REVIEW

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Update on peripheral vascular diseases: From smoking cessation to stenting

ABSTRACT

Newer pharmacologic agents including gene therapy hold promise for the treatment of atherosclerotic peripheral vascular disease (PVD), as do advances in endovascular revascularization techniques. Nonetheless, the two most important treatments remain the same: stopping smoking and starting a walking program.

KEY POINTS

Atherosclerosis is the most common cause of PVD, and patients with PVD have a very high risk of cardiovascular and cerebrovascular events.

The most common symptom of PVD is intermittent claudication. The initial treatment for claudication consists of aggressive risk-factor modification, exercise, lipid-lowering agents, antiplatelet agents, and cilostazol.

Endovascular therapy is the treatment of choice in focal aortoiliac, iliac, and femoropopliteal disease after a trial of medical therapy.

An appropriate management strategy for acute arterial occlusion is thrombolysis followed by definitive correction of the underlying lesion using either percutaneous angioplasty or surgery.

Stopping smoking can reduce the 5-year amputation risk tenfold and decrease the mortality rate by 50%.

PATIENT INFORMATION
Peripheral vascular disease (PVD), page 734

FOR MOST PATIENTS with peripheral vascular disease (PVD), the real nemesis is not the PVD per se but coronary artery disease. Atherosclerosis is the most common cause of PVD, and studies suggest that patients with PVD have a rate of cardiovascular mortality three to five times higher than do age-matched controls.[1]

Percutaneous angioplasty and surgery can restore blood flow and relieve the symptoms of PVD, but for most patients the treatment is medical and should include aggressive risk-factor modification.

In particular, patients with PVD need to hear five words of low-tech advice: stop smoking and start walking.

PVD IS COMMON

PVD is common, with an age-adjusted prevalence of 12% to 20% of adults.[2,3] The ratio of men to women is approximately equal.

ONSET IS USUALLY GRADUAL

The symptoms of PVD of the lower extremities usually begin quite gradually. In fact, persons with PVD are often unaware of subtle early symptoms, and some therefore do not seek medical care until the disease is advanced. On the other hand, thrombosis in situ (acute occlusion) superimposed on preexisting disease has a more acute or "acute-on-chronic" presentation.

SYMPTOMS

Available data suggest that approximately 50% of patients with PVD experience no symptoms, 45% have intermittent claudication,

TABLE 1

Differences between true claudication and pseudoclaudication

FEATURE	CLAUDICATION	PSEUDOCLAUDICATION
Onset	On exertion	Standing, walking
Character	Crampy, ache	Paresthetic, sharp
Bilateral	Sometimes	Usually
Walking distance	Constant	Variable
Etiology	Vascular	Spinal
Relief	Standing	Sitting, leaning forward

and only a small minority present with ischemic pain at rest, ulceration, or gangrene.[4]

Intermittent claudication, the most common symptom of PVD, is usually described as a progressive aching or cramping sensation that occurs with walking but diminishes abruptly with rest or even while standing.

This symptom typically occurs in the muscle group distal to an arterial obstruction, most commonly in the calf or distal thigh as a result of occlusion of the superficial femoral artery at the adductor canal. However, symptoms may occur in the thigh, hip, and buttock if the obstruction is in the aortoiliac segment or internal iliac artery. Patients who complain only of foot discomfort likely have small-vessel occlusive disease, which can occur in diseases such as thromboangiitis obliterans (Buerger disease).

Mortality in symptomatic PVD is 50% in 10 years

The typical site of the obstruction varies with age. In patients younger than 40 years the most common sites are the aorta and the iliac arteries, whereas in those over 40 the obstruction in is the femoral or popliteal arteries in 65% of cases.[4]

Intermittent claudication should be distinguished from so-called *pseudoclaudication*, which is caused by lumbar spinal canal stenosis. Features that distinguish true claudication from pseudoclaudication are found in TABLE 1.

Pain at rest occurs when typical intermittent claudication progresses to a critical level of leg ischemia. Most commonly, the pain occurs at night when the patient is supine. Patients with ischemic pain at rest typically dangle the affected foot over the side of the bed or actually get up and walk about seeking relief. Most patients with pain at rest have multilevel arterial involvement.

