PERIPHERAL ARTERY OCCLUSIVE DISEASE

This CME activity will review the manifestations of arterial occlusive disease affecting peripheral arteries including clinical presentation, diagnosis, and principles of treatment. Consideration will be given to occlusive disease that restricts blood flow and to embolic disease. For the lower extremities, we will consider the differences between acute and chronic presentations and between disease affecting the aortoiliac (inflow) segments versus that affecting the femoral, popliteal, and tibial (outflow) segments. Raynaud's disease and Burgers disease will be reviewed briefly as unusual manifestations of arterial occlusive disorders.

ACUTE ISCHEMIA

Acute ischemia indicates a sudden reduction in blood flow, which typically is caused by arterial occlusion due to traumatic disruption, dissection, atherosclerosis, bypass failure, or embolism.

Trauma and aortic dissection

Trauma can be either from blunt injury that stretches arteries to the point of rupture or penetrating injury that directly injures the vessel (including surgery and catheterization). Arterial disruption by either mechanism can result in limb-threatening loss of blood flow.

Arterial dissection may be a complication of vascular intervention, in which case it usually can be corrected at the time of surgery. Spontaneous dissection typically occurs in the thoracic aorta when blood enters the medial layer of the artery at a site of surface disruption. The underlying cause may be atherosclerosis with severe hypertension, or collagen vascular diseases that weaken the wall, such as Marfan sydrome. Blood dissects through the media, pushing the inner wall of the artery into the lumen, narrowing or occluding it. Patients present with severe chest pain and symptoms of ischemia in the territory of the occluded vessel: extremity pain, stroke, myocardial infarction, acute renal failure, or mesenteric ischemia. Diagnosis is usually by CT angiogram. Vascular laboratory testing can helpful in determining the extent of reduction in extremity, mesenteric, carotid, or renal blood flow. Treatment for dissection beyond the aortic arch is primarily strict blood pressure control. Intervention is required for arch involvement, which can cause death due to coronary occlusion, cardiac tamponade, stroke, or acute aortic valve insufficiency. More distal dissection requires intervention for failure of medical therapy or ischemic complications from occlusion of mesenteric, renal, or lower extremity arteries. Intervention involves repair of the entry site in the artery wall (often by stent grafting), creation of re-entry sites, and revascularization by stenting or bypass when ischemia persists.

Atherosclerosis

Although atherosclerosis is a chronic disease, sudden hemorrhage into a plaque or terminal thrombosis of a stenotic segment can cause an acute decrease in flow. Patients may experience new onset of symptoms, such as claudication, or in extreme cases, acute ischemia with rest pain and threatened limb. Similarly, bypass grafts or arteries that have undergone stenting or angioplasty may suddenly occlude due to thrombosis of a segment with restenosis. The resulting degree of ischemia will depend on the severity of the underlying disease and whether or not the new thrombus extends into previously

uninvolved segments.

Peripheral Embolism

Emboli are symptomatic when they block an end artery supplying blood to tissue that does not have an adequate collateral supply. They may consist of thrombus, cholesterol debris from an atherosclerotic plaque, fragments of vegetations from diseased cardiac valves, or septic embolic from infection that involves the arterial tree. Those that arise from the heart are the most common and typically result from arrythmias, such as atrial fibrillation, that cause thrombus to form in the relatively stagnant pool of atrial blood. Other cardiac sources include mural thrombus from damaged myocardium following myocardial infarction, valvular disease caused by infection or calcification, or most rarely embolization of tumor (atrial myxoma). These sources are identified by echocardiography and electrocardiogram.

Arterial-arterial embolism is caused by release of debris from disruption of atherosclerotic plaque or from mural thrombus that forms on the damaged surface of these lesions. Thrombus can also break off from the wall of aneurysms, which typically develop mural thrombus due to relatively slow flow at the wall of the enlarged vessel. If the source of the embolism is proximal, in heart or aorta, emboli will be bilateral. Whereas, if the source is more distal, in an iliac or femoral artery for example, emboli will be unilateral, in the territory supplied by the involved artery.

Emboli to the carotid circulation may cause stroke, to the renal artery renal failure and hypertension, to the mesenteric circulation acute mesenteric ischemia, and to the extremities acute ischemia. Diagnosis of peripheral embolism is straight forward when a patient with no prior symptoms presents acute onset of pain in an extremity that has absent pulses, particularly when the contralateral extremity has a normal vascular examination. Frequent locations of occlusion include the first branch of the superior mesenteric artery, the brachial bifurcation below the elbow, and the common femoral artery. Duplex scanning can be helpful in identifying the location of occlusion, which is typically at a bifurcation, where the vessel lumen decreases.

Small emboli are well tolerated in most of the peripheral circulation, but may manifest as the “blue toe syndrome,” in which occlusion of digital arteries causes sudden pain and discoloration of one or more toes (Figure 1). If emboli are minor and non-recurrent, the blue toe syndrome often resolves without tissue loss. Extensive embolization, as is sometimes seen after vascular interventions that disrupt mural thrombus or plaque, can result in tissue loss.

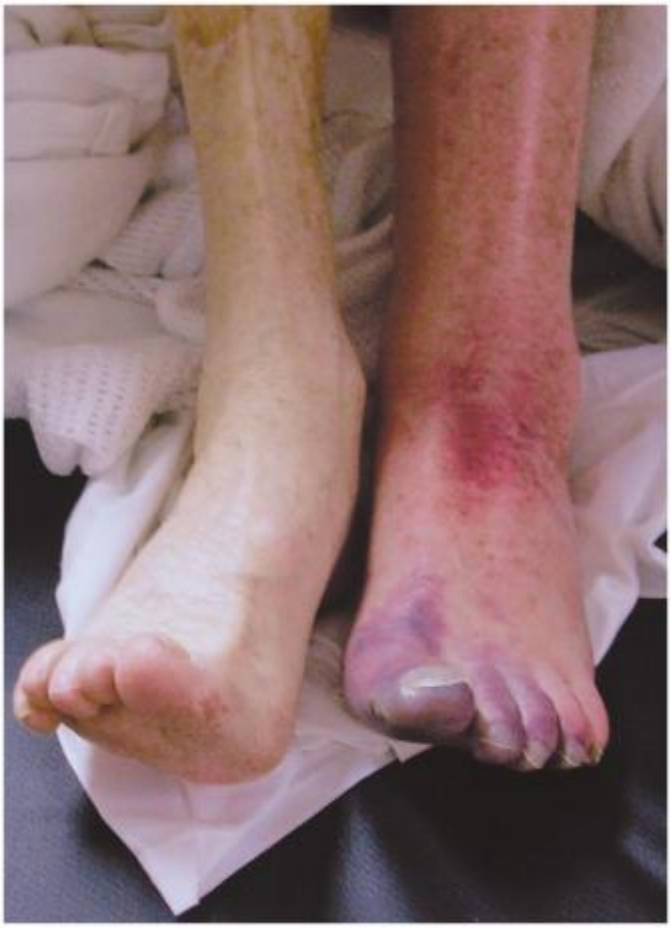

Figure 1

When otherwise occult, the source of embolism should be sought, including a cardiac workup and arterial studies to rule out aneurysm or significant plaques. The later may include ultrasound, CT, or MR imaging. When identified as the source, culprit lesions should be treated: exclusion of aneurysms and bypass or stenting of significant plaques. If arrhythmia is the cause, anticoagulation is generally indicated.

Factors influencing the degree of peripheral ischemia

The degree of ischemia that results from acute peripheral ischemia is dependent on many factors (see Table 1). First is the level of occlusion. Tissues supplied by only a single artery are particularly susceptible to injury. Occlusion of such end arteries causes total ischemia to the vessel's tissue bed. An example is the renal artery, which in most cases, is the single blood supply to the kidney. Acute occlusion of this vessel is poorly tolerated: irreversible renal ischemia follows within minutes to hours of acute renal artery occlusion. The extremities and most organs have multiple feeding arteries. For example, the brain has a robust collateral circulation through the Circle of Willis, and acute occlusion of a vertebral or carotid artery is often asymptomatic. Similarly, abrupt occlusion of the subclavian artery is very well tolerated due to the rich collateral network around the shoulder. Acute occlusion of the common femoral artery is poorly tolerated, whereas occlusion of the superficial femoral artery is well tolerated as long as flow is preserved to the profunda femoris, the major collateral to the lower extremity. Ironically, patients who have chronic arterial occlusive disease tolerate acute artery occlusions better than others because they have developed collateral circulation in response to the chronic disease.

