

## PRACTICE GUIDELINES

# Management of Patients With Peripheral Artery Disease (Compilation of 2005 and 2011 ACCF/AHA Guideline Recommendations)

A Report of the American College of Cardiology Foundation/American Heart Association  
Task Force on Practice Guidelines

*Developed in Collaboration With the Society for Cardiovascular Angiography and Interventions,  
Society of Interventional Radiology, Society for Vascular Medicine, and Society for Vascular Surgery*

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**TABLE OF CONTENTS**

**Introduction** .....1557

**1. Vascular History and Physical Examination:  
Recommendations** .....1557

**2. Lower Extremity PAD: Recommendations** .....1557

**2.1. Clinical Presentation** .....1557

2.1.1. Asymptomatic.....1557  
2.1.2. Claudication.....1558  
2.1.3. Critical Limb Ischemia.....1558  
2.1.4. Acute Limb Ischemia.....1558  
2.1.5. Prior Limb Arterial Revascularization.....1558

**2.2. Diagnostic Methods** .....1558

2.2.1. Ankle- and Toe-Brachial Indices,  
Segmental Pressure Examination.....1558  
2.2.2. Pulse Volume Recording.....1559  
2.2.3. Continuous-Wave Doppler Ultrasound.....1559  
2.2.4. Treadmill Exercise Testing With and Without  
ABI Assessments and 6-Minute Walk Test.....1559  
2.2.5. Duplex Ultrasound.....1559  
2.2.6. Computed Tomographic Angiography.....1559  
2.2.7. Magnetic Resonance Angiography.....1559  
2.2.8. Contrast Angiography.....1559

**2.3. Treatment** .....1560

2.3.1. Cardiovascular Risk Reduction.....1560  
2.3.1.1. LIPID-LOWERING DRUGS.....1560  
2.3.1.2. ANTIHYPERTENSIVE DRUGS.....1560  
2.3.1.3. DIABETES THERAPIES.....1560  
2.3.1.4. SMOKING CESSATION.....1560  
2.3.1.5. HOMOCYSTEINE-LOWERING DRUGS.....1561  
2.3.1.6. ANTIPLATELET AND ANTITHROMBOTIC DRUGS.....1561  
2.3.2. Claudication.....1561  
2.3.2.1. EXERCISE AND LOWER EXTREMITY PAD  
REHABILITATION.....1561  
2.3.2.2. MEDICAL AND PHARMACOLOGICAL TREATMENT  
FOR CLAUDICATION.....1561  
2.3.2.2.1. CILOSTAZOL.....1561  
2.3.2.2.2. PENTOXIFYLLINE.....1561  
2.3.2.2.3. OTHER PROPOSED MEDICAL  
THERAPIES.....1561  
2.3.2.3. ENDOVASCULAR TREATMENT FOR CLAUDICATION.....1561  
2.3.2.4. SURGERY FOR CLAUDICATION.....1562  
2.3.2.4.1. INDICATIONS.....1562

2.3.2.4.2. PREOPERATIVE EVALUATION.....1562

2.3.2.4.3. INFLOW PROCEDURES:  
AORTOILIAC OCCLUSIVE DISEASE.....1562

2.3.2.4.4. OUTFLOW PROCEDURES:  
INFRAINGUINAL DISEASE.....1562

2.3.2.4.5. FOLLOW-UP AFTER VASCULAR  
SURGICAL PROCEDURES.....1563

2.3.3. CLI and Treatment for Limb Salvage.....1563

2.3.3.1. MEDICAL AND PHARMACOLOGICAL TREATMENT  
FOR CLI.....1563

2.3.3.1.1. PROSTAGLANDINS.....1563

2.3.3.1.2. ANGIOGENIC GROWTH FACTORS.....1563

2.3.3.2. ENDOVASCULAR TREATMENTS FOR CLI.....1563

2.3.3.3. THROMBOLYSIS FOR ACUTE AND CLI.....1563

2.3.3.4. SURGERY FOR CLI.....1563

2.3.3.4.1. INFLOW PROCEDURES:  
AORTOILIAC OCCLUSIVE DISEASE.....1563

2.3.3.4.2. OUTFLOW PROCEDURES:  
INFRAINGUINAL DISEASE.....1564

2.3.3.4.3. POSTSURGICAL CARE.....1564

**3. Renal Arterial Disease: Recommendations** .....1564

**3.1. Clinical Clues to the Diagnosis of  
Renal Artery Stenosis** .....1564

**3.2. Diagnostic Methods** .....1565

**3.3. Treatment of Renovascular Disease: RAS** .....1565

3.3.1. Medical Treatment.....1565

3.3.2. Indications for Revascularization.....1565

3.3.2.1. ASYMPTOMATIC STENOSIS.....1565

3.3.2.2. HYPERTENSION.....1565

3.3.2.3. PRESERVATION OF RENAL FUNCTION.....1565

3.3.2.4. IMPACT OF RAS ON CONGESTIVE HEART FAILURE  
AND UNSTABLE ANGINA.....1565

3.3.3. Endovascular Treatment for RAS.....1565

3.3.4. Surgery for RAS.....1565

**4. Mesenteric Arterial Disease: Recommendations** .....1566

**4.1. Acute Intestinal Ischemia** .....1566

4.1.1. Acute Intestinal Ischemia Caused by  
Arterial Obstruction.....1566

4.1.1.1. DIAGNOSIS.....1566

4.1.1.2. SURGICAL TREATMENT.....1566

4.1.1.3. ENDOVASCULAR TREATMENT.....1566

4.1.2. Acute Nonocclusive Intestinal Ischemia.....1566

4.1.2.1. ETIOLOGY.....1566

4.1.2.2. DIAGNOSIS.....1566

4.1.2.3. TREATMENT.....1566

<b>4.2. Chronic Intestinal Ischemia</b> .....	1566
4.2.1. Diagnosis .....	1566
4.2.2. Endovascular Treatment for Chronic Intestinal Ischemia .....	1566
4.2.3. Surgical Treatment .....	1566
<b>5. Aneurysms of the Abdominal Aorta, Its Branch Vessels, and the Lower Extremities: Recommendations</b> .....	1567
<b>5.1. Abdominal Aortic and Iliac Aneurysms</b> .....	1567
5.1.1. Etiology .....	1567
5.1.1.1. ATHEROSCLEROTIC RISK FACTORS .....	1567
5.1.2. Natural History .....	1567
5.1.2.1. AORTIC ANEURYSM RUPTURE .....	1567
5.1.3. Diagnosis .....	1567
5.1.3.1. SYMPTOMATIC AORTIC OR ILIAC ANEURYSMS .....	1567
5.1.3.2. SCREENING HIGH-RISK POPULATIONS .....	1567
5.1.4. Observational Management .....	1567
5.1.4.1. BLOOD PRESSURE CONTROL AND BETA-BLOCKADE .....	1567
5.1.5. Prevention of Aortic Aneurysm Rupture .....	1567
5.1.5.1. MANAGEMENT OVERVIEW .....	1567
<b>5.2. Visceral Artery Aneurysms</b> .....	1567
<b>5.3. Lower Extremity Aneurysms</b> .....	1567
5.3.1. Natural History .....	1567
5.3.2. Management .....	1568
5.3.2.1. CATHETER-RELATED FEMORAL ARTERY PSEUDOANEURYSMS .....	1568