Ulcers. As the disease progresses further over time, patients with ischemic pain at rest may develop "kissing ulcers": ischemic necrosis between two toes. Nonhealing ulcers or dry gangrene after minor trauma to the foot is another common presentation of underlying severe PVD.

Disuse atrophy occurs in patients who cannot walk and may lead to a significant loss of muscle mass in the lower extremity. This form of muscle atrophy often causes difficulty with rehabilitation after revascularization and can be a major impediment to physical therapy.

MORTALITY IS INCREASED

PVD follows a fairly benign course in most patients except for those with diabetes or those who smoke. As collateral vessels develop, symptoms remain stable or improve with time in 65% to 70% of patients, and fewer than 25% ever need surgery or angioplasty.[4,5] There is a low risk of losing a limb—PVD progresses to critical limb-threatening ischemia in only 1.4% of patients per year.

However, patients with diabetes and smokers have a higher overall amputation risk. In addition, symptomatic PVD in patients younger than 50 years generally follows a particularly virulent course.[6]

As noted above, most of the morbidity and mortality in persons with PVD is due to cardiovascular and cerebrovascular events. The average life span is shortened by 10 years depending on the status of the cardiovascular system. The worst prognosis is in diabetic patients who smoke.

The mortality rate increases with the severity of PVD, as assessed by the ankle-brachial index (ABI—see below for explanation)[7–10] or by symptoms.[11] Symptomatic PVD carries at least a 30% risk of death within 5 years and almost 50% within 10 years, primarily due to myocardial infarction (60%) or stroke (12%).[12] Even patients with asymptomatic PVD (an ankle-brachial index < 0.9) have a twofold to fivefold higher risk of cardiovascular events.[12]

HOW TO ASSESS CARDIOVASCULAR AND CEREBROVASCULAR RISK

Because cardiovascular events are the most common adverse outcomes in patients with PVD, the physician should assess the patient's risk for coronary artery disease. The assessment should cover both conventional and nonconventional risk factors, including:

- Blood pressure
- Family history of coronary artery disease
- Lipid profile
- Smoking history
- Presence of diabetes and status of its control
- Homocysteine level.

If coronary artery disease is strongly suspected, a functional study (eg, exercise or pharmacologic stress thallium) is indicated to document any reversible coronary ischemia. In patients who have symptoms of coronary disease, routine coronary angiography may also be considered.

Patients with symptomatic intermittent claudication and a clinical suspicion of carotid atherosclerosis should also be referred for duplex ultrasonography of the carotid arteries. A compelling reason for this recommendation is that only 40% of patients with significant internal carotid artery stenosis actually have an audible cervical bruit.[13]

HOW TO EVALUATE PVD

Clinical evaluation

The clinical history is extremely important in patients with PVD and should include:

- Whether symptoms developed acutely or gradually
- How far the patient can walk before the pain starts
- Whether the pain is relieved by standing
- Any risk factors that are present.

A detailed physical exam should be performed with emphasis on:

- The quality of the femoral, popliteal, dorsalis pedis, and posterior tibial pulses
- Signs of arterial insufficiency, eg, coolness, scaling, paleness (especially with leg elevation), and ulceration
- Systemic illnesses such as hyperlipidemia and diabetes
- The ankle-brachial index (ABI).[14]

The ankle-brachial index (ABI). All physicians providing routine care to patients with coronary or carotid atherosclerosis should be able to measure the ABI: the blood pressure in a pedal artery (usually the dorsalis pedis or posterior tibial artery, sometimes the peroneal) divided by the higher of the systolic pressures in the brachial arteries of the two arms. The ABI cannot pinpoint the area of stenosis but is a very accurate and simple measure of the severity of lower extremity atherosclerosis. TABLE 2 shows a grading scale for the severity of PVD based on the ABI during exercise and at rest.