Tissues with high basal metabolic rates, such as the brain or kidneys, do not tolerate ischemia as well as those with lower metabolic rates, such as the skin or muscle. This is why a severed extremity can be reimplanted after hours but the brain suffers irreversible damage after just minutes of flow cessation. In

the case of trauma, the severity of ischemia resulting from arterial occlusion will also depend on the presence of associated venous and soft tissue injury, infection, shock, and temperature. Because cooling can lengthen the time that ischemic tissue remains viable, severed extremities should be cooled until reimplantation. Cooling is best accomplished by placing the severed part in a towel inside a plastic bag and then placing the bag in ice. This technique avoids a cold-induced injury from direct contact with the ice and injury from exposure to hypo-osmotic water.

Level of occlusion
Metabolic rate of involved tissue
Presence of collaterals
Prior chronic occlusive disease
Associated injuries: veins/soft tissues
Infection
Shock
Temperature

Table 1: Factors Influencing the Degree of Ischemia

Diagnosis of acute peripheral ischemia

Acute extremity ischemia presents with the six P's: pain, pallor, pulselessness, poikilothermia (poikilotherms, like toads and snakes, are unable to control body temperature and feel cold to the touch), paresthesias (numbness), and paralysis. Patients with moderate occlusive disease may have many of these findings, but paralysis and paresthesia indicate critical limb ischemia (CLI): impending tissue loss and the need for immediate intervention. Therefore revascularization is urgent if the extremity is becoming weak or loses sensation (this means loss of fine touch or two-point discrimination, not merely a sensation of tingling). Severely ischemic limbs may have absent arterial signals at the ankle, but venous signals are still present. Limbs with irreversible ischemia are cold, numb, immobile, and mottled with a bluish purple, non-blanching discoloration. They will have neither venous nor arterial signals at the ankle. With CLI, pedal Doppler signals are usually absent or markedly diminished and monophasic, and ankle pressures are usually below 0.4, but these findings alone are not diagnostic since limbs may be viable in chronic ischemia even if Doppler signals are absent.

Pain
Pallor
Poikilothermia (cold)
Pulselessness
Paresthesia
Paralysis
Numbness and weakness indicate impending tissue loss![1]
1 Vascular surgeons on call often add what seems like an inevitable seventh P: Post midnight.

Table 2: The Six P's of Acute Ischemia

Treatment of acute ischemia

Treatment is anticoagulation with heparin followed by restoration of blood flow (unless the ischemia is irreversible) and prevention of recurrence. When the extremity is numb or weak, urgent intervention is

required, usually by thrombectomy, thrombolysis and angioplasty, embolectomy, or bypass. When ischemia is less severe, prolonged, catheter-directed therapy with infusion of lytic agents such as tissue plasminogen activator to dissolve the thrombus or embolus may be attempted. Common locations for embolization from atrial fibrillation are the femoral and brachial bifurcations. The typical presentation is of an elderly patient with atrial fibrillation who has stopped taking her anticoagulant (warfarin) and comes to the hospital with the acute onset of ischemia of a previously asymptomatic limb. Surgical thrombectomy can be performed under local anesthesia and is highly successful in this circumstance since the occlusion tends to be isolated to the arterial bifurcation. Clots distal or proximal to the operative site can be removed using balloon catheters that can pull thrombus out of the artery. In contrast, a patient who has had an arterial bypass that suddenly fails or who suffers acute thrombosis of a distal artery in the presence of occlusive disease may have extensive thrombosis involving distal vessels. In this case, lytic therapy with tissue plasminogen activator or similar agents is often more effective because it can open small arteries that cannot be reached surgically (Figure 2).

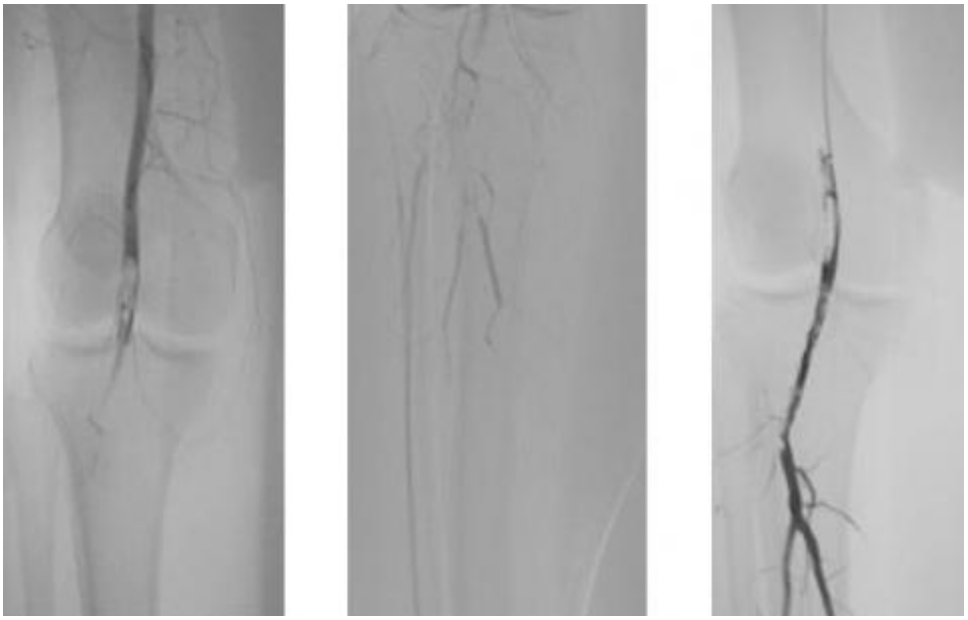

Figure 2

Compartment Syndrome

Revascularization of profoundly ischemic extremities can cause local and systemic complications. The local complication of compartment syndrome results from leaking of capillaries caused by reperfusion injury within muscular fascial compartments. The lower extremity has four such compartments (Figure 3). The anterior compartment of the leg is most commonly affected. Because tissue oxygenation and nutrition depends on capillary flow, tissue damage occurs when compartment pressures exceed capillary pressure (around 30 mm Hg). Patients may have the freeway syndrome (a palpable distal pulse but no “exits” for nutrient blood flow in the limb). Velocity waveforms will have low diastolic flow due to lack of capillary runoff. Early signs and symptoms of compartment syndrome are a tense compartment and pain on passive stretch of the muscles affected. Late findings of loss of sensation and motor weakness suggest eminent tissue loss and the need for urgent treatment, which is fasciotomy (surgical incision of the fascial compartments) to relieve pressure. In the anterior compartment (which is most commonly affected after lower extremity revascularization because it is a particularly tight compartment) neuromuscular compromise consists of loss of sensation in the first web space (deep peroneal nerve) and

weakness of the extensor hallicus longus (ability to hold the big toe up against downward pressure). A very late finding is loss of the distal pulse. This occurs when compartment pressures exceed arterial pressures. Treatment is by longitudinal incision of the fascia surrounding the compartments (Figure 4).