<b>Appendix 1. Author Relationships With Industry (Relevant)—2005 ACC/AHA Writing Committee to Develop Guidelines on Peripheral Arterial Disease</b> .....	1568
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<b>Appendix 2. Author Relationships With Industry and Other Entities (Relevant)—2011 ACCF/AHA Focused Update of the Guideline for the Management of Patients With Peripheral Artery Disease</b> .....	1569
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## Introduction

This document is a compilation of the current American College of Cardiology Foundation/American Heart Association (ACCF/AHA) practice guideline recommendations for peripheral artery disease from the “ACC/AHA 2005 Guidelines for the Management of Patients With Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic)”<sup>\*</sup> and the “2011 ACCF/AHA Focused Update of the Guideline for the Management of Patients With Peripheral Artery Disease (Updating the 2005 Guideline)”<sup>†</sup>. Updated and new recommendations from 2011 are noted and outdated recommendations have been removed. No new evidence was reviewed, and no recommendations included herein are original to this document. The ACCF/AHA Task Force on Practice Guidelines chooses to republish the recommendations in this format to provide the complete set of practice guideline recommendations in a single resource. Because this document includes recommendations only, please refer to the

respective 2005 and 2011 articles for all introductory and supportive content until the entire full-text guideline is revised. In the future, the ACCF/AHA Task Force on Practice Guidelines will maintain a continuously updated full-text guideline.

## 1. Vascular History and Physical Examination: Recommendations

### CLASS I

1. Individuals at risk for lower extremity peripheral artery disease (PAD) should undergo a vascular review of symptoms to assess walking impairment, claudication, ischemic rest pain, and/or the presence of nonhealing wounds. (*Level of Evidence: C*)
2. Individuals at risk for lower extremity PAD should undergo comprehensive pulse examination and inspection of the feet. (*Level of Evidence: C*)
3. Individuals over 50 years of age should be asked if they have a family history of a first-order relative with an abdominal aortic aneurysm (AAA). (*Level of Evidence: C*)

## 2. Lower Extremity PAD: Recommendations

### 2.1. Clinical Presentation

#### 2.1.1. Asymptomatic

### CLASS I

1. A history of walking impairment, claudication, ischemic rest pain, and/or nonhealing wounds is recommended as a required component of a standard review of symptoms for adults 50 years and older who have atherosclerosis risk factors and for adults 70 years and older. (*Level of Evidence: C*)
2. Individuals with asymptomatic lower extremity PAD should be identified by examination and/or measurement of the ankle-brachial index (ABI) so that therapeutic interventions known to diminish their increased risk of myocardial infarction (MI), stroke, and death may be offered. (*Level of Evidence: B*)
3. Smoking cessation, lipid lowering, and diabetes and hypertension treatment according to current national treatment guidelines are recommended for individuals with asymptomatic lower extremity PAD. (*Level of Evidence: B*)
4. Antiplatelet therapy is indicated for individuals with asymptomatic lower extremity PAD to reduce the risk of adverse cardiovascular ischemic events. (*Level of Evidence: C*)

### CLASS IIa

1. An exercise ABI measurement can be useful to diagnose lower extremity PAD in individuals who are at risk for lower extremity PAD who have a normal ABI (0.91 to 1.30), are without classic claudication symptoms, and have no other clinical evidence of atherosclerosis. (*Level of Evidence: C*)
2. A toe-brachial index or pulse volume recording measurement can be useful to diagnose lower extremity PAD in individuals who are at risk for lower extremity PAD who have an ABI greater than 1.30 and no other clinical evidence of atherosclerosis. (*Level of Evidence: C*)

\*J Am Coll Cardiol 2006;47:1239–12, <http://dx.doi.org/10.1016/j.jacc.2005.10.009>

†J Am Coll Cardiol 2011;58:2020–45, <http://dx.doi.org/10.1016/j.jacc.2011.08.023>

**CLASS IIb**

1. Angiotensin-converting enzyme (ACE) inhibition may be considered for individuals with asymptomatic lower extremity PAD for cardiovascular risk reduction. (*Level of Evidence: C*)

### 2.1.2. Claudication

**CLASS I**

1. Patients with symptoms of intermittent claudication should undergo a vascular physical examination, including measurement of the ABI. (*Level of Evidence: B*)
2. In patients with symptoms of intermittent claudication, the ABI should be measured after exercise if the resting index is normal. (*Level of Evidence: B*)
3. Patients with intermittent claudication should have significant functional impairment with a reasonable likelihood of symptomatic improvement and absence of other disease that would comparably limit exercise even if the claudication was improved (e.g., angina, heart failure, chronic respiratory disease, or orthopedic limitations) before undergoing an evaluation for revascularization. (*Level of Evidence: C*)
4. Individuals with intermittent claudication who are offered the option of endovascular or surgical therapies should: (a) be provided information regarding supervised claudication exercise therapy and pharmacotherapy; (b) receive comprehensive risk factor modification and antiplatelet therapy; (c) have a significant disability, either being unable to perform normal work or having serious impairment of other activities important to the patient; and (d) have lower extremity PAD lesion anatomy such that the revascularization procedure would have low risk and a high probability of initial and long-term success. (*Level of Evidence: C*)

**CLASS III**

1. Arterial imaging is not indicated for patients with a normal postexercise ABI. This does not apply if other atherosclerotic causes (e.g., entrapment syndromes or isolated internal iliac artery occlusive disease) are suspected. (*Level of Evidence: C*)

