



Objectives:

After completing this CME, the reader should be able to:

- 1) define peripheral arterial disease.
- 2) comprehend the pathophysiology of peripheral arterial disease.
- 3) understand the risk factors for peripheral arterial disease.
- 4) differentiate the symptoms of P.A.D. from other conditions that share similar presentation.
- 5) encourage early detection of P.A.D. to empower podiatric physicians in the prevention of critical limb ischemia and other catastrophic events, such as heart attack and stroke.
- 6) recognize the financial impact of peripheral arterial disease, amputation, and limb preservation
- 7) apply knowledge of medical management of peripheral arterial disease into
- 8) understand the need of when to refer patients for endovascular or surgical intervention to prevent amputation and to

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Following this article, an answer sheet and full set of instructions are provided (p. 218).—**Editor**

By Desmond Bell, DPM

Introduction

Never as much as now has the team approach to wound management and limb salvage presented the opportunity for podiatric physicians to serve as the gatekeepers in the reduction of lower extremity amputation.

Allie reported that a three billion dollar annual saving to the U.S. healthcare system can be realized with a 25% reduction in lower extremity amputations.1,2

As podiatric physicians, we are positioned to identify the early and the advanced symptoms of periph-

eral arterial disease (P.A.D.) in our patients. The link between P.A.D. and coronary arterial disease makes recognition of P.A.D. symptoms not only important for potentially helping to reduce amputation rates, but also for preventing catastrophic events, such as heart attack and

stroke. There is an increased risk of myocardial infarction (MI), stroke, and cardiovascular death in patients with lower extremity P.A.D., including a 20% to 60% increased risk for MI and a two- to six-fold increased risk of death due to coronary heart disease events. The risk of cerebral vascular accident (CVA) is increased by 40% in this patient population.3

Medical-legally, we also find ourselves in the position where recognition of P.A.D. and pro-active intervention will not only be expected, but also necessary for better risk management. An article by Janov entitled "Seven Keys to Preventing Malpractice Lawsuits" included information regarding a case in Michigan where a jury awarded a patient \$1.23 million for a podiatrist's failure to refer to a vascular specialist in a timely manner. The patient of the podiatrist was initially treated for an ulcer that deteriorated within approximately a three week period and led subsequently to ischemic gangrene and a below-the-knee amputation. The jury rejected the podiatrist's defense which included "the patient had chronic, but stable symptoms," and "the patient did not need an urgent referral".4

The point here is that where P.A.D. is an underlying issue, especially where a wound is present, deterioration is likely to occur. Referral to the appropriate vascular specialist is not only critical, but is a standard of care. Failure to refer a P.A.D. patient to a specialist can have tragic consequences for both patient and provider, whether podiatrist, primary care physician, internist, or nurse practitioner.

Team Approach

As P.A.D. recognition and treatment modalities improve with advances in technology, so does the opportunity to partner with other specialists. "Interventionalists" is a term used to refer to those physicians who can provide revascularization of arteries that are occluded due to P.A.D. Interventionalists include not only vascular surgeons, but also interventional cardiologists and interventional radiologists. The key to not only reduce amputation rates, but also improve wound-healing rates often lies in the amount of perfusion available. Working closely with interventionalists ensures better outcomes, reduced risk, and opportunities to not only increase referrals, but to further advance the importance and role of podiatry in the medical community.

P.A.D. Defined

Peripheral arterial disease is a type of occlusive peripheral vascular disease. P.A.D is the most common type of peripheral vascular disease; P.V.D., as defined, includes disease of both the arteries and

P.A.D. affects the arteries outside the heart and brain and most com-

monly affects the arteries of the pelvis and legs. It affects 12-20% of Americans age 65 and older.5 An estimated 12 million people in the U.S. alone from P.A.D.(6) Compare these numbers with those of other common diseases and the fact is that P.A.D. is not only more prevalent, but is more deadly than diseases that have been better publicized.



non-diabetic patient with long term tobacco abuse

Consider the fact that there is a three-time greater risk in those persons with diabetes over the age of 50 to have peripheral arterial disease.7

The disease prevalence of P.A.D. is approximately 12 million; this is greater than cancer (8.9 million), stroke (5 million), congestive heart failure (4.8)million), Alzheimer's disease (4 million). The five-year mortality rate of P.A.D. is 30%, compared to colorectal cancer at 39%, stroke at 28%, CAD at 21%, and breast cancer at 14%.8

Atherosclerosis is the most common form of P.A.D. and P.A.D. is a systemic marker of atherosclerosis.3 The word atherosclerosis is derived from the Greek words athero ("gruel" or "paste") and sclerosis ("hardness"). It is a process by which plaque builds up in the wall of an artery leading to varying degrees of reduced blood flow through the vessel, ranging from mild to complete (and often multisegmental) occlusion. Plaque is made up of deposits of fats, cholesterol and other substances.