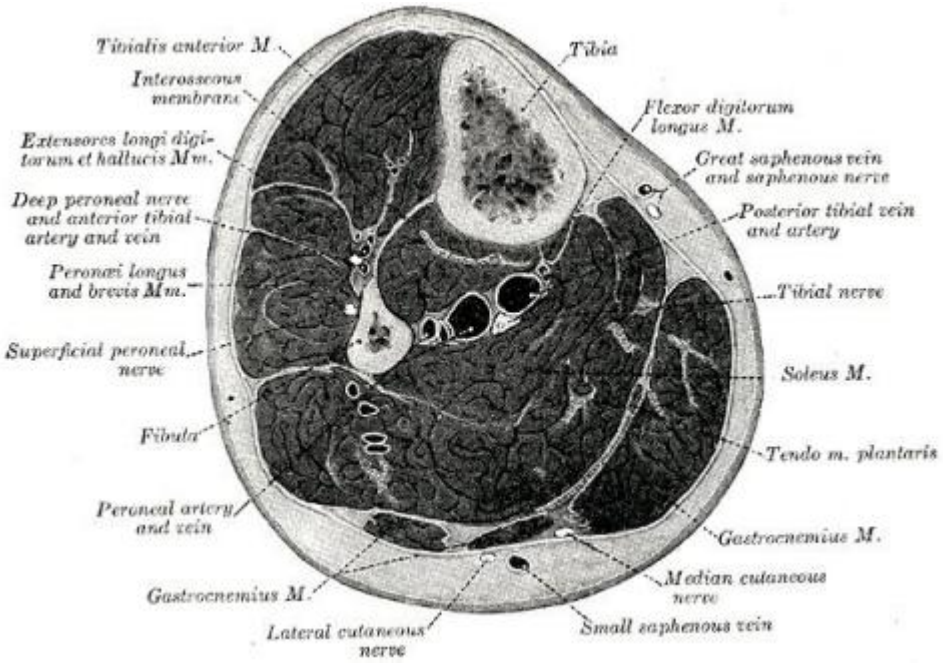

Figure 3

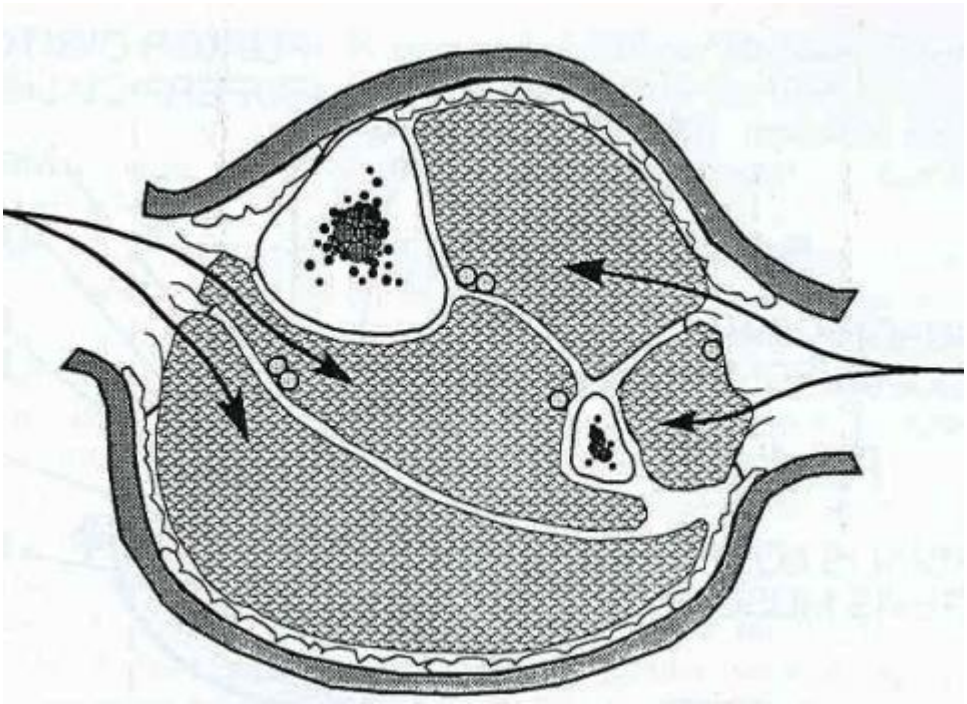
Figure 4

Systemic complications are caused by the release of potassium and myoglobin from injured muscle. Resulting hyperkalemia (diagnosed as peaked T waves on electrocardiogram) can cause fatal arrhythmia and is treated by infusion of glucose, insulin, and bicarbonate to drive the potassium into cells. Myoglobinemia causes acute renal failure due to crystallization of the pigment in microtubules. This is treated by administration of fluid, osmotic diuretics (mannitol), and bicarbonate (which alkalinizes the urine and solubilizes the pigment).

Vasospasm (Raynaud’s disease)

An overly sensitive sympathetic response to cold can cause vasospasm and acute, temporary ischemia (Raynaud's disease). which involves the digital vessels of the hands and sometimes the feet and is usually caused by exposure to cold. The diagnosis is generally made on the basis of the characteristic history, but the vascular lab can do confirmatory testing by documenting abnormally profound and prolonged decreases in digital pressures following cold exposure. The response to cold has three clinical phases: the involved digits are first white, due to lack of blood flow, then as flow returns they become blue due to venous congestion with desaturated blood, and finally they become hyperemic, pink, and may cause throbbing pain. Not every patient has this typical color sequence. Raynaud's disease refers to the primary process. It occurs mostly in women and is a benign condition, almost never causing tissue loss. Treatment primarily consists of avoidance of cold exposure, but calcium channel blockers may be helpful in some cases. *Raynaud's phenomenon* refers to this symptom complex when it is secondary to an underlying process (often collagen vascular diseases such as sclerodoma, lupus, Sjogren's syndrome, or rheumatoid arthritis, diseases of the arteries such as Buerger's disease, or other causes such as repetitive injury, smoking, and some medications). Loss of finger tips may occur when the associated disease causes occlusion of digital arteries. Because the occlusions are permanent, these patients will have decreased digital pressures and blunted arterial waveforms in the affected digits by photoplethysmography or Doppler, even when warm. Treatment is directed at the underlying disease.

CHRONIC ISCHEMIA

Symptoms

Atherosclerotic peripheral vascular occlusive disease (PVD) causes chronic reduction of flow to the lower extremities. This process is almost universal in the United States population, but is not manifest in most. When disease is severe enough to cause symptoms, patients first present with claudication: pain in the calf, thigh, or buttock brought on by exercise in a very reproducible, repetitive fashion. Symptoms are worse going up hill, carrying heavy objects, or climbing stairs since the lower extremity muscles must work harder. Pain is relieved when the patient stops for just a few minutes, even if still standing, as the muscles recover. They can then walk a similar distance again. This symptom does not vary from time to time or throughout the day. Pseudoclaudication is caused by lumbosacral disk disease and mimics arterial claudication. It differs in that it may vary during the day (typically worse in the morning) and from day to day. Usually patients with this problem must sit and rest for a prolonged time (up to twenty or thirty minutes), and their walking distance diminishes when they resume walking. They can walk further if leaning on something, for example using a shopping cart to relieve pressure on their back as they walk. Progressive occlusive disease may next manifest as rest pain (or sometimes numbness), which is experience in the distal foot, typically occurring at night, exacerbated by elevation and relieved by dependency (hanging the leg over the side of the bed). It may be confused with nocturnal leg cramps, which, unlike rest pain, are experienced in the calf muscle and are episodic. Patients may then go on to have tissue loss (gangrene or ulceration) due to severe, chronic ischemia. Rest pain and tissue loss are indications of critical limb ischemia and a threatened limb. They require relatively urgent intervention for limb salvage. The severity of disease can be described using the Rutherford classification (see Table 3). Elective intervention can be undertaken for Category 3 patients, whereas the higher categories require more urgent intervention to prevent limb loss.

Grade 0, Category 0: Asymptomatic
Grade I, Category 1: Mild claudication

Grade II, Category 2: Moderate claudication
Grade III, Category 3: Severe claudication
Grade IV, Category 4: Rest pain
Grade V, Category 5: Minor tissue loss – small ulcerations
Grade VI, Category 6: Major tissue loss – large ulceration, gangrene

Table 3: The Rutherford Classification

Lower extremity arterial occlusive disease may be in the inflow vessel (aorta and iliac arteries) or outflow vessels (femoral and tibial arteries), with the former being less common (Figure 5). Aortoiliac occlusive disease causes the Leriche Syndrome, named after the French physician, René Leriche. It has four components, all due to decreased flow in the aortoiliac system: high claudication (thigh and buttock pain with walking rather than the calf pain associated with more distal disease), erectile dysfunction, decreased femoral pulses, and lower extremity muscle atrophy. Femorotibial occlusive disease is more common, with the most common location being at the distal superficial femoral artery where it emerges from the adductor hiatus and continues into the popliteal space. Diabetic patients tend to have more distal disease (sparing the aortoiliac femoral arteries and involving tibial, pedal, and digital vessels) and medial calcification.

