and present to the emergency room with severe abdominal pain, if they don’t exsanguinate first. These patients will undergo emergency abdominal CT followed by emergency surgery, if they are fortunate enough to survive. In general, symptomatic AAA’s should undergo immediate evaluation by a surgeon. Patients with AAA may also present with an otherwise asymptomatic pulsatile mass, in which case they should undergo US or CT evaluation.

Most AAA’s are completely asymptomatic, however, and will be discovered either incidentally when imaging the abdomen for an unrelated abnormality or upon screening (Figure 7). With respect to screening, patients may have an AAA after self-referral to a community screening program. Current recommendations call for screening any man greater than 60 years of age with a parent or sibling with an AAA, and for screening male smokers (or ex-smokers) between the ages of 65 and 75. Aneurysms larger than 5.5 cm should be referred for surgical consultation. For aneurysms between 3.0 and 4.0 cm, US should be performed every 2 to 3 years, whereas aneurysms measuring 4.0 to 5.4 cm should be monitored every 6 – 12 months, with referral for surgical consultation if the aneurysm grows to greater than 5.5 cm, if the aneurysm grows more than 0.5 cm in a 6 month period, or if the aneurysm becomes symptomatic.
Figure 7. Abdominal aortic aneurysm incidentally discovered in a 74 year old with back pain undergoing lumbar spine MR study. A. Sagittal T1 weighted lumbar spine MR study shows an aneurysm of the lower abdominal aorta (arrow). B. Axial T2 weighted MR image through the L4 vertebral body level shows the aneurysm anterior to the lumbar spine (arrow). C. Contrast-enhanced axial CT angiogram shows the abdominal aortic aneurysm (arrow). D. Surface rendering based on CT data of the aneurysm, showing the location along the distal infrarenal abdominal aorta (arrow).

Other abdominal arteries (and veins)
Patients with chronic mesenteric ischemia or “intestinal angina” present with cramping abdominal pain following a meal (particularly a large fat-containing meal) which typically subsides approximately two hours after the meal\textsuperscript{12}. These patients may have associated involuntary weight loss because of food avoidance caused by postprandial pain. CTA of the abdominal aorta and branches offers an excellent method of evaluation for suspected intestinal angina. Given the relatively rich anastomotic connections between the celiac artery, superior mesenteric artery, inferior mesenteric artery, and iliac artery branch vessels, stenosis or occlusion of only one branch rarely causes symptoms in the absence of simultaneous stenosis or occlusion of other branches, and therefore the diagnosis is usually made only when there are symptoms and stenosis or occlusion of at least two vessels\textsuperscript{13}.

Patients with acute mesenteric ischemia may have preceding symptoms of chronic intestinal ischemia, an “acute-on-chronic” situation where the patients have passed the threshold from intermittent to permanent symptoms. Patients with acute mesenteric ischemia usually have severe periumbilical pain\textsuperscript{13}. Acute mesenteric ischemia may be caused by superior mesenteric artery embolism (50% of cases), superior mesenteric artery thrombosis (15-25%), mesenteric venous thrombus (5%) or nonocclusive ischemia (20-30%)\textsuperscript{14}, with each of these causes typically having a different clinical scenario (although the patients will all have abdominal pain regardless of the ultimate cause of the ischemia). Those with superior mesenteric embolism typically have cardiac disease with the embolism originating from the left atrium, left ventricle, or cardiac valves; those with acute thrombus usually have “acute-on-chronic” atherosclerotic disease of the mesenteric vessels with a prior history of intestinal angina (as noted above); those with acute venous thrombus may have a history of a hypercoagulable state (Figure 8), portal hypertension, abdominal infections, or abdominal trauma; and those with nonocclusive mesenteric ischemia (NOMI) are usually elderly patients, often with a cardiac condition being treated with drugs (e.g., diuretics) which reduce intestinal perfusion\textsuperscript{14}. When acute mesenteric ischemia is strongly suspected, angiography is recommended since it is the reference standard for the diagnosis and may also allow for percutaneous intervention such as angioplasty\textsuperscript{14}. When angiography is unavailable, or when the diagnostic suspicion is not high, CTA is an excellent alternative since it allows not only evaluation of the arterial tree but also evaluation of findings of associated bowel infarction (and other causes of abdominal pain).

![Portal venous thrombosis in a 34-year-old woman on oral contraceptives with abdominal pain. Axial contrast-enhanced abdominal CT study during the portal venous phase study shows a clot filling the portal vein (arrow).](image)

Evaluation of gastrointestinal bleeding typically relies on endoscopy. Occasionally, intermittent bleeding or bleeding with a low flow rate may be difficult to diagnose with endoscopy. In these cases, arteriography or CTA may be performed\textsuperscript{14}, but nuclear medicine studies allow diagnoses of lower rates of bleeding (even less than 0.5 mL/minute) and can reliably direct surgical intervention\textsuperscript{15} (Figure 9).
Figure 9. Gastrointestinal bleeding in a 75 year old. Sequential images (left to right, top to bottom) from a tagged red cell nuclear medicine examination demonstrate tracer in the right upper quadrant which on subsequent images shows a typical pattern of proximal small bowel distribution (arrows). The patient had already undergone upper and lower endoscopy without a clear source of hemorrhage; following the tagged red cell study he underwent repeat upper endoscopy which showed an ulceration along a duodenal diverticulum with active bleeding.