Only 40% of patients with significant carotid stenosis have a bruit

When to refer the patient to a vascular specialist

Patients should be referred to a vascular medicine specialist for an assessment if they have any of the following:

- Lifestyle-limiting claudication
- Any sign of potential critical limb ischemia, such as foot or limb ulceration, skin changes (nail or skin atrophy, dependent rubor), or gangrene
- An ABI less than 0.50 at rest
- An incompressible ankle artery (systolic ankle pressure > 300 mm Hg); incom-

TABLE 3

Ultrasound criteria for diagnosis of significant lesions in the carotid, renal, mesenteric, and peripheral arteries*

ARTERY	ULTRASOUND CRITERIA
Carotid	Peak end-diastolic velocity > 135 cm/sec,† stenosis > 80% Peak end-systolic velocity > 240 cm/sec,† stenosis > 80%
Renal	Peak systolic velocity > 180 cm/sec, stenosis > 60% End-diastolic velocity > 150 cm/sec, stenosis > 80% Renal artery ratio‡ > 3.5, stenosis > 60%
Celiac trunk	Peak systolic velocity > 250 cm/sec, stenosis > 70%
Superior mesenteric	Peak systolic velocity > 275 cm/sec, stenosis > 70%, failure to increase postprandial velocity by at least twice compared to fasting velocity
Inferior mesenteric	Not reliably seen in most patients In hypertrophied vessel or in cases of celiac and superior mesenteric artery occlusion, duplex pattern mimics that of superior mesenteric artery with postprandial increase in velocity
Peripheral arteries	Peak systolic velocity > 200 cm/sec, or peak systolic velocity double the proximal adjacent segment, monophasic waveform

*Based on criteria used at the Cleveland Clinic
†Carotid velocities may be artificially elevated in the case of contralateral occlusion, thus overestimating stenosis
‡The peak systolic velocity in the renal artery divided by the peak systolic velocity in the aorta

Exercise tests are better than studies done at rest

pressible ankle arteries suggest significant medial wall calcification and likely reflect significant PVD
- Blood pressure more than 75 mm Hg higher in the ankle than in the arm.

Specialized tests

Pulse volume recordings are very useful for the noninvasive study of limb perfusion. They measure changes in the volume of the limb throughout the cardiac cycle. Since the volume of muscle, bone, fat, skin, and venous blood remains relatively constant over time, changes in volume in the resting limb reflect changes in arterial flow.

When performing a pulse volume recording, appropriately sized cuffs are placed at various levels of the leg, starting from proximal to distal: first at the thighs, then above and below the knees, above the ankles, and at the transmetatarsal and first toe levels. Pressures and waveforms are measured at one level and then at the next level down to the toes by inflating the cuff to either a known pressure or a known volume. If an arterial occlusion is present, pulsatile flow is lower in segments distal to the occlusion. Vascular calcification does not affect the reliability of the pulse volume recording.

Pulse volume recordings are especially useful in two situations:
- In determining the need for revascularization in diabetic patients with a foot ulcer or gangrene and a spuriously elevated ABI due to medial calcification.
- In detecting improvement or deterioration before, during, and after vascular surgery or angioplasty.

Duplex ultrasound scanning, as the name implies, has two major components: B-mode imaging and pulsed Doppler frequency spectral analysis. The B-mode shows the anatomy and can identify the narrowed segment or segments of vessels for the Doppler exam to focus on. The pulsed Doppler measures the blood velocity past the stenosis. Experimental observations and direct clinical correlations show that arterial occlusive lesions of progressive

severity are associated with characteristic changes in velocity in systole and diastole. Thus, by measuring velocity at specific points in the cardiac cycle, such as the peak systolic velocity or the end-diastolic velocity, one can estimate the severity of a focal arterial occlusive lesion.

TABLE 3 enumerates the duplex ultrasound criteria for the diagnosis of significant lesions in the carotid, renal, and mesenteric arteries.

Exercise testing is commonly used to evaluate cardiac performance because it provides a much better index of myocardial perfusion than studies performed at rest. The principles are similar for the lower extremities.