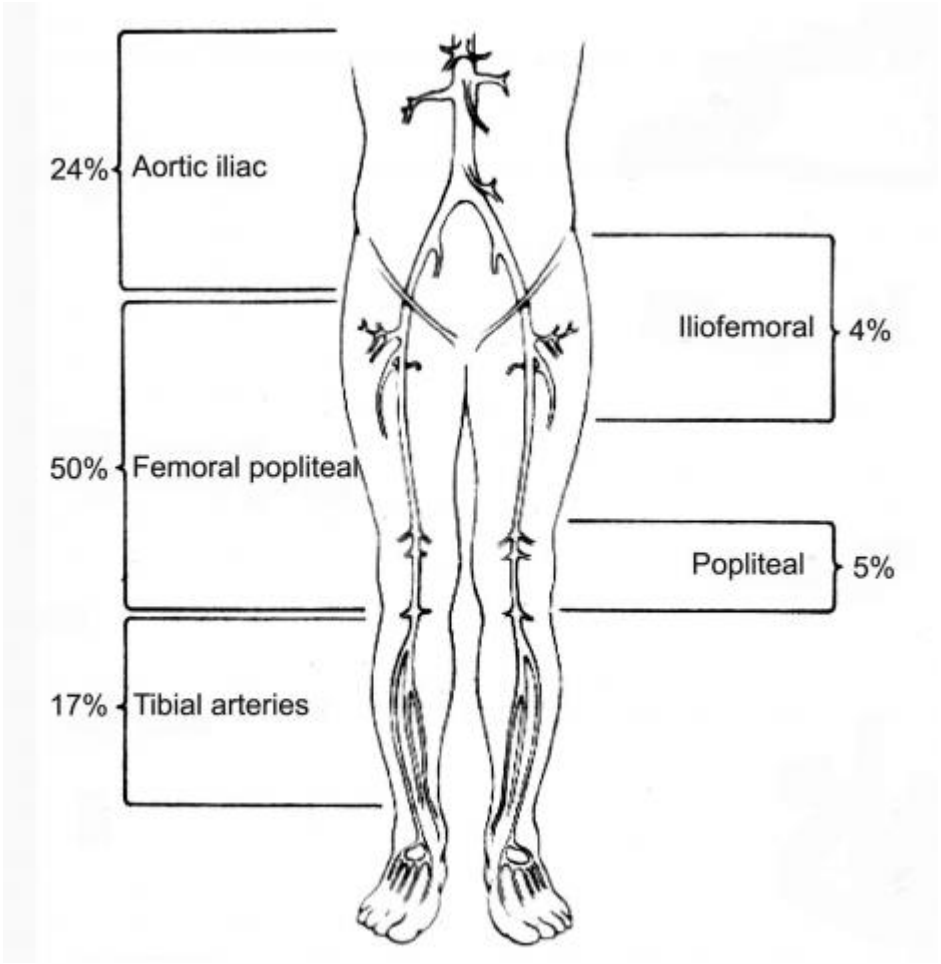

Figure 5

Diagnosis: History and physical examination

The diagnosis of PVD can be made by a careful history and physical examination. History should include the pattern of pain and the presence of risk factors for atherosclerosis (smoking, hypercholesterolemia,

diabetes mellitus, hypertension) and other manifestations of atherosclerosis (coronary disease or stroke). The physical exam may reveal signs of inadequate perfusion, such as hair loss of the distal leg and foot, thickened nails (since they grow slowly), and ulceration. Arterial ulcers are distinguished from venous ulcers because they are dry, pale, punched out, painful, and tend to occur over pressure areas and in the toes and distal foot where blood supply is the worst and trauma is most frequent (see Table 4). In contrast, venous ulcers, which are caused by venous hypertension due to valvular dysfunction, are moist, geographic in appearance (large with irregular borders) and pink and in the “gator” region of the leg (from the ankle to the mid calf) (Figure 6). Red blood cells entering the interstitial space by diapedesis out of capillaries. They deposit hemosiderin and create the hyperpigmentation in the gator area that is typical of this process. Increased interstitial protein causes fibrosis and an hourglass appearance to the lower leg as the region above the ankle contracts. Valvular dysfunction can be documented on duplex examination as reflux (reversed flow) lasting more than 5 seconds after compression maneuvers. When present in the superficial venous system, ablation or phlebectomy of the involved segments may prevent recurrence of ulceration and some relief of symptoms.

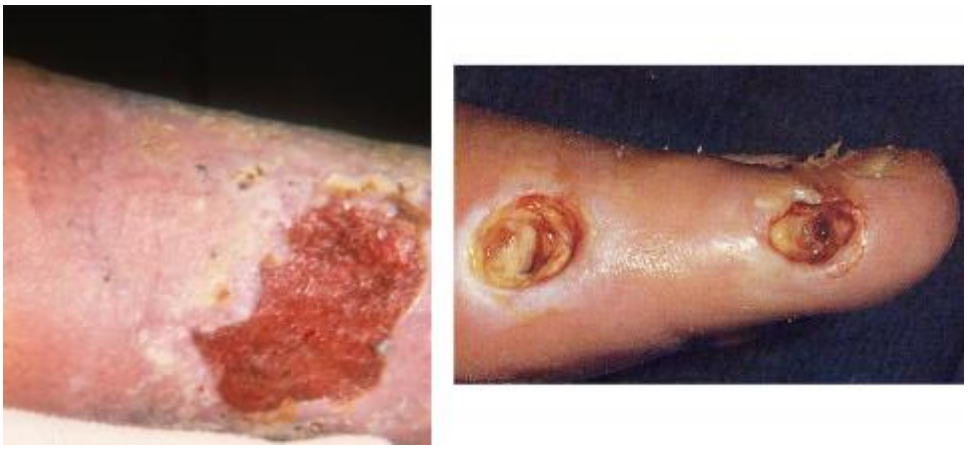

Figure 6

	Arterial Ulcers	Venous Ulcers
Cause	Arterial occlusions	Venous hypertension
Diagnosis	Absent pulses, diminished AAI’s	Valvular incompetence on ultrasound examination
Location	Distal foot and toes	Medial ankle and calf
Appearance	Dry, pale, punched out, sometimes black	Moist, pink
Symptoms	Painful (except in the case of diabetic neuropathy	Tend to be less painful
Treatment	Revascularization	Compression, local wound care

Table 4: Arterial versus Venous Ulceration

On physical examination of patients with PVD, elevation pallor suggests severe disease. If the examiner elevates the lower extremity with the patient supine, the foot will become pale when arterial pressure is lower than the hydrostatic pressure of the blood column from the heart to the foot. Thus, elevation pallor indicates pressures as low as 40 mm Hg. Pressures below 80 mm Hg are associated with tissue loss. The companion finding is dependent rubor. Distal arterioles lose their tone if chronically ischemic. The foot becomes red when dependent due to increased perfusion through dilated arterioles with the increase in

hydrostatic pressure. Peripheral pulses will not be palpable if occlusive disease is significant. Presence of a femoral pulse indicates good inflow but poor outflow (femoropopliteal or tibial disease).