Lower extremity arterial evaluation

Symptoms of lower extremity peripheral arterial disease (PAD) include classic claudication, atypical leg pain, non-healing ulcers, and pain while at rest (when severe). However, up to 90% of patients with lower extremity peripheral arterial disease as determined by ankle-brachial ratio measurements may be asymptomatic. For patients with symptoms, the initial diagnostic study is an ankle-brachial ratio determination, which involves comparison of the blood pressure at the ankle (using the posterior tibial or dorsalis pedis artery) with the higher of the right and left arm pressures: an ABI of <.90 is diagnostic of PAD (with pressures between .85 and .90 indicating mild arterial impairment, between 0.40 and 0.85 moderate arterial impairment, less than 0.39 severe arterial impairment), whereas an ABI of greater than 1.3 suggests calcified, noncompressible vessels which may also be a source of symptoms. ABI readings between 0.91 and 1.0 are borderline and should be followed by an exercise exam wherein serial ABI calculations are made at 1-minute intervals following walking on a treadmill for five minutes at 2 mph on a 12% incline; ABI will typically remain stable or increase following exercise and decreases of 20% or more are diagnostic of PAD. For patients with symptoms and (either standard or exercise) abnormal ABI measurements who are candidates for revascularization, further evaluation can be performed with segmental pressure evaluation or CTA or MRA. Segmental pressure evaluation records the blood pressure at the ankle while inflating blood pressure cuffs at each of several lower extremity locations to better localize diseased vessels. Segmental pressure evaluation may be
supplemented by pulse volume recordings which are particularly helpful when extensive calcification makes vessels non-compressible. CTA and MRA can provide a visual “road map” of the lower extremity vascular tree documenting the location and quantifying the degree of arterial stenosis.

While screening for lower extremity PAD is not recommended for the general population, patients may self-refer to community screening programs and then come to the primary care practitioner with abnormal results. Indications for obtaining an ABI include evaluation of high risk patients, including those over 50 years of age who are smokers or diabetic.

**LOWER EXTREMITY VENOUS STUDIES**

**Pain and swelling**

Symptoms of acute deep venous thrombosis include pain and swelling of the calf. Other disease processes (e.g., muscle tear, lymphangitis, venous insufficiency, and Baker’s cyst) may demonstrate similar clinical features, however, and distinction among these entities is important for patient management, since DVT can result in pulmonary embolism with significant associated morbidity and mortality. Typically, ultrasound examination consists of a combination of gray-scale imaging without and with compression where possible, color Doppler examination, and spectral Doppler examination with augmentation to assess for appropriate deep venous flow. Since reflux accounts for some cases of lower extremity pain and swelling even when there is no deep venous thrombus, it makes sense to routinely evaluate the saphenofemoral junction for reflux during Valsalva or during an upright position in those patients with lower extremity symptoms when the remainder of the study is normal. Documentation of either a filling defect (Figure 10) or lack of compressibility within the deep venous system is diagnostic of deep venous thrombosis. The examination will typically include compression evaluation of at least the popliteal and femoral veins*, and color flow images of the deep venous system including the calf to evaluate for filling defects. Note that clot may be isoechoic and therefore difficult or impossible to visualize without Doppler imaging and compression.

Once clot has formed, differentiation between persistent and recurrent deep venous thrombus by ultrasound is difficult. Impedence plethysmography and MRI examination may be of benefit in these situations.

- Note that the preferred term for the vein formed by the continuation of the common femoral vein after the deep femoral vein has branched off, and connecting the common femoral vein to the popliteal vein, is the “femoral vein” and not the “superficial femoral vein” as indicated in some texts. The misleading term “superficial femoral vein” should be eliminated from use: the structure in question is part of the deep venous system and referring to the structure as “superficial” is a source of confusion which may have severe clinical consequences.
Varicose veins and valvular insufficiency

Patients with varicose veins from venous valvular insufficiency may have imaging performed of the deep and superficial systems prior to venous ablation procedures. Such evaluation typically includes the deep venous system for thrombosis (since valvular insufficiency and DVT may both result in leg swelling and pain) and documentation of the location and severity of venous valvular insufficiency. Such insufficiency is typically documented at the saphenofemoral junction by scanning during Valsalva or with the patient in an upright position. The examination can also document varicose veins and the caliber and depth of the saphenous veins (important for surgical planning).

SUMMARY

Modern imaging of the vascular tree typically involves a combination of ultrasound, CTA, and MRA. Catheter angiography is typically reserved for cases where therapy is necessary, or where detail beyond the current resolution of noninvasive vascular studies is necessary. Specific recommendations for imaging symptomatic patients and screening asymptomatic patients vary with the anatomic location and are reviewed above.
REFERENCES

1 Singer RJ, Ogilvy CS, Rordorf G. Unruptured intracranial aneurysms. UpToDate, accessed 10/12/09.
2 Greelish JP, Mohler ER, Fairman RM. Carotid endarterectomy in symptomatic patients. UpToDate, accessed 12/10/09.
4 McCarron, MO, Goldstein LB, Matchar DB. Screening for asymptomatic carotid artery stenosis. UpToDate, accessed 10/10/09.
8 Woo YJ, Mohler ER. Management and outcome of thoracic aortic aneurysm.
12 Tendler DA, LaMont JT. Chronic mesenteric ischemia. UpToDate, accessed 11/25/09.
13 Tendler DA, LaMont JT. Acute mesenteric ischemia. UpToDate, accessed 11/25/09.
17 Mohler ER. Noninvasive vascular diagnosis in lower extremity peripheral arterial disease. UpToDate, accessed 11/23/09.