P.A.D Risk Factors

The risk factors for P.A.D. can be classified as traditional and nontraditional. Non-traditional risk factors include race/ethnicity, elevated levels of inflammatory markers: (Creactive protein, fibrinogen, leukocytes, interleukin-6), chronic renal disease, genetics, hypercoagulable states (altered levels of D-dimer, homocysteine, lipoprotein[a]), abnormal waist to hip ratio, and seden-

tary lifestyle.3

Among the traditional risk factors are those that include advanced aging, smoking, diabetes, hypertension and hyperlipidemia.3

The prevalence of P.A.D. has been shown to increase with age. The Framingham Heart Study found subjects >65 years old were at increased risk for development of P.A.D., while the National Health and Nutrition Examination Survey, 1999-

2000 (NHANES) report found patients >40 years old had a prevalence of 4.3%, while patients who were >70 yearsold had a prevalence of 14.5%.8,9

Smoking

Smoking increases the risk of P.A.D. four times and accelerates the onset of P.A.D. symptoms, particularly intermittent claudication, by nearly 10 years. Additionally, a dose-response relationship between pack year history and P.A.D. risk has been established. Smoking is the single most important modifiable risk factor for prevention of P.A.D. (Figure 1)!³

Diahetes

Diabetes increases the risk of de-Continued on page 211

veloping P.A.D. whether asymptomatic or symptomatic, by 1.5 to 4 times. Diabetes is also associated with an increased risk of cardiovascular events. Among diabetics, there is an early mortality among those with P.A.D. Diabetics are also at higher risk for developing ischemic ulcerations and gangrene. Diabetics also have more risk factors for developing P.A.D. than do

size. Advanced lesions intrude into the lumen of vessels, resulting in flow-limiting stenoses and chronic ischemia syndromes. Risk factors are involved in the initiation and acceleration of this process.

Atherosclerosis and the evolution of plaque formation may be characterized by four distinct stages that include:

- lesion initiation
- formation of fatty streak
- fibroproliferative atheroma

TABLE 1 **Differentiating Leg Pain Symptoms**

Pain Symptomology	PAD pain	Pain from other causes			
Character	Variable. Fatique to severe pain	Same. May have weakness			
Location	Buttock, hip, thigh, calf, feet				
Exercise induced	Yes	Same			
Distance to claudication	Same each time (may vary with speed)	variable			
Occurs with standing	No	Yes or no			
Relief	Stop walking	Often must sit or change body position			

non-diabetics. These risk factors include elevated blood pressure, increased triglycerides and increased cholesterol. Diabetics also appear to have greater vascular inflammation, increased endothelial cell dysfunction, abnormalities in vascular smooth muscle cells, increased platelet aggregation, and impaired fibrinolytic function when compared to non-diabetics.¹⁰

Pathophysiology of P.A.D.

Atherosclerosis frequently occurs at arterial bifurcations and branches where endogenous atheroprotective mechanisms are impaired as a result of disturbed flow on endothelial cells. Atherosclerosis causes the arteries to compensate initially by remodeling vessels which become larger in development

advanced lesion development

Lesion Initiation

Any mechanical or chemical injury can damage the endothelial cell layer. This alters the normal blood flow and provides sites for the adhesion and aggregation of thrombocytes, leading to the formation of blood clots or thrombi in the arterial wall. Alterations of the endothelial layer white blood (macrophages) to stick and to migrate into this layer, where they become active macrophages.

Fatty Streaks

The earliest recognizable lesions of the inner arterial layer are called "fatty streaks", which are aggregations of foam cells. Foam cells induce the further replacement of smooth endothelial cells by muscle cells from the medium arterial wall layer. The fatty streak affects the intima of the artery. The lesion consists largely of smooth muscle cells, monocytes, macrophages, T and B

Fibroproliferative Atheroma

Fibroproliferative Atheroma originates from the fatty streak and contains larger numbers of smooth muscle cells filled with lipids.