The ankle/arm index (AAI)

An indication of the severity of the disease is given by the blood pressure at the ankle. Since systemic pressure various considerably over time, the ankle pressure is normalized to the systemic pressure, estimated by the brachial pressure. To accomplish this, blood pressure taken at the ankle is divided by the arm pressure, yielding the ankle/arm index (AAI), sometimes referred to as the ankle/brachial index (ABI).

$$\text{AAI} = \frac{\text{Ankle level arterial pressure}}{\text{Arm (systemic) pressure}}$$

Systolic arm pressure is obtained using a blood pressure cuff above the elbow and a hand-held Doppler to detect the return of flow in the brachial or radial artery. Prior to taking measurements, a period of five to ten minutes of supine rest in a warm room is optimal to allow the blood pressure to stabilize. The higher of the two arm pressures is used for calculation of the AAI in both legs since a low arm pressure indicates subclavian disease and is not a good indicator of true systemic pressure. For the ankle pressures, the cuff is placed as low as possible on the leg since disease below the level of the cuff is not detected (Figure 7). The patient must be supine to eliminate elevation of ankle pressure due to the hydrostatic column of blood (ankle pressure may increase by as much at 90 mm Hg when the patient goes from supine to standing just due to the hydrostatic component).

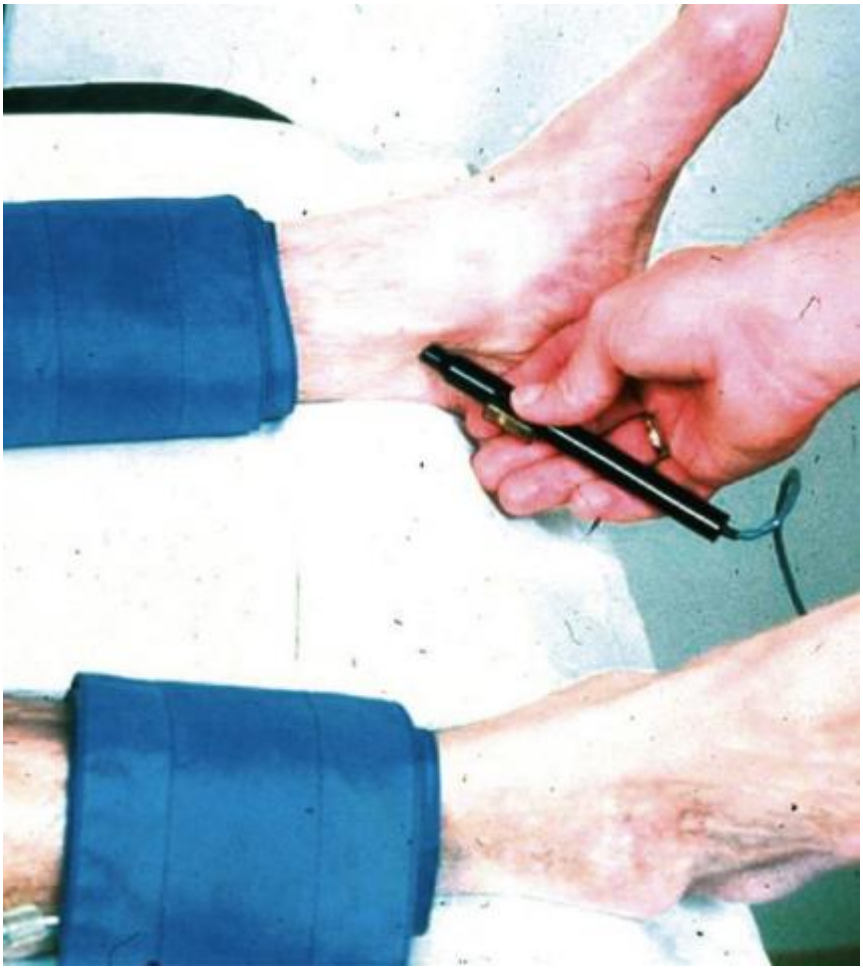

Figure 7

Ankle pressures are measured with the Doppler placed over the dorsalis pedis and over the posterior

tibial artery. In calculating the AAI, the higher of the two ankle pressures is used. The concept is that both of these arteries provide flow to the entire foot through the pedal arch, so adequate perfusion pressure in either is sufficient. However, in interpreting ankle pressures, the clinician must keep in mind that the pedal arch is not always complete. This is particularly true in diabetic and renal failure patients, who may have pedal and even digital artery occlusive disease. When signals cannot be obtained in either tibial artery, the examiner should attempt to measure an ankle pressuring using the Doppler over the medial branch of the peroneal artery, lateral to the dorsalis pedis artery, since this vessel may be preserved when the others are occluded (Figure 8).

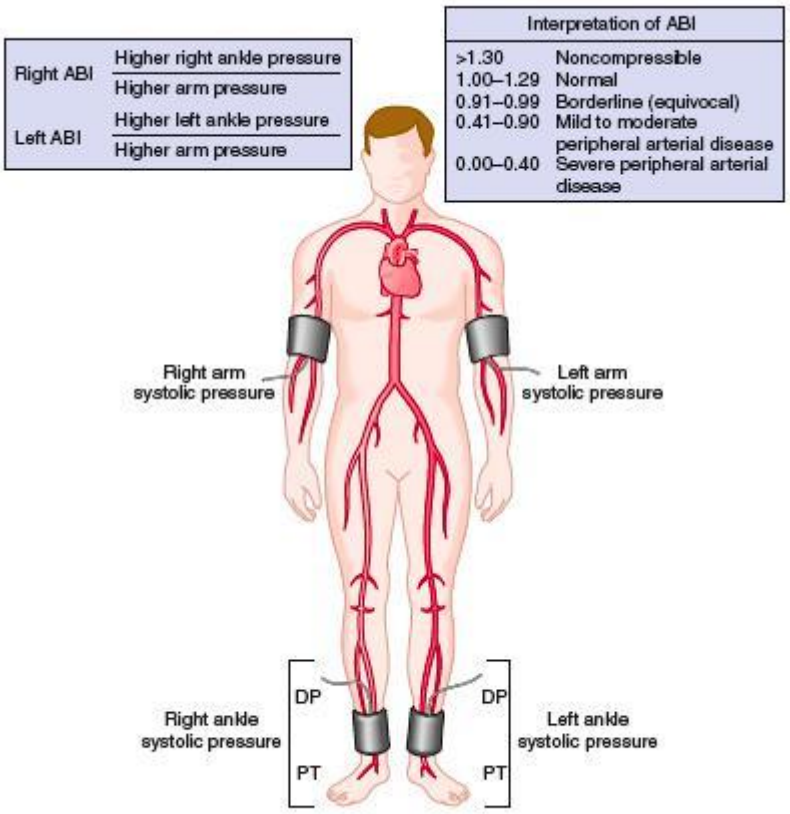

Figure 8

The normal peripheral artery velocity waveform is triphasic, with forward flow in systole, a brief reverse flow component in diastole due to reflected waves from the periphery, and then another forward component.

Following revascularization of an ischemic limb, the reverse component is lost due to hyperemia caused by arteriolar dilation with reduction in peripheral resistance. The waveform becomes monophasic with high velocities. This response can be demonstrated by listening to the hand-held Doppler over the radial artery when the hand is relaxed after clenching the fist for as little as thirty seconds.

If there is significant proximal obstruction, the velocity waveform is dampened and the signal at the ankle will become biphasic and with a greater degree of narrowing, monophasic (Figure 9). These waveform differences can be appreciated while listening to the Doppler signal or by displaying the waveform visually over time.

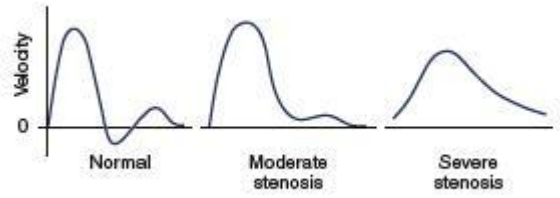

Figure 9

Significant atherosclerotic lesions (those that narrow the lumen diameter by >50%) produce a pressure gradient across the stenosis. A drop in the systolic pressure is a relatively good indication of the presence of a proximal arterial stenosis or occlusion.