### 2.1.3. Critical Limb Ischemia

**CLASS I**

1. Patients with critical limb ischemia (CLI) should undergo expedited evaluation and treatment of factors that are known to increase the risk of amputation. (*Level of Evidence: C*)
2. Patients with CLI in whom open surgical repair is anticipated should undergo assessment of cardiovascular risk. (*Level of Evidence: B*)
3. Patients with a prior history of CLI or who have undergone successful treatment for CLI should be evaluated at least twice annually by a vascular specialist owing to the relatively high incidence of recurrence. (*Level of Evidence: C*)
4. Patients at risk of CLI (ABI <0.4 in an individual with diabetes, or any individual with diabetes and known lower extremity PAD) should undergo regular inspection of the feet to detect objective signs of CLI. (*Level of Evidence: B*)
5. The feet should be examined directly, with shoes and socks removed, at regular intervals after successful treatment of CLI. (*Level of Evidence: C*)
6. Patients with CLI and features to suggest atheroembolization should be evaluated for aneurysmal disease (e.g., abdominal aortic, popliteal, or common femoral aneurysms). (*Level of Evidence: B*)

7. Systemic antibiotics should be initiated promptly in patients with CLI, skin ulcerations, and evidence of limb infection. (*Level of Evidence: B*)
8. Patients with CLI and skin breakdown should be referred to healthcare providers with specialized expertise in wound care. (*Level of Evidence: B*)
9. Patients at risk for CLI (those with diabetes, neuropathy, chronic renal failure, or infection) who develop acute limb symptoms represent potential vascular emergencies and should be assessed immediately and treated by a specialist competent in treating vascular disease. (*Level of Evidence: C*)
10. Patients at risk for or who have been treated for CLI should receive verbal and written instructions regarding self-surveillance for potential recurrence. (*Level of Evidence: C*)

### 2.1.4. Acute Limb Ischemia

**CLASS I**

1. Patients with acute limb ischemia and a salvageable extremity should undergo an emergent evaluation that defines the anatomic level of occlusion and that leads to prompt endovascular or surgical revascularization. (*Level of Evidence: B*)

**CLASS III**

1. Patients with acute limb ischemia and a nonviable extremity should not undergo an evaluation to define vascular anatomy or efforts to attempt revascularization. (*Level of Evidence: B*)

### 2.1.5. Prior Limb Arterial Revascularization

**CLASS I**

1. Long-term patency of infrainguinal bypass grafts should be evaluated in a surveillance program, which should include an interval vascular history, resting ABIs, physical examination, and a duplex ultrasound at regular intervals if a venous conduit has been used. (*Level of Evidence: B*)

**CLASS IIa**

1. Long-term patency of infrainguinal bypass grafts may be considered for evaluation in a surveillance program, which may include conducting exercise ABIs and other arterial imaging studies at regular intervals. (*Level of Evidence: B*)
2. Long-term patency of endovascular sites may be evaluated in a surveillance program, which may include conducting exercise ABIs and other arterial imaging studies at regular intervals. (*Level of Evidence: B*)

## 2.2. Diagnostic Methods

### 2.2.1. Ankle- and Toe-Brachial Indices, Segmental Pressure Examination

**CLASS I**

1. **2011 Updated Recommendation:** The resting ABI should be used to establish the lower extremity PAD diagnosis in patients with suspected lower extremity PAD, defined as individuals with 1 or more of the following: exertional leg symptoms, nonhealing wounds, age 65 and older, or 50 years and older with a history of smoking or diabetes. (*Level of Evidence: B*)
2. The ABI should be measured in both legs in all new patients with PAD of any severity to confirm the diagnosis of lower extremity PAD and establish a baseline. (*Level of Evidence: B*)
3. The toe-brachial index should be used to establish the lower extremity PAD diagnosis in patients in whom lower extremity



PAD is clinically suspected but in whom the ABI test is not reliable due to noncompressible vessels (usually patients with long-standing diabetes or advanced age). (Level of Evidence: B)

4. Leg segmental pressure measurements are useful to establish the lower extremity PAD diagnosis when anatomic localization of lower extremity PAD is required to create a therapeutic plan. (Level of Evidence: B)
5. **2011 New Recommendation:** ABI results should be uniformly reported with noncompressible values defined as greater than 1.40, normal values 1.00 to 1.40, borderline 0.91 to 0.99, and abnormal 0.90 or less. (Level of Evidence: B)

### 2.2.2. Pulse Volume Recording

#### CLASS IIa

1. Pulse volume recordings are reasonable to establish the initial lower extremity PAD diagnosis, assess localization and severity, and follow the status of lower extremity revascularization procedures. (Level of Evidence: B)

### 2.2.3. Continuous-Wave Doppler Ultrasound

#### CLASS I

1. Continuous-wave Doppler ultrasound blood flow measurements are useful to provide an accurate assessment of lower extremity PAD location and severity, to follow lower extremity PAD progression, and to provide quantitative follow-up after revascularization procedures. (Level of Evidence: B)

### 2.2.4. Treadmill Exercise Testing With and Without ABI Assessments and 6-Minute Walk Test

#### CLASS I

1. Exercise treadmill tests are recommended to provide the most objective evidence of the magnitude of the functional limitation of claudication and to measure the response to therapy. (Level of Evidence: B)
2. A standardized exercise protocol (either fixed or graded) with a motorized treadmill should be used to ensure reproducibility of measurements of pain-free walking distance and maximal walking distance. (Level of Evidence: B)
3. Exercise treadmill tests with measurement of pre-exercise and postexercise ABI values are recommended to provide diagnostic data useful in differentiating arterial claudication from nonarterial claudication (“pseudoclaudication”). (Level of Evidence: B)
4. Exercise treadmill tests should be performed in individuals with claudication who are to undergo exercise training (lower extremity PAD rehabilitation) so as to determine functional capacity, assess nonvascular exercise limitations, and demonstrate the safety of exercise. (Level of Evidence: B)

#### CLASS IIb

1. A 6-minute walk test may be reasonable to provide an objective assessment of the functional limitation of claudication and response to therapy in elderly individuals or others not amenable to treadmill testing. (Level of Evidence: B)

### 2.2.5. Duplex Ultrasound

#### CLASS I

1. Duplex ultrasound of the extremities is useful to diagnose anatomic location and degree of stenosis of PAD. (Level of Evidence: A)
2. Duplex ultrasound is recommended for routine surveillance after femoral-popliteal or femoral-tibial-pedal bypass with a venous

conduit. Minimum surveillance intervals are approximately 3, 6, and 12 months, and then yearly after graft placement. (Level of Evidence: A)

#### CLASS IIa

1. Duplex ultrasound of the extremities can be useful to select patients as candidates for endovascular intervention. (Level of Evidence: B)
2. Duplex ultrasound can be useful to select patients as candidates for surgical bypass and to select the sites of surgical anastomosis. (Level of Evidence: B)

#### CLASS IIb

1. The use of duplex ultrasound is not well established to assess long-term patency of percutaneous transluminal angioplasty. (Level of Evidence: B)
2. Duplex ultrasound may be considered for routine surveillance after femoral-popliteal bypass with a synthetic conduit. (Level of Evidence: B)