In using exercise to assess intermittent claudication, one measures the time the patient can walk and the changes in the ankle systolic blood pressure in response to walking. A patient in whom the ankle systolic blood pressure drops markedly within 2 minutes of starting to walk is likely to have more severe disease than a patient in whom the blood pressure drops by the same amount after 7 minutes of walking. In contrast, the ankle systolic pressure decreases very little or not at all in a healthy person walking at approximately 1.5 to 2 miles per hour for 5 to 7 minutes.

True intermittent claudication does not occur without a decrease in the ankle systolic blood pressure and a decrease in the pulse volume amplitude. This is particularly important to remember when trying to determine if a patient has pseudoclaudication (related to lumbar spinal canal stenosis) vs true intermittent claudication.

Magnetic resonance angiography (MRA) is now used to evaluate the aorta and the carotid, renal, and lower-extremity arteries. The MRA signal is a reflection of the velocity and flow patterns of moving protons within the bloodstream. A major advantage of MRA is that it does not require catheters or contrast agents. A disadvantage is that MRA overestimates the severity of lesions, and a high-grade stenosis may create a long signal void and appear as an occlusion. This is especially important in carotid stenosis, in which a distinction between high-grade stenosis and occlusion is crucial.

Angiography remains the gold standard to determine the severity and extent of PVD. Digital substraction technology can give high-quality images using small amounts of contrast material. However, angiography is invasive and is indicated only in patients in whom revascularization is being considered. This includes patients with lifestyle-limiting claudication, pain at rest, ischemic ulceration, or gangrene, and patients with diabetes and intermittent claudication (who have a high incidence of limb loss).

MEDICAL THERAPY

Risk-factor modification

Treat hypertension. The higher the blood pressure the greater the risk of claudication. In men and women age 65 and older in the Framingham study,[15] the relative risk of claudication was 1.27 if the systolic blood pressure was 20 mm Hg higher than normal and 1.62 if it was 40 mm Hg higher. Thus it is imperative to control blood pressure adequately. Strict control of hypertension slows the progression of PVD and reduces cardiovascular events.[16]

There is no consensus about what type of antihypertensive drugs to use in patients with PVD. Beta-blockers used to be avoided, but most experts now believe that their benefits outweigh the risk. Based on the HOPE trial,[17] angiotensin-converting enzyme (ACE) inhibitors would be the agents of choice.

Stopping smoking reduces the mortality rate by half

Treat elevated lipid levels. Hypercholesterolemia doubles the incidence of intermittent claudication and is found in as many as 50% of patients with PVD.[18] Angiographic studies have confirmed that lipid-lowering retards the progression of femoral atherosclerosis,[19,20] and HMG-CoA reductase inhibitors have been shown to reverse the progression of carotid atherosclerosis.[21,22] All patients with PVD and elevated lipid levels should be on lipid-lowering therapy, and the target LDL level should be less than 100 mg/dL.

Encourage patients to quit smoking. The progression of peripheral vascular atherosclerosis is significantly greater in patients who continue to smoke. In a study in Sweden,[23] the incidence of myocardial infarction 10 years after the diagnosis of claudication was

11% in former smokers and 53% in active smokers, and the 10-year overall survival rates were 82% in former smokers vs 42% in active smokers. Complete cessation of tobacco use should be the goal. Stopping smoking can reduce the 5-year amputation risk tenfold and decrease the mortality rate by 50%.[24–27]

Control diabetes. The combination of diabetes mellitus and PVD is ominous, because PVD rapidly progresses to ischemic pain at rest and ulceration in these patients.[28] Persons with claudication and diabetes have an overall amputation risk of 20% and a 5-year mortality rate of up to 50%. Among the risk factors for amputation in diabetic patients are neuropathic symptoms and lack of outpatient diabetes education[29]; therefore, patient education should be integrated in the evaluation of PVD.

Optimal glycemic control should be a consideration. Large-scale studies have demonstrated that optimal glycemic control decreases the microvascular complications of diabetes such as retinopathy and nephropathy, but failed to show any effect on macrovascular complications such as coronary artery disease and PVD. Nevertheless, optimal glycemic control should be attempted.