The pressure drop indicates loss of energy, and in large arteries with mild to moderate disease, it is caused by turbulence that increases with increasing flow and velocity. Thus a lesion that is not hemodynamically significant under resting conditions (i.e. there is no measurable pressure drop distal to the stenosis) may become significant with the increased flow demands of exercise. This is the rationale for measuring ankle pressures before and after exercise (e.g., treadmill testing, Figure 10). Intermittent claudication from peripheral vascular occlusive disease causes a drop in ankle pressures (typically >20 mmHg) at the conclusion of the test, whether it was stopped due to symptoms or completion of the entire exercise period. Lack of a pressure drop reliably rules out the diagnosis. It is important for the examiner to note symptoms, time of their onset, and total time the patient was able to exercise. For example, if a patient stops primarily due to shortness of breath after only a few minutes of exercise, he or she is unlikely to benefit from revascularization even if there is associated calf pain. Thus, a treadmill exercise test is the best method for diagnosing significant lower extremity atheroscleosis as a cause for leg pain versus other causes, such as pseudoclaudication.

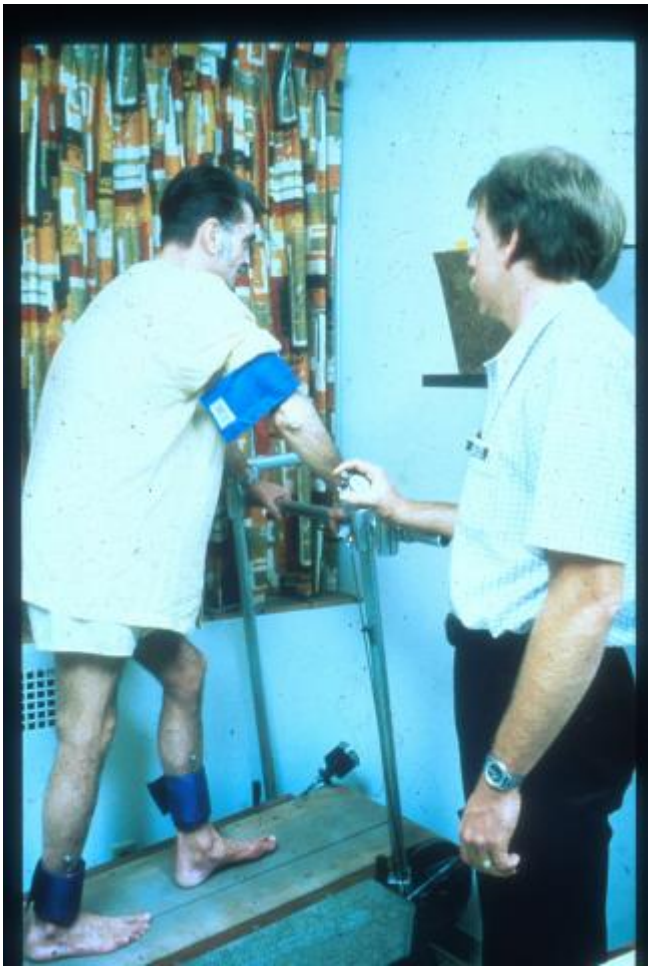

Figure 10

The level and degree of disease correlates with the degree to which the AAI is diminished (Figures 11 and 12).

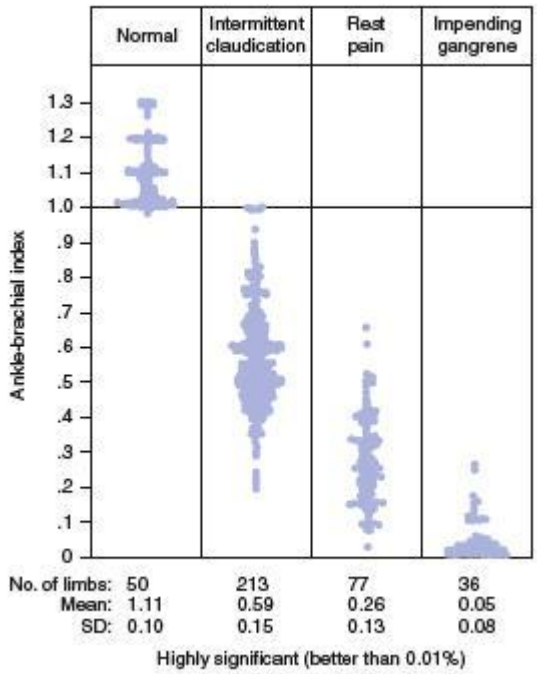

Figure 11

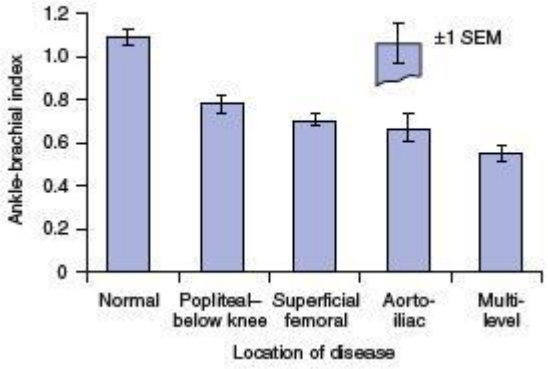

Figure 12

Interpretation of the AAI:

a. The normal AAI is ≥ 1.0 or (100%) (1.00 ±.010), because normal systolic arterial pressures at the ankle are higher than central systolic pressures (Figure 13). This occurs because of the properties of the elastic and muscular arteries; the mean pressure is lower at the ankle than it is centrally. Patients with mild claudication may have normal ankle pressures at rest, but these will drop with exercise.

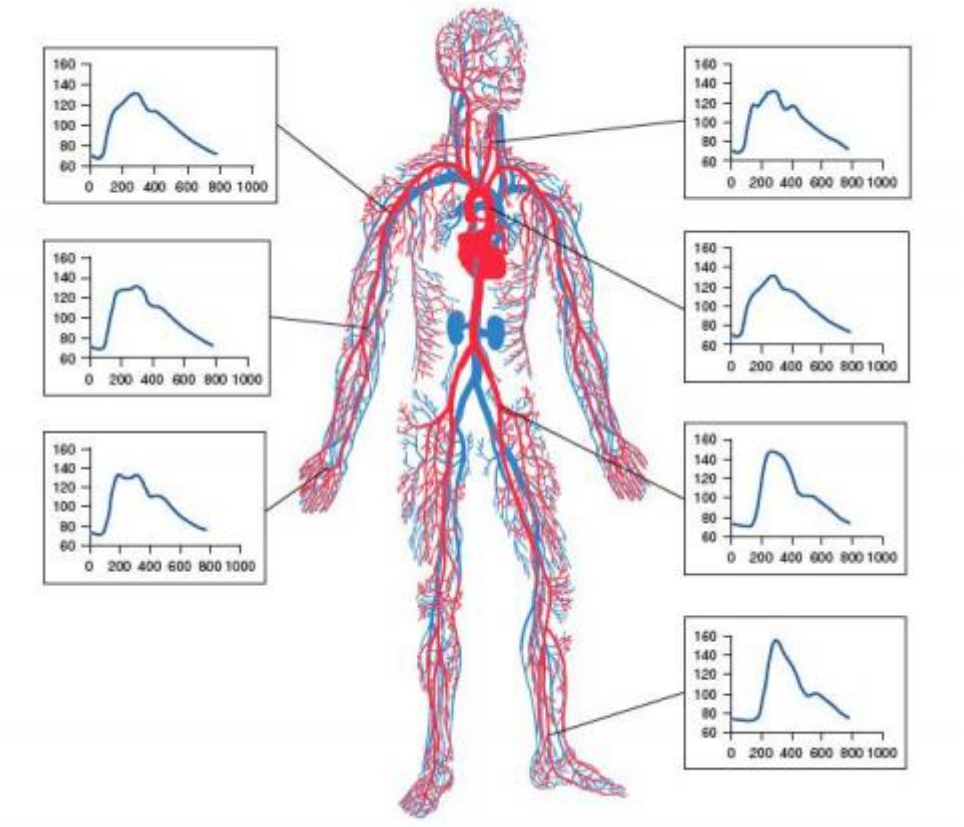

Figure 13

b. AAIs of 0.50 to 0.90 are associated with moderate arteriosclerosis, usually at a single level. These patients usually claudicate with exercise (and their ankle pressures will drop further).