### 2.2.6. Computed Tomographic Angiography

#### CLASS IIb

1. Computed tomographic angiography (CTA) of the extremities may be considered to diagnose anatomic location and presence of significant stenosis in patients with lower extremity PAD. (Level of Evidence: B)
2. CTA of the extremities may be considered as a substitute for magnetic resonance angiography (MRA) for those patients with contraindications to MRA. (Level of Evidence: B)

### 2.2.7. Magnetic Resonance Angiography

#### CLASS I

1. MRA of the extremities is useful to diagnose anatomic location and degree of stenosis of PAD. (Level of Evidence: A)
2. MRA of the extremities should be performed with gadolinium enhancement. (Level of Evidence: B)
3. MRA of the extremities is useful in selecting patients with lower extremity PAD as candidates for endovascular intervention. (Level of Evidence: A)

#### CLASS IIb

1. MRA of the extremities may be considered to select patients with lower extremity PAD as candidates for surgical bypass and to select the sites of surgical anastomosis. (Level of Evidence: B)
2. MRA of the extremities may be considered for postrevascularization (endovascular and surgical bypass) surveillance in patients with lower extremity PAD. (Level of Evidence: B)

### 2.2.8. Contrast Angiography

#### CLASS I

1. Contrast angiography provides detailed information about arterial anatomy and is recommended for evaluation of patients with lower extremity PAD when revascularization is contemplated. (Level of Evidence: B)
2. A history of contrast reaction should be documented before the performance of contrast angiography and appropriate pretreatment administered before contrast is given. (Level of Evidence: B)
3. Decisions regarding the potential utility of invasive therapeutic interventions (percutaneous or surgical) in patients with lower extremity PAD should be made with a complete anatomic

assessment of the affected arterial territory, including imaging of the occlusive lesion, as well as arterial inflow and outflow with angiography or a combination of angiography and noninvasive vascular techniques. (Level of Evidence: B)

4. Digital subtraction angiography is recommended for contrast angiographic studies because this technique allows for enhanced imaging capabilities compared with conventional unsubtracted contrast angiography. (Level of Evidence: A)
5. Before performance of contrast angiography, a full history and complete vascular examination should be performed to optimize decisions regarding the access site, as well as to minimize contrast dose and catheter manipulation. (Level of Evidence: C)
6. Selective or super selective catheter placement during lower extremity angiography is indicated because this can enhance imaging, reduce contrast dose, and improve sensitivity and specificity of the procedure. (Level of Evidence: C)
7. The diagnostic lower extremity arteriogram should image the iliac, femoral, and tibial bifurcations in profile without vessel overlap. (Level of Evidence: B)
8. When conducting a diagnostic lower extremity arteriogram in which the significance of an obstructive lesion is ambiguous, transstenotic pressure gradients and supplementary angulated views should be obtained. (Level of Evidence: B)
9. Patients with baseline renal insufficiency should receive hydration before undergoing contrast angiography. (Level of Evidence: B)
10. Follow-up clinical evaluation, including a physical examination and measurement of renal function, is recommended within 2 weeks after contrast angiography to detect the presence of delayed adverse effects, such as atheroembolism, deterioration in renal function, or access site injury (e.g., pseudoaneurysm or arteriovenous fistula). (Level of Evidence: C)

#### CLASS IIa

1. Noninvasive imaging modalities, including MRA, CTA, and color flow duplex imaging, may be used in advance of invasive imaging to develop an individualized diagnostic strategic plan, including assistance in selection of access sites, identification of significant lesions, and determination of the need for invasive evaluation. (Level of Evidence: B)
2. Treatment with *n*-acetylcysteine in advance of contrast angiography is suggested for patients with baseline renal insufficiency (creatinine >2.0 mg per dL). (Level of Evidence: B)

## 2.3. Treatment

### 2.3.1. Cardiovascular Risk Reduction

#### 2.3.1.1. LIPID-LOWERING DRUGS

##### CLASS I

1. Treatment with a hydroxymethyl glutaryl coenzyme-A reductase inhibitor (statin) medication is indicated for all patients with PAD to achieve a target low-density lipoprotein cholesterol level of less than 100 mg per dL. (Level of Evidence: B)

##### CLASS IIa

1. Treatment with a hydroxymethyl glutaryl coenzyme-A reductase inhibitor (statin) medication to achieve a target low-density lipoprotein cholesterol level of less than 70 mg per dL is reasonable for patients with lower extremity PAD at very high risk of ischemic events. (Level of Evidence: B)

2. Treatment with a fibric acid derivative can be useful for patients with PAD and low high-density lipoprotein cholesterol, normal low-density lipoprotein cholesterol, and elevated triglycerides. (Level of Evidence: C)

#### 2.3.1.2. ANTIHYPERTENSIVE DRUGS

##### CLASS I

1. Antihypertensive therapy should be administered to hypertensive patients with lower extremity PAD to achieve a goal of less than 140 mm Hg systolic over 90 mm Hg diastolic (individuals without diabetes) or less than 130 mm Hg systolic over 80 mm Hg diastolic (individuals with diabetes and individuals with chronic renal disease) to reduce the risk of MI, stroke, congestive heart failure, and cardiovascular death. (Level of Evidence: A)
2. Beta-adrenergic blocking drugs are effective antihypertensive agents and are not contraindicated in patients with PAD. (Level of Evidence: A)

##### CLASS IIa

1. The use of ACE inhibitors is reasonable for symptomatic patients with lower extremity PAD to reduce the risk of adverse cardiovascular events. (Level of Evidence: B)

##### CLASS IIb

1. ACE inhibitors may be considered for patients with asymptomatic lower extremity PAD to reduce the risk of adverse cardiovascular events. (Level of Evidence: C)

#### 2.3.1.3. DIABETES THERAPIES

##### CLASS I

1. Proper foot care, including use of appropriate footwear, chiropody/podiatric medicine, daily foot inspection, skin cleansing, and use of topical moisturizing creams, should be encouraged and skin lesions and ulcerations should be addressed urgently in all patients with diabetes and lower extremity PAD. (Level of Evidence: B)

##### CLASS IIa

1. Treatment of diabetes in individuals with lower extremity PAD by administration of glucose control therapies to reduce the hemoglobin A1C to less than 7% can be effective to reduce microvascular complications and potentially improve cardiovascular outcomes. (Level of Evidence: C)