Tell patients to walk until the pain starts, rest, and then keep walking

Walking program

A regular walking regimen is extremely helpful. Patients should walk at least three times a week (preferably every day) for 30 to 45 minutes, and keep up this regimen for at least 6 months. They should walk as far as possible using near-maximal pain as a signal to stop, and resume walking when the pain goes away.

In supervised exercise programs, sessions typically last 60 minutes and are monitored by a skilled nurse or technician. Patients walk on a treadmill initially set to a speed and grade that bring on the pain of claudication within 3 to 5 minutes. Patients walk at this rate until they experience claudication of moderate severity, rest until the claudication abates, and then resume walking. This repeated on-and-off exercise is continued throughout the supervised rehabilitation setting. At home, patients are encouraged to continue walking primarily on a treadmill so that the intensity of the workout can be controlled.

Patients should be reassessed clinically every week while in a supervised program. As they are able to walk further and further at their chosen workload, the speed or grade or both should be increased. This scenario will induce a training benefit.[30] Patients can walk 180% to 400% farther with this regimen. There is some evidence that older age, femoropopliteal disease, and more aggressive exercise sessions predict better response to treatment.[31]

Pharmacologic therapy

Antiplatelet agents reduce both the risk of limb loss and the need for surgical revascularization in patients with intermittent claudication.[32,33] Antiplatelet therapy also substantially reduces the risk of myocardial infarction, stroke, or death in patients with PVD.[31]

All patients should take aspirin unless it is contraindicated. Newer antiplatelet agents such as ticlopidine (Ticlid) and clopidogrel (Plavix) also have beneficial effects in patients with PVD.[34] Both ticlopidine and clopidogrel are adenosine diphosphate antagonists and have similar mechanisms of action, but ticlopidine has significant adverse effects such as neutropenia and thrombotic thrombocytopenic purpura that limit its use.

Pentoxifylline (Trental, Pentoxil) has several actions. It relaxes vascular smooth muscle, causing vasodilation. It also inhibits cyclic AMP phosphodiesterase and stimulates prostacyclin formation, inhibiting platelet aggregation. And it increases the deformability of erythrocytes and leukocytes, decreasing blood viscosity.

Unfortunately, only about 20% of patients benefit from pentoxifylline, but a trial of 2 to 3 months in most patients is reasonable.[14,35] Patients most likely to benefit include those with symptoms lasting more than a year and an ABI less than 0.8.

Cilostazol (Pletal), a newer antiplatelet and vasodilating agent, significantly increased the distance patients with claudication could walk at all measured time points in a study in 239 patients.[36] This agent may be more effective than pentoxifylline for patients with intermittent claudication.[37] However, it is a phosphodiesterase inhibitor and is absolutely contraindicated in patients with congestive heart failure and an ejection fraction less than

40%, because phosphodiesterase inhibitors have been shown to increase mortality in patients with congestive heart failure.

Revascularization

Revascularization (ie, percutaneous angioplasty or surgery) is indicated for lifestyle-limiting claudication, which includes pain at rest, ischemic ulceration, and gangrene.

Revascularization is also indicated for diabetic patients with moderately severe or severe intermittent claudication. It is appropriate to have a lower threshold for performing revascularization in diabetes because patients with diabetes have a higher incidence of limb loss. Diabetic patients also may have a blunted sensation of claudication pain, owing to neuropathy.

Several studies have now documented the benefit of angioplasty in critical limb ischemia.[38,39] The indications for revascularization in patients with stable intermittent claudication are less well-defined. Whyman et al[40] randomly assigned 62 patients with intermittent claudication to angioplasty plus medical treatment or medical treatment alone. At 6 months, patients treated with angioplasty had a better outcome, being able to walk an average of six times farther on the treadmill and having lower pain scores.