Advanced Lesion

This highly cellular lesion contains intrinsic vascular wall cells (endothelial and smooth muscle). Advanced lesions also contain inflammatory cells (monocytes, macrophages, T-lymphocytes). The advanced lesion also contains a lipid core that is covered by a fibrous cap. Acute arterial events can occur if the fibrous cap is disrupted. The resulting exposure of the "prothrombotic" necrotic lipid core and subendothelial tissue leads to thrombus formation and flow occlusion.

P.A.D. Symptoms and **Differentiating Leg Pain Symptoms**

An understanding of symptoms of P.A.D. may help to isolate the level(s) of the disease, regions of stenosis and, in most severe cases, the level of possible occlusions (Table 1). Since many patients who are afflicted with diabetes also suffer from P.A.D and peripheral neuropathy (among other disease manifestations), it is important to recognize the similarities and overlapping of symptoms. Remember that a patient does not have to be diabetic to have P.A.D., peripheral neuropathy, or both. Neuropathy may mask symptoms of P.A.D. Conversely, symptoms of P.A.D. may be mistaken for those of neuropathy.

Claudication is widely recognized as a dull cramping or pain in muscles of hips, thighs, or calf muscles when walking, climbing stairs, or exercising which is relieved with cessation of activity. Claudication is

the most common manifestation of P.A.D.

Claudication may also be characterized by fatigue in the legs, which may require a patient to stop and rest while walking. These persons may also exhibit a slow or antalgic gait and may have difficulty keeping up with others when ambulating.

Rest pain or night pain that occurs when legs elevated in bed, and relieved when placed in dependent position is another common symptom of P.A.D. Pain may typically occur in the distal foot, possibly in the vicinity of an ulcer. Questions that should be asked of patients where P.A.D. is suspected should in-

clude: "Do you have pain when you elevate your legs at night? And "Do you ever sleep sitting up with pillows behind your back, or with your legs dangling from the side of the bed?"

Impotence may also be a sign of P.A.D. and patients may see some relief with sildenafil citrate.

Critical Limb Ischemia

Critical limb ischemia is characterized by persistently recurring rest pain requiring regular analgesia. Externally, CLI may reveal non-healing ulceration or gangrene of the leg, ankle, foot, or toes. These ulcers are typically exquisitely painful, even in cases where patients are afflicted with concomitant decreased sensation due to neuropathy. Threatened limb loss or tissue loss is impending.

The Rutherford-Becker categorization of lower extremity P.A.D. specifies the extent of symptoms from asymptomatic to those seen in critical limb ischemia (Rutherford-Becker Category 4-6).(11) The Rutherford-Becker categories include:

- 0. asymptomatic
- 1. mild
- 2. moderate
- 3. severe
- 4. Ischemic rest pain



Figure 2: Patient with acute limb ischemia and the 6 P's

- 5. minor tissue loss, e.g. nonhealing ulcer, focal gangrene
- 6. major tissue loss, i.e. above transmetatarsal level

CHART 1

The 6 P's of Acute Limb

Pulselessness

• Paresthesia

Paralysis

Poikilothermy (cold)

Ischemia

• Pain

• Pallor

Where vascular shutdown of a lower extremity is occurring as an advanced symptom of critical limb ischemia, the "6 P's of acute limb ischemia" may also be observed (Chart

The 6 P's of acute limb ischemia will be seen in the absence of compensating collateral

circulation. Long-standing P.A.D. typically results in the formation of collateralization off the primary arteries; in essence, the body's creation of its own bypassing of the diseased and occluded vessels.

The evolution to paresthesia and paralysis reflects the presence of severe and potentially irreversible ischemia (Figure 2).

Recognition of P.A.D. Symptoms by Anatomical Level

Cerebrovascular Symptoms of P.A.D.

Patients with carotid artery stenosis present with ipsilateral transient ischemic attacks, strokes or amaurosis fugax (transient monocular vision loss). Symptomatic carotid ischemia is caused

 Hemodynamic factors relating to the degree of stenosis in the internal carotid artery (usually at least 70%) ipsilateral to the involved hemisphere.

- Degree of collateral flow from both the Circle of Willis and the external carotid artery.
 - Degree of intracranial disease.