#### 2.3.1.4. SMOKING CESSATION

##### CLASS I

1. **2011 New Recommendation:** Patients who are smokers or former smokers should be asked about status of tobacco use at every visit. (Level of Evidence: A)
2. **2011 New Recommendation:** Patients should be assisted with counseling and developing a plan for quitting that may include pharmacotherapy and/or referral to a smoking cessation program. (Level of Evidence: A)
3. **2011 Updated Recommendation:** Individuals with lower extremity PAD who smoke cigarettes or use other forms of tobacco should be advised by each of their clinicians to stop smoking and offered behavioral and pharmacological treatment. (Level of Evidence: C)
4. **2011 New Recommendation:** In the absence of contraindication or other compelling clinical indication, 1 or more of the following pharmacological therapies should be offered: varenicline, bupropion, and nicotine replacement therapy. (Level of Evidence: A)

### 2.3.1.5. HOMOCYSTEINE-LOWERING DRUGS

#### CLASS IIB

1. The effectiveness of the therapeutic use of folic acid and B<sub>12</sub> vitamin supplements in individuals with lower extremity PAD and homocysteine levels greater than 14 micromoles per liter is not well established. (Level of Evidence: C)

### 2.3.1.6. ANTIPLATELET AND ANTITHROMBOTIC DRUGS

#### CLASS I

1. **2011 Updated Recommendation:** Antiplatelet therapy is indicated to reduce the risk of MI, stroke, and vascular death in individuals with symptomatic atherosclerotic lower extremity PAD, including those with intermittent claudication or CLI prior lower extremity revascularization (endovascular or surgical), or prior amputation for lower extremity ischemia. (Level of Evidence: A)
2. **2011 Updated Recommendation:** Aspirin, typically in daily doses of 75 to 325 mg, is recommended as safe and effective antiplatelet therapy to reduce the risk of MI, stroke, or vascular death in individuals with symptomatic atherosclerotic lower extremity PAD, including those with intermittent claudication or CLI, prior lower extremity revascularization (endovascular or surgical), or prior amputation for lower extremity ischemia. (Level of Evidence: B)
3. **2011 Updated Recommendation:** Clopidogrel (75 mg per day) is recommended as a safe and effective alternative antiplatelet therapy to aspirin to reduce the risk of MI, ischemic stroke, or vascular death in individuals with symptomatic atherosclerotic lower extremity PAD, including those with intermittent claudication or CLI, prior lower extremity revascularization (endovascular or surgical), or prior amputation for lower extremity ischemia. (Level of Evidence: B)

#### CLASS IIA

1. **2011 New Recommendation:** Antiplatelet therapy can be useful to reduce the risk of MI, stroke, or vascular death in asymptomatic individuals with an ABI less than or equal to 0.90. (Level of Evidence: C)

#### CLASS IIB

1. **2011 New Recommendation:** The usefulness of antiplatelet therapy to reduce the risk of MI, stroke, or vascular death in asymptomatic individuals with borderline abnormal ABI, defined as 0.91 to 0.99, is not well established. (Level of Evidence: A)
2. **2011 New Recommendation:** The combination of aspirin and clopidogrel may be considered to reduce the risk of cardiovascular events in patients with symptomatic atherosclerotic lower extremity PAD, including those with intermittent claudication or CLI, prior lower extremity revascularization (endovascular or surgical), or prior amputation for lower extremity ischemia and who are not at increased risk of bleeding and who are high perceived cardiovascular risk. (Level of Evidence: B)

#### CLASS III: NO BENEFIT

1. **2011 Updated Recommendation:** In the absence of any other proven indication for warfarin, its addition to antiplatelet therapy to reduce the risk of adverse cardiovascular ischemic events in individuals with atherosclerotic lower extremity PAD is of no benefit and is potentially harmful due to increased risk of major bleeding. (Level of Evidence: B)

## 2.3.2. Claudication

### 2.3.2.1. EXERCISE AND LOWER EXTREMITY PAD REHABILITATION

#### CLASS I

1. A program of supervised exercise training is recommended as an initial treatment modality for patients with intermittent claudication. (Level of Evidence: A)
2. Supervised exercise training should be performed for a minimum of 30 to 45 minutes, in sessions performed at least 3 times per week for a minimum of 12 weeks. (Level of Evidence: A)

#### CLASS IIB

1. The usefulness of unsupervised exercise programs is not well established as an effective initial treatment modality for patients with intermittent claudication. (Level of Evidence: B)

### 2.3.2.2. MEDICAL AND PHARMACOLOGICAL TREATMENT FOR CLAUDICATION

#### 2.3.2.2.1. CILOSTAZOL

#### CLASS I

1. Cilostazol (100 mg orally 2 times per day) is indicated as an effective therapy to improve symptoms and increase walking distance in patients with lower extremity PAD and intermittent claudication (in the absence of heart failure). (Level of Evidence: A)
2. A therapeutic trial of cilostazol should be considered in all patients with lifestyle-limiting claudication (in the absence of heart failure). (Level of Evidence: A)

#### 2.3.2.2.2. PENTOXIFYLLINE

#### CLASS IIB

1. Pentoxifylline (400 mg 3 times per day) may be considered as second-line alternative therapy to cilostazol to improve walking distance in patients with intermittent claudication. (Level of Evidence: A)
2. The clinical effectiveness of pentoxifylline as therapy for claudication is marginal and not well established. (Level of Evidence: C)

#### 2.3.2.2.3. OTHER PROPOSED MEDICAL THERAPIES

#### CLASS IIB

1. The effectiveness of L-arginine for patients with intermittent claudication is not well established. (Level of Evidence: B)
2. The effectiveness of propionyl-L-carnitine as a therapy to improve walking distance in patients with intermittent claudication is not well established. (Level of Evidence: B)
3. The effectiveness of ginkgo biloba to improve walking distance for patients with intermittent claudication is marginal and not well established. (Level of Evidence: B)

#### CLASS III

1. Oral vasodilator prostaglandins such as beraprost and iloprost are not effective medications to improve walking distance in patients with intermittent claudication. (Level of Evidence: A)
2. Vitamin E is not recommended as a treatment for patients with intermittent claudication. (Level of Evidence: C)
3. Chelation (e.g., ethylenediaminetetraacetic acid) is not indicated for treatment of intermittent claudication and may have harmful adverse effects. (Level of Evidence: A)

### 2.3.2.3. ENDOVASCULAR TREATMENT FOR CLAUDICATION

#### CLASS I

1. Endovascular procedures are indicated for individuals with a vocational or lifestyle-limiting disability due to intermittent clau-

dication when clinical features suggest a reasonable likelihood of symptomatic improvement with endovascular intervention and (a) there has been an inadequate response to exercise or pharmacological therapy and/or (b) there is a very favorable risk-benefit ratio (e.g., focal aortoiliac occlusive disease). (*Level of Evidence: A*)