c. AAIs of 0.30 to 0.50 are associated with severe occlusive disease, usually with multi-level disease. These patients will claudicate (decrease ankle pressures) with exercise and will have prolonged recovery

times (≥10 minutes).
d. AAIs of less than 0.30 are indicative of severe ischemia. These patients will generally have rest pain, impaired healing, and face the threat of limb loss without revascularization.
e. Absent ankle signals (AAI = 0) may indicate acute or advanced chronic ischemia.
Example:

Resting Systolic Pressures	Right	Left
Brachial Arteries	148	154
Posterior Tibial Arteries	0	160 (triphasic)
Anterior Tibial Arteries	84 (mono)	152 (triphasic)
Ankle/Arm Index	0.56	>1.0

a. There is a slight gradient between the arms, so use the higher value for both AAIs.
b. Right leg: the PTA is not audible, indicating probable occlusion, so use the ATA. Therefore, right AAI = 84 / 154 = 0.56
c. Left leg: the signals are both triphasic, so use the higher (PTA). Left AAI = 160 / 154 = 1.03

The AAI either resting or with exercise is not reliable if tibial vessels are stiff and incompressible due to medial calcinosis (an abnormal deposition of calcium in the media of the arteries). These patients will still have audible Doppler flow signals at the ankle at cuff pressures approaching or exceeding the brachial pressure. Medial calcinosis is most commonly associated with diabetes, but may also occur with renal failure or chronic steroid therapy. Elevated AAIs (≥ 1.3) are characteristic of this condition, but pressure may be in the normal range, giving false negative results. This should be suspected when normal readings are associated with abnormal, monophasic, waveforms, absence of palpable pulses, and presence of typical signs and symptoms of PVD. In this circumstance, pressures measured at the toe level with digital cuffs and a Doppler or PPG flow detection device are often useful. A toe/brachial index can be calculated. Toe pressures are normally 20 to 40 mm Hg less than ankle pressures. A TBI of 0.7 is normal. Pressures of at least 40 mm Hg are needed for healing, probably more in diabetic patients (Figure 14).

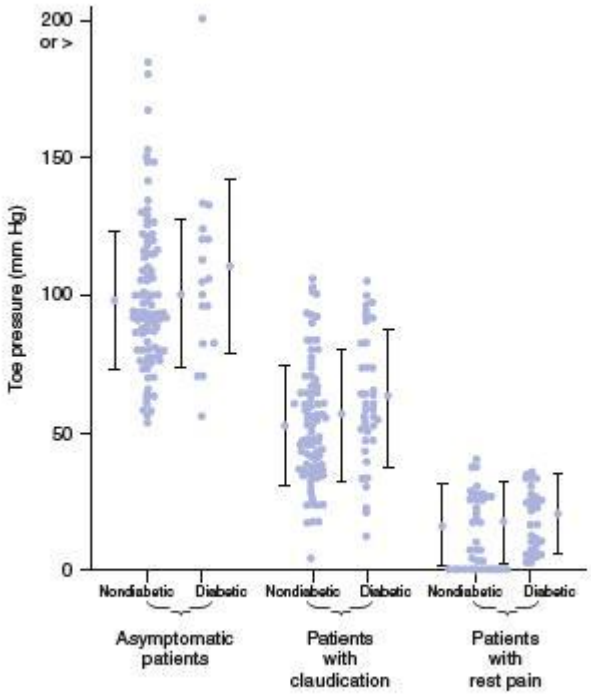

Figure 14

AAIs are often obtained during follow-up for patients who have had an intervention. However, this test has significant variability and so is of limited value in detecting early restenosis. Generally, a change in a measured value is statistically significant if it exceeds two standard deviations. For the AAI, this means that a difference in AAI of at least 0.15 is required to be considered to be truly significant.

Duplex scanning

Duplex scanning combines a pulsed Doppler with ultrasound imaging. It was developed at the University of Washington in the 1970's by Dr. Eugene Strandness and coworkers in bioengineering. The Doppler signal can be steered to point anywhere in the visualized vessel. Varying the time delay from transmission to reception determines the depth from which the reflected signal is obtained. In this way velocity information can be obtained from specific locations along the visualized artery. As noted, waveforms from normal arteries are triphasic (Figure 15). Stenosis causes an increase in velocity at the site of the narrowing and dampening of the signal beyond it (Figure 16). There is also flow disturbance beyond the stenosis: a widening of the velocity waveform due to non-uniform flow. Thus, duplex scanning can assess the location of arterial lesions throughout the lower extremity vasculature. The degree of stenosis can be estimated by the extent of increase of velocity, presence or absence of reverse flow, and post-stenotic turbulence (Figure 17, also, see Table 5 for the criteria used by the University of Washington Vascular Laboratory).