Embolism from an ulcerated plaque or from the stump of a completely occluded internal carotid artery may also cause a neurological defect.

Mesenteric Ischemia

Post-prandial abdominal pain begins 30-90 minutes after eating and persists for two to three hours. This may cause patients to avoid food, resulting in weight loss. Acute mesenteric ischemia is caused by thrombosis of a severely diseased supermesenteric artery or by an embolic event originating from the heart.

Aneurysmal Disease

Abdominal aortic aneurysm (AAA) is more commonly recognized than

peripheral aneurysm. It is a condition marked by inherent weakening of the arterial wall, and subsequent focal edema. A peripheral aneurysm usually leads to acute thrombosis rather than rupture. Femoral and popliteal athery aneurysms enlarge and compress surrounding venous structures causing unilateral leg edema, venous hypertension, venous thrombosis with occlusion, or pain due to local nerve compression. Ischemia is the most common presentation of peripheral aneurysm. It presents as mild claudication to severe limb threatening ischemia. Urgent reperfusion or revascularization is necessary to avoid limb loss. As in the case of abdominal aortic aneurysm, recognition of the presence of peripheral aneurysm must be detected by obtaining a thorough history and by ordering the appropriate vascular studies.

Aorto-iliac Disease

Manifested by claudication in the buttocks, thigh and calves, isolated aorto-iliac disease is associated with sexual impotence in men and is referred to as Leriche's syndrome. Its symptoms may be re-

TABLE 2 **Vascular Surgical Procedures for Inflow Improvement**

Inflo Procedure	Oprerative Mortality (%)	Expectied Patency Rate (%) 87.5 (5 years)		
Abortobifemoral bypass	3.3			
Aortoliac or aortofemoral bypass	1 to 2	85 to 90 (5 years)		
Iliac endarterectomy	0	79 to 90 (5 years)		
Fermorofemoral bypass	6	71 (5 years)		
Axillofemoral bypass	6	49 to 80 (3 years)		
Axxillofemoral- femoral bypass	4.9	63 to 67.7 (5 years)		

TABLE 3 **Vascular Surgical Procedures for Outflow Improvement**

Outflow Procedure	Oprerative Mortality (%)	Expectied Patency Rate (%)
Fem-AK popliteal vein	1.3 to 6.3	66 (5 years)
Fem-AK popliteal prosthetic	1.3 to 6.3	50 (5 years)
Fem-BK popliteal vein	1.3 to 6.3	66 (5 years)
Fem-BK popliteal prosthetic	1.3 to 6.3	33 (5 years)
Fem-Tib vein	1.3 to 6.3	74 to 80 (5 years)
Fem-Tib prosthetic	1.3 to 6.3	25 (3 years)
Composite sequential bypass	0 to 4	28 to 40 (5 years)
Fem-Tib blind segmental bypass	2.7 to 3.2	64 to 67 (2 years)
Profundoplasty	0 to 3	49 to 50 (3 years)

lieved with sildenafil nitrate. Aorto-iliac disease of this nature may be seen in patients <50 years of age.

Lower Extremity Disease

Superficial femoral artery or popliteal arteries occlusion usually begins at Hunter's canal (adductor canal of thigh). P.A.D. at this anatomical level typically occurs in patients over the age of 40. The profunda femoris functions as a bridge between the aortofemoral segment and the femoropopliteal segment. The profunda femoris also provides collaterals to keep the lower leg viable and free of severe ischemia. It is not uncommon for patients with isolated SFA occlusion to have only mild to moderate, stable intermittent claudication.

Femoropopliteal disease is manifested by claudication in the calves and occasionally in the arches of the feet. Walking for consistent distances (example-"2 blocks") where the onset of pain occurs and cessation of ambulation must result, is the hallmark symptom of intermittent claudication.

Pseudoclaudication is a differential diagnosis to intermittent claudication. Pseudoclaudication is caused by lumbar canal stenosis.

Patients with pseudoclaudication experience leg pain with walking or prolonged standing. Calf pain arises when walking variable distances, unlike in cases of intermittent claudication. In cases of pseudoclaudication. relief is obtained from sitting or stooping, which decompresses the lumbar canal stenosis, usually taking at least 20 minutes for symptoms to abate. These patients often experience numbness and tingling in feet.