2. Endovascular intervention is recommended as the preferred revascularization technique for TASC type A iliac and femoropopliteal arterial lesions. (*Level of Evidence: B*)
3. Translesional pressure gradients (with and without vasodilation) should be obtained to evaluate the significance of angiographic iliac arterial stenoses of 50% to 75% diameter before intervention. (*Level of Evidence: C*)
4. Provisional stent placement is indicated for use in the iliac arteries as salvage therapy for a suboptimal or failed result from balloon dilation (e.g., persistent translesional gradient, residual diameter stenosis >50%, or flow-limiting dissection). (*Level of Evidence: B*)
5. Stenting is effective as primary therapy for common iliac artery stenosis and occlusions. (*Level of Evidence: B*)
6. Stenting is effective as primary therapy in external iliac artery stenoses and occlusions. (*Level of Evidence: C*)

#### CLASS IIa

1. Stents (and other adjunctive techniques such as lasers, cutting balloons, atherectomy devices, and thermal devices) can be useful in the femoral, popliteal, and tibial arteries as salvage therapy for a suboptimal or failed result from balloon dilation (e.g., persistent translesional gradient, residual diameter stenosis >50%, or flow-limiting dissection). (*Level of Evidence: C*)

#### CLASS IIb

1. The effectiveness of stents, atherectomy, cutting balloons, thermal devices, and lasers for the treatment of femoral-popliteal arterial lesions (except to salvage a suboptimal result from balloon dilation) is not well-established. (*Level of Evidence: A*)
2. The effectiveness of uncoated/uncovered stents, atherectomy, cutting balloons, thermal devices, and lasers for the treatment of infrapopliteal lesions (except to salvage a suboptimal result from balloon dilation) is not well established. (*Level of Evidence: C*)

#### CLASS III

1. Endovascular intervention is not indicated if there is no significant pressure gradient across a stenosis despite flow augmentation with vasodilators. (*Level of Evidence: C*)
2. Primary stent placement is not recommended in the femoral, popliteal, or tibial arteries. (*Level of Evidence: C*)
3. Endovascular intervention is not indicated as prophylactic therapy in an asymptomatic patient with lower extremity PAD. (*Level of Evidence: C*)

#### 2.3.2.4. SURGERY FOR CLAUDICATION

##### 2.3.2.4.1. INDICATIONS

#### CLASS I

1. Surgical interventions are indicated for individuals with claudication symptoms who have a significant functional disability that is vocational or lifestyle limiting, who are unresponsive to exercise or pharmacotherapy, and who have a reasonable likelihood of symptomatic improvement. (*Level of Evidence: B*)

#### CLASS IIb

1. Because the presence of more aggressive atherosclerotic occlusive disease is associated with less durable results in patients

younger than 50 years of age, the effectiveness of surgical intervention in this population for intermittent claudication is unclear. (*Level of Evidence: B*)

#### CLASS III

1. Surgical intervention is not indicated to prevent progression to limb-threatening ischemia in patients with intermittent claudication. (*Level of Evidence: B*)

#### 2.3.2.4.2. PREOPERATIVE EVALUATION

#### CLASS I

1. A preoperative cardiovascular risk evaluation should be undertaken in those patients with lower extremity PAD in whom a major vascular surgical intervention is planned. (*Level of Evidence: B*)

#### 2.3.2.4.3. INFLOW PROCEDURES:

##### AORTOILIAC OCCLUSIVE DISEASE

#### CLASS I

1. Aortobifemoral bypass is beneficial for patients with vocational- or lifestyle-disabling symptoms and hemodynamically significant aortoiliac disease who are acceptable surgical candidates and who are unresponsive to or unsuitable for exercise, pharmacotherapy, or endovascular repair. (*Level of Evidence: B*)
2. Iliac endarterectomy and aortoiliac or iliofemoral bypass in the setting of acceptable aortic inflow should be used for the surgical treatment of unilateral disease or in conjunction with femoral-femoral bypass for the treatment of a patient with bilateral iliac artery occlusive disease if the patient is not a suitable candidate for aortobifemoral bypass grafting. (*Level of Evidence: B*)

#### CLASS IIb

1. Axillofemoral-femoral bypass may be considered for the surgical treatment of patients with intermittent claudication in very limited settings, such as chronic infrarenal aortic occlusion associated with symptoms of severe claudication in patients who are not candidates for aortobifemoral bypass. (*Level of Evidence: B*)

#### CLASS III

1. Axillofemoral-femoral bypass should not be used for the surgical treatment of patients with intermittent claudication except in very limited settings. (*Level of Evidence: B*)

#### 2.3.2.4.4. OUTFLOW PROCEDURES: INFRAINGUINAL DISEASE

#### CLASS I

1. Bypasses to the popliteal artery above the knee should be constructed with autogenous vein when possible. (*Level of Evidence: A*)
2. Bypasses to the popliteal artery below the knee should be constructed with autogenous vein when possible. (*Level of Evidence: B*)

#### CLASS IIa

1. The use of synthetic grafts to the popliteal artery below the knee is reasonable only when no autogenous vein from ipsilateral or contralateral leg or arms is available. (*Level of Evidence: A*)

#### CLASS IIb

1. Femoral-tibial artery bypasses constructed with autogenous vein may be considered for the treatment of claudication in rare instances for certain patients. (*Level of Evidence: B*)



2. Because their use is associated with reduced patency rates, the effectiveness of the use of synthetic grafts to the popliteal artery above the knee is not well established. (*Level of Evidence: B*)

**CLASS III**

1. Femoral-tibial artery bypasses with synthetic graft material should not be used for the treatment of claudication. (*Level of Evidence: C*)

2.3.2.4.5. FOLLOW-UP AFTER VASCULAR SURGICAL PROCEDURES

**CLASS I**

1. Patients who have undergone placement of aortobifemoral bypass grafts should be followed up with periodic evaluations that record any return or progression of claudication symptoms, the presence of femoral pulses, and ABIs at rest and after exercise. (*Level of Evidence: C*)
2. Patients who have undergone placement of a lower extremity bypass with autogenous vein should undergo periodic evaluations for at least 2 years that record any claudication symptoms; a physical examination and pulse examination of the proximal, graft, and outflow vessels; and duplex imaging of the entire length of the graft, with measurement of peak systolic velocities and calculation of velocity ratios across all lesions. (*Level of Evidence: C*)
3. Patients who have undergone placement of a synthetic lower extremity bypass graft should, for at least 2 years after implantation, undergo periodic evaluations that record any return or progression of claudication symptoms; a pulse examination of the proximal, graft, and outflow vessels; and assessment of ABIs at rest and after exercise. (*Level of Evidence: C*)