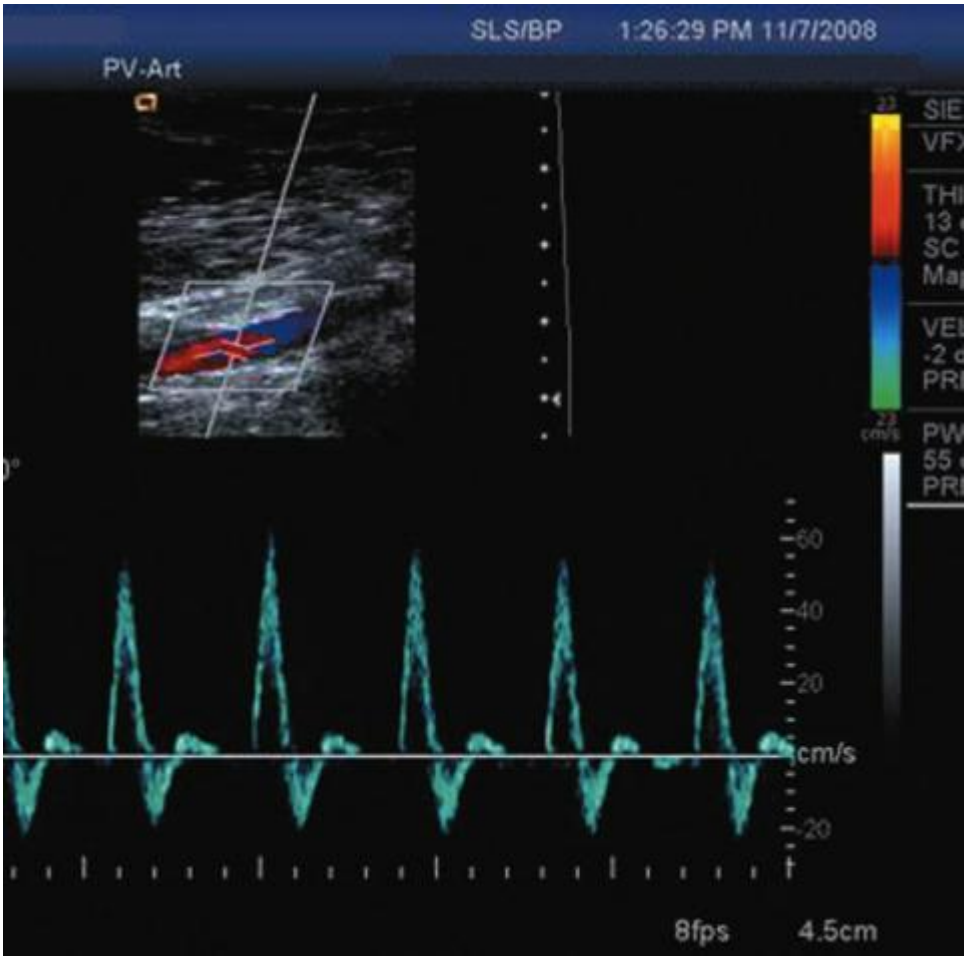

Figure 15

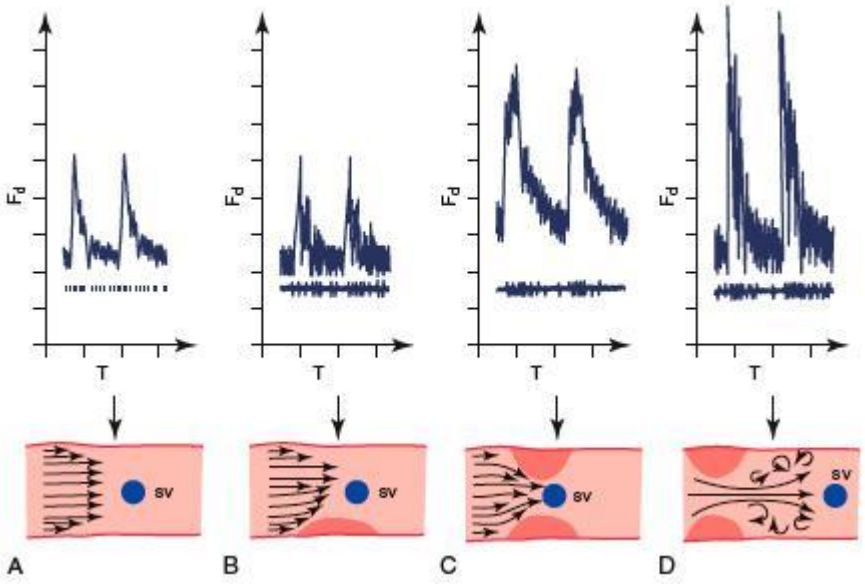

Figure 16

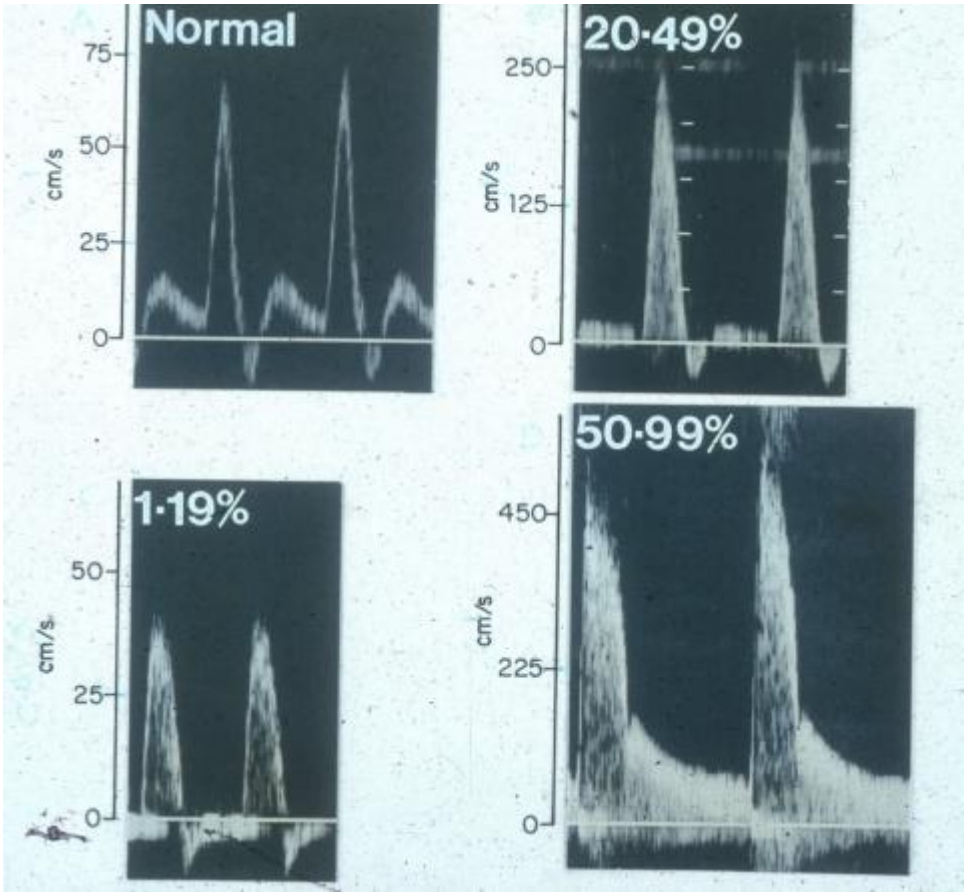

Figure 17

% DIAMETER REDUCTION	WAVEFORM CRITERIA
NORMAL	No evidence of systolic velocity increase Clear window under the systolic peak Reverse flow is present
1 to 19	No evidence of systolic peak increase Spectral broadening present Reverse flow is present
20 to 49	Peak systolic velocity increase (but not doubled) Reverse flow present No evidence of post-stenotic flow
50 to 99	Peak systolic velocity increase (more than double from previous arterial segment) Absence of reverse flow Post-stenotic flow present
Occluded	No detectable Doppler signal

Table 5: Duplex criteria for estimation of degree of peripheral stenosis

Duplex scanning is the preferred method for follow-up of reconstructions such as bypass grafts, stents, and angioplasty. Stenosis in lower extremity vein bypass grafts causes a velocity increase at the site of stenosis and if significant, a reduction in flow that is reflected in a decrease in the velocity along the non-stenotic portion of the graft. Work by Bandyk and others has shown that grafts are in danger of occlusion if the peak systolic velocity at the stenosis is more than 400 cm/s or is more than 3.5 times that of the normal segment and also if the mid-graft peak systolic velocity is less than 50 cm/s.

Treatment

Very few patients who present with claudication go on to limb loss (only about 10 percent over ten years). Treatment of these patients is elective and based on their degree of disability. Revascularization

(by standard open surgical bypass or by endovascular methods such as balloon angioplasty) is reserved for patients with disabling claudication. In contrast, revascularization is urgent in patients with critical limb ischemia (threatened limb), which is indicated by rest pain or tissue loss (gangrene or ulcers on the feet that are slow to heal).

Inflow disease is always treated before outflow disease and treatment of larger arteries (aortoiliac) is always more durable than treatment of smaller vessels (femorotibial). Since the diagnosis of arterial disease depends on history and physical examination and the decision to revascularize a patient depends on the degree of disability and the presence of rest pain or ulceration, duplex scanning or other imaging is not required to make this decision. These tests are useful for planning intervention and for following bypass grafts after intervention to detect narrowing due to intimal hyperplasia before such narrowing can cause graft failure.

Outcome depends on the severity of the ischemia and control of risk factors. Most patients will not require revascularization and can be managed medically. Control of risk factors, including aggressive lowering of cholesterol with statin therapy and administration of aspirin are important particularly since up to half of the patients with symptomatic PVD die of coronary events over five years. Smoking cessation and a supervised exercise program are the interventions of choice for claudication. Drug therapy is not very effective. Pentoxyphylline, once widely used, has been shown to be ineffective. Cilostazol, a phosphodiesterase inhibitor, vasodilator, and inhibitor of platelet aggregation can improve walking distance. If revascularization is required, this may be accomplished by endovascular treatment (balloon angioplasty, stenting, stent grafting, or atherectomy) or surgical procedures such as endarterectomy or bypass using prosthetic grafts for large arteries (aorta, iliac, and above-the-knee femoral), or vein (preferred for below-the-knee bypasses).

Buerger’s disease

Buerger’s disease (thromboangiits obliterans) is a rare form of chronic peripheral vascular occlusive disease. It is an inflammatory process of medium and small vessels associated with smoking addiction. It is more common in the Middle East and Far East. Young men (age 20 to 40) are most commonly afflicted. Occlusion of very distal tibial or digital arteries causes ulcers of the tips of fingers and toes and can lead to loss of digits. These findings are often associated with superficial thrombophlebitis. Peripheral pulses may be palpable at the wrist or ankle and the AAI may be normal despite severe occlusive disease more distally. The pathology reveals inflammation across the artery and its associated vein and nerve. The clinical diagnosis is made on the basis of the angiogram, which reveals cork-screw arteries running along the course of occluded distal vessels. These are enlarged vaso vasora. Buerger’s disease is caused by smoking in susceptible patients and can be arrested by cessation of smoking. However, most Buerger’s disease patients are strongly addicted to smoking and are unable to quit even as they lose digits from the disease.

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