Patients with severe chronic lower extremity ischemia manifest pallor on elevating the leg above the level of the heart and reveal dependent rubor or rubor-cyanosis. Many patients experience ischemic rest pain, especially when supine, and often dangle their feet from the side of the bed for relief. They may find additional relief from symptoms by sleeping sitting up with pillows behind their back or in a chair to allow for dependent position of legs. In both scenarios, gravity assists in the perfusion of the lower extremities.

Surgical Management Treatment Options for P.A.D.

Revascularization is indicated where chronic arterial occlusive disease has caused tissue loss, ischemic rest pain, or "lifestyle limiting intermittent claudication." Surgery is not recommended as a prophylactic therapy in asymptomatic patients with lower extremity P.A.D. Revascularization of diseased vessels can be categorized according to the ap-



Figure 3: Annual cost of post-amputation care is approximately \$49,000 per patient

proach of the Interventionalist. The approach of the Interventionalist can be classified in terms of traditional surgical revascularization (by-pass procedures) versus endovascular intervention (angioplasty, atherectomy, stent placement, and thermal devices—i.e. cold laser,

Claudication does not usually progress to limb-threatening ischemia. Surgery is infrequently used to treat claudicants and should be considered only when

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American College of Cardiology/American Heart Association (ACC/AHA) Guidelines for the Treatment of P.A.D.

he high prevalence of atherosclerotic risk factors places these patients at a "markedly" increased risk of atherosclerotic ischemic events, including MI and stroke. All patients with lower extremity P.A.D. should achieve risk reduction and specific treatment targets comparable to those of individuals with established coronary artery disease.

Medical Management of P.A.D.:

To reduce adverse cardiovascular events associated with lower extremity P.A.D., lifelong treatment should include:

- Modification or elimination of atherosclerotic risk factors, such as cigarette smoking, diabetes, dyslipidemia, and hypertension
 - Promotion of daily exercise
 - Non-atherogenic diet

Lipid-Lowering Drugs:

Statins (Hydroxymethyl glutaryl [HMG] co-enzyme Q reductase inhibitors) are recommended to achieve a target LDL of <100 mg / dl in ALL patients with P.A.D.

Statins used with goal of < 70 mg/dl in patients with very high risk of ischemic events.

Among the statins recommended in the ACC/AHA guidelines are the following:

- Lipitor (atorvastatin),
- Lescol (fluvastatin)
- Mevacor, Altoprev(lovastatin)
- Pravachol (pravastatin)
- Crestor (rosuvastatin)
- Zocor (simvistatin)

Fibric acid derivatives can be useful for patients with P.A.D. and low HDL cholesterol, normal LDL cholesterol and elevated triglycerides. The fibric acid derivatives include:

Antara, Tricor (fenofibrate) and Lopid (genfibrozil).

Anti-hypertensive Therapy

Anti-hypertensive therapy should be administered to hypertensive patients with lower extremity P.A.D. to achieve a goal of: reducing blood pressure to < 140 / 90 in non-diabetics and < 130 / 80in diabetics to reduce the risk of MI, stroke, CHF and cardiovascular death.

Beta-adrenergic blocking drugs are effective antihypertensive agents and are not contraindicated.

Angiotensin-converting enzyme inhibitors are reasonable for symptomatic patients with lower extremity P.A.D. to reduce the risk of adverse cardiovascular events.

Angiotensin-converting enzyme inhibitors may be considered for patients with asymptomatic lower extremity P.A.D. to reduce the risk of adverse cardiovascular events.

ACE Inhibitors recommended for use in the treatment of patients with P.A.D., both symptomatic and asymptomatic, include: Captopril, Enalapril, Fosinopril, Ramipril, Perindopril, Quinapril, Verapamil, and Trandolapril.

Anti-platelet therapy is indicated to reduce the risk of MI, stroke, or vascular death in patients with atherosclerotic lower extremity P.A.D.

ASA (Aspirin) in daily doses of 75mg to 325 mg is recommended as a safe an effective anti-platelet therapy to reduce the risk of MI, stroke or vascular death in patients with atherosclerotic lower extremity P.A.D.

Clopidogrel (Plavix) 75 mg per day is recommended as an effective alternative anti-platelet therapy to ASA.

atherosclerotic risk factors have been treated and appropriate trials of exercise and pharmacotherapy have been utilized.

Endovascular procedures may be useful where patients suffer from symptomatic focal aorto-iliac disease, iliac and femoropopliteal lesions.