2.3.3. CLI and Treatment for Limb Salvage

2.3.3.1. MEDICAL AND PHARMACOLOGICAL TREATMENT FOR CLI

**CLASS III**

1. Parenteral administration of pentoxifylline is not useful for the treatment of CLI. (*Level of Evidence: B*)

2.3.3.1.1. PROSTAGLANDINS

**CLASS IIb**

1. Parenteral administration of PGE-1 or iloprost for 7 to 28 days may be considered to reduce ischemic pain and facilitate ulcer healing in patients with CLI, but its efficacy is likely to be limited to a small percentage of patients. (*Level of Evidence: A*)

**CLASS III**

1. Oral iloprost is not an effective therapy to reduce the risk of amputation or death in patients with CLI. (*Level of Evidence: B*)

2.3.3.1.2. ANGIOGENIC GROWTH FACTORS

**CLASS IIb**

1. The efficacy of angiogenic growth factor therapy for treatment of CLI is not well established and is best investigated in the context of a placebo-controlled trial. (*Level of Evidence: C*)

2.3.3.2. ENDOVASCULAR TREATMENTS FOR CLI

**CLASS I**

1. For individuals with combined inflow and outflow disease with CLI, inflow lesions should be addressed first. (*Level of Evidence: C*)
2. For individuals with combined inflow and outflow disease in whom symptoms of CLI or infection persist after inflow revascularization, an outflow revascularization procedure should be performed. (*Level of Evidence: B*)

larization, an outflow revascularization procedure should be performed. (*Level of Evidence: B*)

3. If it is unclear whether hemodynamically significant inflow disease exists, intra-arterial pressure measurements across suprainguinal lesions should be measured before and after the administration of a vasodilator. (*Level of Evidence: C*)

**CLASS IIa**

1. **2011 New Recommendation:** For patients with limb-threatening lower extremity ischemia and an estimated life expectancy of 2 years or less in patients in whom an autogenous vein conduit is not available, balloon angioplasty is reasonable to perform when possible as the initial procedure to improve distal blood flow. (*Level of Evidence: B*)
2. **2011 New Recommendation:** For patients with limb-threatening ischemia and an estimated life expectancy of more than 2 years, bypass surgery, when possible and when an autogenous vein conduit is available, is reasonable to perform as the initial treatment to improve distal blood flow. (*Level of Evidence: B*)

2.3.3.3. THROMBOLYSIS FOR ACUTE AND CLI

**CLASS I**

1. Catheter-based thrombolysis is an effective and beneficial therapy and is indicated for patients with acute limb ischemia (Rutherford categories I and IIa) of less than 14 days' duration. (*Level of Evidence: A*)

**CLASS IIa**

1. Mechanical thrombectomy devices can be used as adjunctive therapy for acute limb ischemia due to peripheral arterial occlusion. (*Level of Evidence: B*)

**CLASS IIb**

1. Catheter-based thrombolysis orthombectomy may be considered for patients with acute limb ischemia (Rutherford category IIb) of more than 14 days' duration. (*Level of Evidence: B*)

2.3.3.4. SURGERY FOR CLI

**CLASS I**

1. For individuals with combined inflow and outflow disease with CLI, inflow lesions should be addressed first. (*Level of Evidence: B*)
2. For individuals with combined inflow and outflow disease in whom symptoms of CLI or infection persist after inflow revascularization, an outflow revascularization procedure should be performed. (*Level of Evidence: B*)
3. Patients who have significant necrosis of the weight-bearing portions of the foot (in ambulatory patients), an uncorrectable flexion contracture, paresis of the extremity, refractory ischemic rest pain, sepsis, or a very limited life expectancy due to comorbid conditions should be evaluated for primary amputation of the leg. (*Level of Evidence: C*)

**CLASS III**

1. Surgical and endovascular intervention is not indicated in patients with severe decrements in limb perfusion (e.g., ABI <0.4) in the absence of clinical symptoms of CLI. (*Level of Evidence: C*)

2.3.3.4.1. INFLOW PROCEDURES: AORTOILIAC OCCLUSIVE DISEASE

**CLASS I**

1. When surgery is to be undertaken, aortobifemoral bypass is recommended for patients with symptomatic, hemodynamically

significant, aortobilliic disease requiring intervention. (*Level of Evidence: A*)

2. Iliac endarterectomy, patch angioplasty, or aortoiliac or iliofemoral bypass in the setting of acceptable aortic inflow should be used for the treatment of unilateral disease or in conjunction with femoral-femoral bypass for the treatment of a patient with bilateral iliac artery occlusive disease if the patient is not a suitable candidate for aortobifemoral bypass grafting. (*Level of Evidence: B*)
3. Axillofemoral-femoral bypass is indicated for the treatment of patients with CLI who have extensive aortoiliac disease and are not candidates for other types of intervention. (*Level of Evidence: B*)

#### 2.3.3.4.2. OUTFLOW PROCEDURES: INFRAINGUINAL DISEASE

##### CLASS I

1. Bypasses to the above-knee popliteal artery should be constructed with autogenous saphenous vein when possible. (*Level of Evidence: A*)
2. Bypasses to the below-knee popliteal artery should be constructed with autogenous vein when possible. (*Level of Evidence: A*)
3. The most distal artery with continuous flow from above and without a stenosis greater than 20% should be used as the point of origin for a distal bypass. (*Level of Evidence: B*)
4. The tibial or pedal artery that is capable of providing continuous and uncompromised outflow to the foot should be used as the site of distal anastomosis. (*Level of Evidence: B*)
5. Femoral-tibial artery bypasses should be constructed with autogenous vein, including the ipsilateral greater saphenous vein, or if unavailable, other sources of vein from the leg or arm. (*Level of Evidence: B*)
6. Composite sequential femoropopliteal-tibial bypass and bypass to an isolated popliteal arterial segment that has collateral outflow to the foot are both acceptable methods of revascularization and should be considered when no other form of bypass with adequate autogenous conduit is possible. (*Level of Evidence: B*)
7. If no autogenous vein is available, a prosthetic femoral-tibial bypass, and possibly an adjunctive procedure, such as arteriovenous fistula or vein interposition or cuff, should be used when amputation is imminent. (*Level of Evidence: B*)

##### CLASS IIa

1. Prosthetic material can be used effectively for bypasses to the below-knee popliteal artery when no autogenous vein from ipsilateral or contralateral leg or arms is available. (*Level of Evidence: B*)