Choosing between angioplasty and surgery

In general, angioplasty is favored for shorter lesions, while surgery is reserved for chronic long-segment occlusions and after failure of angioplasty (TABLE 4). FIGURE 1 shows the long-term patency rates after angioplasty or surgery in the lower extremity.[14]

For aortic or iliac disease, angioplasty and stenting is the initial treatment of choice for focal lesions (< 5 cm in the iliac artery), with a technical success rate of 90%, an angiographic patency rate of 73% at 2 years, a complication rate of less than 10% (mostly access site-related and minor), and a mortality rate of less than 1%. A meta-analysis of six published angioplasty series and eight stent placement studies suggested that stents significantly improve the outcome of iliac angioplasty.[41]

For femoral or popliteal disease, balloon angioplasty should be considered after a trial of medical therapy, provided there is only one short lesion and there are no adverse factors such as diabetes or concomitant tibial artery disease.[14,42] Stenting should be used selectively for short, focal lesions (< 10 cm). FIGURE 2 demonstrates successful angioplasty and stenting of a lesion in the superficial femoral artery.

Hunink et al[43] examined the use of angioplasty or bypass surgery for femoropopliteal disease by decision analysis. On the basis of improvement in quality of life and cost-effectiveness, they concluded that angioplasty is the preferred initial treatment for claudication due to femoropopliteal stenosis and for occlusion after a trial of medical therapy.

For tibial disease, long-term medical therapy is appropriate. Surgical revascularization carries a lower patency rate and a higher amputation risk in tibial disease than in other arterial segments,[44,45] though better results have recently been reported.[46,47] Endovascular interventions have a limited role in tibial disease, although encouraging preliminary results have been reported.[48]

Consider angioplasty for short lesions, surgery for longer lesions

Cardiac risk with vascular surgery

Peripheral vascular disease is a marker for coronary artery disease. Even in patients in whom coronary artery disease is not suspected, elective vascular surgery is an independent significant risk factor for myocardial infarction (adjusted odds ratio 3.72; 95% confidence interval 1.12 to 12.37), and is consid-

TABLE 4

Clinical variables favoring angioplasty and those favoring surgery

Variables favoring angioplasty

Short occlusions
- < 2 cm in the tibial artery
- < 5 cm in the iliac artery
- < 10 cm in the superficial femoral artery

Stenosis uncovered after occlusions are treated with thrombolysis

Variables favoring surgery

Long occlusions
- > 5 cm in the iliac artery
- > 2 cm in the tibial artery
- > 10 cm in the superficial femoral artery