Stenting may be useful where iliacs have had suboptimal or failed balloon dilation, in cases of common and or external iliac stenosis or occlusion.

Stents, lasers, cutting balloons, atherectomy devices, and thermal devices can be useful in femoral, popliteal and tibial artery revascularization as salvage treatments or when failed results are found after balloon dilation.

It should be noted that the improvements in endovascular technology have been dramatic over the past five years and have played a major role in re-perfusion of occluded lower extremity vessels in patients previously presented with

no further surgical options for revascularization. There exists a debate as to whether endovascular procedures are worthwhile, as a perception exists among some that re-occlusion rates are high. One can argue that even in cases where re-occlusion may occur, the benefit of increased blood flow in a patient with wounds complicated by ischemia may be enough to allow for the healing of such wounds.

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- Oral anti-coagulation therapy with Warfarin is not indicated to reduce the risk of cardiovascular ischemic events in patients with atherosclerotic lower extremity P.A.D.
 - Homocysteine-Lowering Drugs:

The effectiveness of the therapeutic use of folic acid and B12 supplements in patients with lower extremity P.A.D. and homocysteine levels of $> 14 \mu$ moles/liter is not well established.

- Cilostazol (Pletal) 100mg p.o., b.i.d. is indicated as an effective therapy to improve symptoms and increase walking distance in patients with lower extremity P.A.D. and intermittent claudication (in the absence of heart failure).
- A therapeutic trial of cilastazol should be considered in all patients with lifestyle-limiting claudication (in the absence of heart failure).
- Pentoxifylline (Trental) 400mg p.o., t.i.d may be considered as a second line alternative therapy to cilastazol to improve walking distance for patients with intermittent claudication.
- The clinical effectiveness of pentoxifylline as therapy for claudication is marginal and not well established.

Other Proposed Medical Therapies:

The effectiveness of L-arginine for patients with intermittent claudication is not well established.

The effectiveness of propionly-L-carnitine as a therapy to improve walking distance in patients with intermittent claudication is not well established.

The effectiveness of ginkgo biloba to improve walking distance is marginal and not well established.

• Oral vasodilator prostaglandins such as beraprost and iloprost are not effective medications to improve the walking distance for patients with intermittent claudication.

- Vitamin E is not recommended as a treatment for patients with intermittent claudication.
- Chelation (e.g. Disodium ethylenediaminetetraacetic acid [EDTA]) is not indicated for treatment of intermittent claudication and may have harmful adverse effects, including hypocalcemia, renal insufficiency, proteinuria, and gastro-intestinal distress.

Additional ACC/AHA Non-surgical Recommendations for the Management of P.A.D.

Patients with lower extremity P.A.D. who smoke or use other forms of tobacco should be advised by each of their clinicians to stop smoking and should be offered comprehensive interventions, including behavior modification therapy, nicotine replacement therapy, or buproprion (Wellbutrin, Zyban).

Proper foot care including use of appropriate footwear, referral to podiatric medicine, daily foot inspection, skin cleansing and use of topical moisturizing creams should be encouraged.

Skin lesions and ulcerations should be addressed urgently in all diabetic patients with lower extremity P.A.D. (e.g. all diabetic patients should be assumed to have lower extremity P.A.D.).

Treatment of diabetes in individuals with lower extremity P.A.D. by administration of glucose control therapies to reduce HbA1C to < 7% can be effective to reduce microvascular complications and potentially improve cardiovascular outcomes.

A program of supervised exercise training is recommended as an initial treatment modality for patients with intermittent claudication.

Supervised exercise training should be performed for a minimum of 30-45 minutes, in sessions performed at least three times per week for a minimum of 12 weeks. The usefulness of unsupervised exercise is not well established as an effective initial treatment modality for patients with intermittent claudication.

Evidence to support the use of endovascular procedures is limited to date and can only be established after significant numbers of cases are performed along with follow-up data accumulation. Anecdotal and clinical evidence appears to strongly support the use of endovascular procedures for limb preservation and it is anticipated that further evidence will be forthcoming in the near future.

Surgical bypass data is presented in Tables 2 and 3, for a comparative perspective.

Conclusion:

- <50% of all amputees regain the ability to ambulate.
- <50% post BKA <25% post AKA.
- <50% of amputees survive past 2-3 years.
- 25% of all diabetics will face critical limb ischemia.
- Associated costs of amputation are between \$10-20 BILLION annually in the U.S. alone.
- Lower extremity amputations are still the most common treatment for critical limb ischemia.
- In 2000-01, 67% of CLI patients had amputation as their ini-
- Most shockingly, 50% of amputations are performed without prior angiography or even ABI!
- Annual cost of post-amputation care is approximately \$49,000, per patient.
- Annual cost of care and follow-up, post-limb salvage: \$600.
- Nursing home care after amputation is approximately \$100,000 per patient (Figure 3).
- Steps are highlighted to improve P.A.D. treatment and outcomes per the Prevention of Atherothrombotic Disease network (P.A.D. Network).
- 1. Increase awareness of P.A.D. and its consequences;
- 2. Identify people with symptomatic P.A.D.;
- 3. Screen for patients at high risk:
- 4. Improve treatment for symptomatic P.A.D. cases;
- 5. Increase early detection of asymptomatic cases.

The P.A.D. network also references the American Diabetes Association's recommendation that people with diabetes who are over 50 years old be regularly screened for P.A.D. as well as recommend that they have an annual foot exam.

In conclusion, the problems associated with peripheral arterial disease are extensive, and the impact of P.A.D. affects millions of patients and their families and costs billions of dollars to our healthcare system. Baby-boomers are aging and the number of diabetics continues to increase. Fortunately, technology is improving to address complications of P.A.D., yet prevention remains the best way to address the overall problem. Greater understanding of P.A.D. by all healthcare providers through greater awareness, early recognition, and knowledge of treatment options can have an enormous benefit to individual patients and society as a whole. ■

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CWS) and a Fellow of the American Professional Wound Care Association (FAP-WCA). Dr. Bell also serves as the Vice-President of the Southeastern component of APWCA. He is the co-founder of Wound Care on Wheels, LLC, and the First Coast Diabetic Foot and Wound Management Center.

He is the co-founder and Director of the Southeastern Interactive Wound Summit (SIWS), a multidisciplinary conference on advanced wound management, and is the co-founder and President of the "Save A Leg, Save A Life" Foundation. Dr. Bell is the CEO of Wound Summit Outreach, Inc., a nonprofit organization. He is in private practice in Jacksonville, FL and is on staff at Memorial Hospital of Jacksonville, St. Luke's Hospital, and Specialty Hospital of Jacksonville.

M ATI 0



See answer sheet on page 219.

- 1) Allie reported that a 25% reduction in lower extremity amputations could result in multi-billion dollar annual savings to the U.S. healthcare system. This amount is:
 - A) \$ 3 billion
 - B) \$5 billion
 - C) \$30 billion
 - D) \$50 billion
- 2) There is an increased risk of myocardial infarction (MI), stroke and cardiovascular death for patients with lower extremity P.A.D. The risk of these patients suffering a heart attack or stroke, respectively, is:
 - A) 20-30% and 50%
 - B) 40-60% and 30%
 - C) 20-60% and 40%
 - D) 40-60% and 20%
- 3) An interventionalist is a term used to refer to those physicians who can provide revascularization of arteries that are occluded due to P.A.D. Among those considered interventionalists are:
- A) cardiologists, podiatrists, radiologists
- B) cardiologists, podiatrists, vascular surgeons
- C) cardiologists, radiologists, vascular surgeons
- D) podiatrists, radiologists, vascular surgeons
- 4) The most common type of peripheral vascular disease is:
 - A) atherosclerosis
 - B) peripheral arterial disease
 - C) amaurosis fugax
 - D) hyperlipidemia
- 5) Which of the following is not considered a traditional risk factor for the development of P.A.D.?
 - A) Diabetes
 - B) Smoking
 - C) Hypertension
 - D) Hypercoagulable state