Stenoses adjacent to aneurysms

Lesions causing atheromatous embolism

Angioplasty or surgery? Long-term patency rates

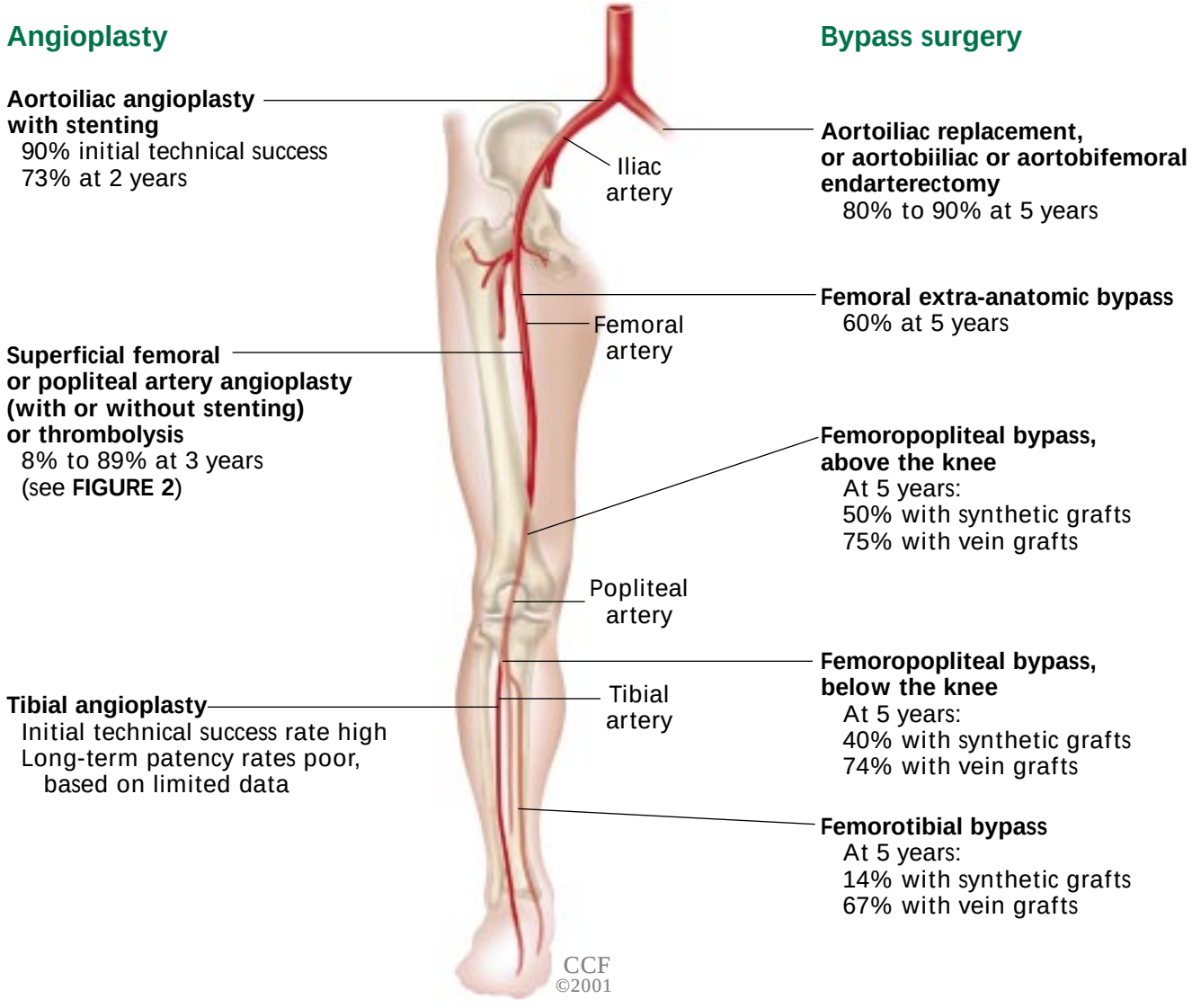

FIGURE 1. Long-term patency rates after revascularization.

FROM GRAY BH, SULLIVAN TM. VASCULAR CLAUDICATION: HOW TO INDIVIDUALIZE TREATMENT. CLEVE CLIN J MED 1997; 64:429–436.

ered high-risk surgery.[49] Clinical markers such as age greater than 70, angina, diabetes, arrhythmias, pathologic Q waves, and carotid bruits identify patients at higher risk.

Assessment of resting left ventricular function alone is not predictive of perioperative cardiac risk in vascular surgery. Therefore, noninvasive imaging assessment of cardiac risk, ie, with dipyridamole-thallium imaging, should usually be done prior to vascular surgery.

The promise of gene therapy

Improved understanding of the molecular biology of vascular diseases has brought new opportunities for therapeutic intervention at the molecular and genetic levels. The tools for genetic manipulation in vivo and our knowledge of potential molecular targets are still incomplete, but gene therapy is already being used experimentally and clinically to treat PVD.[50] Therapeutic angiogenesis has shown

promising results in early clinical studies as shown by improved clinical status and angiographic results.[51–58] The treatment of PVD remains limited by vascular proliferative lesions and the inability to modulate the progression of disease. Gene therapy, using angiogenic growth factors to stimulate growth of new blood vessels, may prove to be a new paradigm in the treatment of PVD.

MANAGEMENT OF ACUTE ARTERIAL OCCLUSION

Acute arterial occlusion presents with sudden onset (< 5 hours) and the "five Ps": pain, pallor, paresthesia (numbness), poikilothermy (coldness), and pulselessness.[59] It can either be thrombotic (suggested by previous occlusive disease in the involved limb, occlusive disease in other extremities, hematologic diseases, and arteritis) or embolic (suggested by presence of cardiac disease, left ventricular thrombus, atrial fibrillation, or proximal aneurysm).

Management includes intravenous heparin, confirmation of the diagnosis by angiography, and consideration of thrombolysis or surgical or percutaneous thromboembolectomy. A multispecialty working group[60] made the following consensus recommendations for patients with acutely occluded arteries:

- Intravenous heparin at full anticoagulant dosages should be given as soon as possible
- Intravenous thrombolytic agents should no longer be used because they are not effective
- Full imaging by angiography or duplex scanning should be performed
- Intrathrombus lysis, using a catheter to deliver a lytic agent directly inside the thrombus, should be initiated if a guidewire can be advanced across the thrombus; if the guidewire does not cross the thrombus, regional lysis, delivering the lytic agent above the thrombus, can be attempted for a limited time (4–6 hours)
- If a guidewire cannot be advanced after 4 to 6 hours of regional lysis, thrombolysis should be stopped and other treatment considered
- A management strategy incorporating intrathrombus or regional thrombolysis followed by definitive correction of the underlying lesion is appropriate for acute arterial occlusion. If there is clinical deterioration in the involved limb during regional lysis, the process should be aborted and definitive endovascular or surgical therapy initiated.

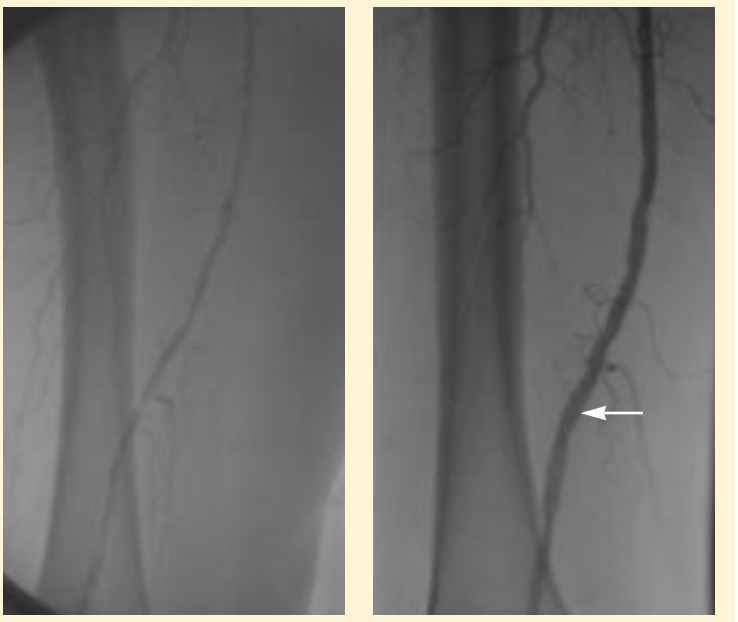

FIGURE 2. Successful angioplasty and stenting of a diffusely diseased superficial femoral artery. **Left,** before angioplasty; **right,** after angioplasty. Arrow points to stent.

The 5 Ps of acute occlusion: pain, pallor, paresthesia, poikilothermy, pulselessness

MANAGEMENT OF ANEURYSMS

The most common cause of aneurysms is atherosclerosis. Many patients with aneurysms also have coronary and carotid atherosclerosis. Other predisposing factors include hypertension, trauma, infection, and inflammatory diseases. Most aneurysms are asymptomatic, and complications are related to the site of the aneurysm (aortic rupture; femoral and popliteal embolism, thrombosis).

Common iliac aneurysms are usually associated with abdominal aortic aneurysms but may rarely occur in isolation. They may cause atheroembolism, obstructive urologic symptoms, groin or perineal pain, and iliac vein compression and may rupture.

Either computed tomography or magnetic resonance imaging is diagnostic, and surgery is

indicated if the aneurysm causes symptoms or is larger than 3 cm in diameter.

Popliteal artery aneurysms may be complicated by thrombosis, venous obstruction, embolization, popliteal neuropathy, popliteal thrombophlebitis, rupture, and infection. They are bilateral in 50% of patients, and 40% of patients have multiple aneurysms at other sites. The diagnosis is made by ultrasound, but angiograms are needed prior to surgical resection. Surgical treatment is usually indicated to prevent thromboembolic complications.

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