- 6) Which of the following is not considered a non-traditional risk factor for the development of P.A.D.?
 - A) advanced age
 - B) race/ethnicity
 - C) abnormal waist to hip ratio
- D) elevated levels of inflammatory markers: (C-reactive protein, fibrinogen, leukocytes, interleukin-6)
- 7) The single-most important modifiable risk factor in the prevention of P.A.D. is:
 - A) diet
 - B) sedentary lifestyle
 - C) smoking
 - D) abnormal hip-to waist ratio
- 8) Atherosclerosis and the evolution of plaque formation may be characterized by four distinct stages, which include the following, EXCEPT:
 - A) lesion initiation
 - B) formation of fatty streak
- C) fibroproliferative atheroma development
 - D) foam cell induction
- 9) Diabetics have more risk factors for developing P.A.D. than do non-diabetics. Which of the following is not considered among those risk factors?
 - A) elevated blood pressure
 - B) increased triglycerides
 - C) increased cholesterol
 - D) peripheral neuropathy
- 10) The most common manifestation of P.A.D. is:
 - A) Arch pain in feet
 - B) Claudication
 - C) Abdominal pain after eating
 - D) Amaurosis fugax
- 11) Pseudoclaudication is a differential diagnosis for claudication. Which statement is false regarding pseudoclaudication?
 - A) Calf pain arises after walking

consistent distances.

- B) Leg pain is experienced with walking or standing for prolonged pe-
- C) Numbness and tingling in feet are common symptoms.
- D) Relief is obtained from sitting or stooping, typically for at least 20 minutes.
- 12) The Rutherford-Becker is a classification system used to identify the degree of symptoms of P.A.D. At what levels of Rutherford-Becker can critical limb ischemia be found?
 - A) 0,1,2
 - B) 1,2,3
 - C) 3,4,5
 - D) 4,5,6
- 13) Acute limb ischemia is an advanced form of critical limb ischemia. The 6 P.'s of acute limb ischemia include: pulselessness, pain, pallor, poikilothermy, paresthesia and paralysis. Which two symptoms represent the presence of severe and potentially irreversible ischemia?
 - A) pain and paresthesia
 - B) poikilothermy and paralysis
 - C) paresthesia and paralysis
 - D) pulselessness and pain
- 14. Which of the following is manifested by claudication in the calves and occasionally in the arches of the feet?
 - A) Leriche's syndrome
 - B) mesenteric ischemia
 - C) peripheral aneurysm
 - D) femorpopliteal disease
- 15) Which is not indicated to reduce the risk of cardiovascular ischemic events in patients with atherosclerotic lower extremity P.A.D.?
 - A) ACE Inhibitors
 - B) Statins
 - C) Coumadin
 - D) Clopidogrel (Plavix)

Continuing dien

EXAMINATION

(cont'd)

- 16) The following are recommended to reduce the risk of myocardial infarction, stroke, or vascular death in patients with atherosclerotic lower extremity P.A.D.:
 - A) Coumadin
 - B) Aspirin
 - C) Clopidogrel (Plavix)
 - D) B and C
- 17) Treatment of diabetes in individuals with lower extremity P.A.D. by administration of glucose control therapies can be effective to reduce microvascular complications and potentially improve cardiovascular outcomes. What is the recommended HbA1C level for this patient population?
 - A) < 8.5%
 - B) <8.0%
 - C) <7.5%
 - D) <7.0%
- 18) Endovascular intervention to open occluded arteries includes all of the following modalities, except:
 - A) atherectomy
 - B) angioplasty
 - C) chelation
 - D) stent placement
- 19) Which of the following statements regarding pentoxifylline (Trental) is false?
- A) Pentoxifylline may be considered as a second line alternative therapy to cilastazol (Pletal) to improve walking distance for patients with intermittent claudication.
- B) Pentoxifylline may be considered as a first line therapy over cilastazol (Pletal) to improve walking distance for patients with intermittent claudication.
- C) The clinical effectiveness of pentoxifylline as therapy for claudication is marginal and not well established.
- D) The typical dosing schedule of pentoxifylline is 400mg three times daily.
- 20) Which of the following statements is false?
- A) Less than 50% of all amputees will regain the ability to ambulate (<50% of below knee and <25% of above knee amputees).
- B) Amputation is still the most common form of treatment for critical limb ischemia.
- C) Less than 50% of lower extremity amputees survive past 2-3 years.
- D) Amputation should be recommended versus endovascular procedures in cases where patients have been classified as having no further surgical options at revascularization.

See answer sheet on page 219.

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ENROLLMENT FORM & ANSWER SHEET (cont'd)

EXAM #4/09 Peripheral Arterial Disease Overview (D. Bell)

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4.	A	В	C	D	1	14.	A	В	C	D
5.	A	В	C	D	1	15.	A	В	C	D
6.	A	В	C	D	1	16.	A	В	C	D
7.	A	В	C	D			A	_	C	D
8.	A	В	C	D			A		C	D
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