#### 2.3.3.4.3. POSTSURGICAL CARE

##### CLASS I

1. Unless contraindicated, all patients undergoing revascularization for CLI should be placed on antiplatelet therapy, and this treatment should be continued indefinitely. (*Level of Evidence: A*)
2. Patients who have undergone placement of aortobifemoral bypass grafts should be followed up with periodic evaluations that record any return or progression of ischemic symptoms, the presence of femoral pulses, and ABIs. (*Level of Evidence: B*)
3. If infection, ischemic ulcers, or gangrenous lesions persist and the ABI is less than 0.8 after correction of inflow, an outflow procedure should be performed that bypasses all major distal stenoses and occlusions. (*Level of Evidence: A*)
4. Patients who have undergone placement of a lower extremity bypass with autogenous vein should undergo for at least 2 years

periodic examinations that record any return or progression of ischemic symptoms; a physical examination, with concentration on pulse examination of the proximal, graft, and outflow vessels; and duplex imaging of the entire length of the graft, with measurement of peak systolic velocities and calculation of velocity ratios across all lesions. (*Level of Evidence: A*)

5. Patients who have undergone placement of a synthetic lower extremity bypass graft should undergo periodic examinations that record any return of ischemic symptoms; a pulse examination of the proximal, graft, and outflow vessels; and assessment of ABIs at rest and after exercise for at least 2 years after implantation. (*Level of Evidence: A*)

### 3. Renal Arterial Disease: Recommendations

#### 3.1. Clinical Clues to the Diagnosis of Renal Artery Stenosis

##### CLASS I

1. The performance of diagnostic studies to identify clinically significant renal artery stenosis (RAS) is indicated in patients with the onset of hypertension before the age of 30 years. (*Level of Evidence: B*)
2. The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with the onset of severe hypertension [as defined in The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC-7 report] after the age of 55 years. (*Level of Evidence: B*)
3. The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with the following characteristics: (a) accelerated hypertension (sudden and persistent worsening of previously controlled hypertension); (b) resistant hypertension (defined as the failure to achieve goal blood pressure in patients who are adhering to full doses of an appropriate 3-drug regimen that includes a diuretic); or (c) malignant hypertension (hypertension with coexistent evidence of acute end-organ damage, i.e., acute renal failure, acutely decompensated congestive heart failure, new visual or neurological disturbance, and/or advanced [grade III to IV] retinopathy). (*Level of Evidence: C*)
4. The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with new azotemia or worsening renal function after the administration of an ACE inhibitor or an angiotensin receptor blocking agent. (*Level of Evidence: B*)
5. The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with an unexplained atrophic kidney or a discrepancy in size between the 2 kidneys of greater than 1.5 cm. (*Level of Evidence: B*)
6. The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with sudden, unexplained pulmonary edema (especially in azotemic patients). (*Level of Evidence: B*)

##### CLASS IIa

1. The performance of diagnostic studies to identify clinically significant RAS is reasonable in patients with unexplained renal failure, including individuals starting renal replacement therapy (dialysis or renal transplantation). (*Level of Evidence: B*)

##### CLASS IIb

1. The performance of arteriography to identify significant RAS may be reasonable in patients with multivessel coronary artery dis-

ease and none of the clinical clues or PAD at the time of arteriography. (*Level of Evidence: B*)

2. The performance of diagnostic studies to identify clinically significant RAS may be reasonable in patients with unexplained congestive heart failure or refractory angina. (*Level of Evidence: C*)

## 3.2. Diagnostic Methods

### CLASS I

1. Duplex ultrasonography is recommended as a screening test to establish the diagnosis of RAS. (*Level of Evidence: B*)
2. CTA (in individuals with normal renal function) is recommended as a screening test to establish the diagnosis of RAS. (*Level of Evidence: B*)
3. MRA is recommended as a screening test to establish the diagnosis of RAS. (*Level of Evidence: B*)
4. When the clinical index of suspicion is high and the results of noninvasive tests are inconclusive, catheter angiography is recommended as a diagnostic test to establish the diagnosis of RAS. (*Level of Evidence: B*)

### CLASS III

1. Captopril renal scintigraphy is not recommended as a screening test to establish the diagnosis of RAS. (*Level of Evidence: C*)
2. Selective renal vein renin measurements are not recommended as a useful screening test to establish the diagnosis of RAS. (*Level of Evidence: B*)
3. Plasma renin activity is not recommended as a useful screening test to establish the diagnosis of RAS. (*Level of Evidence: B*)
4. The captopril test (measurement of plasma renin activity after captopril administration) is not recommended as a useful screening test to establish the diagnosis of RAS. (*Level of Evidence: B*)

## 3.3. Treatment of Renovascular Disease: RAS

### 3.3.1. Medical Treatment

#### CLASS I

1. ACE inhibitors are effective medications for treatment of hypertension associated with unilateral RAS. (*Level of Evidence: A*)
2. Angiotensin receptor blockers are effective medications for treatment of hypertension associated with unilateral RAS. (*Level of Evidence: B*)
3. Calcium-channel blockers are effective medications for treatment of hypertension associated with unilateral RAS. (*Level of Evidence: A*)
4. Beta blockers are effective medications for treatment of hypertension associated with RAS. (*Level of Evidence: A*)

### 3.3.2. Indications for Revascularization

#### 3.3.2.1. ASYMPTOMATIC STENOSIS

#### CLASS IIb

1. Percutaneous revascularization may be considered for treatment of an asymptomatic bilateral or solitary viable kidney with a hemodynamically significant RAS. (*Level of Evidence: C*)
2. The usefulness of percutaneous revascularization of an asymptomatic unilateral hemodynamically significant RAS in a viable kidney is not well established and is presently clinically unproven. (*Level of Evidence: C*)

#### 3.3.2.2. HYPERTENSION

#### CLASS IIa

1. Percutaneous revascularization is reasonable for patients with hemodynamically significant RAS and accelerated hypertension, resistant hypertension, malignant hypertension, hypertension with an unexplained unilateral small kidney, and hypertension with intolerance to medication. (*Level of Evidence: B*)

#### 3.3.2.3. PRESERVATION OF RENAL FUNCTION

#### CLASS IIa

1. Percutaneous revascularization is reasonable for patients with RAS and progressive chronic kidney disease with bilateral RAS or a RAS to a solitary functioning kidney. (*Level of Evidence: B*)

#### CLASS IIb

1. Percutaneous revascularization may be considered for patients with RAS and chronic renal insufficiency with unilateral RAS. (*Level of Evidence: C*)

#### 3.3.2.4. IMPACT OF RAS ON CONGESTIVE HEART FAILURE AND UNSTABLE ANGINA

#### CLASS I

1. Percutaneous revascularization is indicated for patients with hemodynamically significant RAS and recurrent, unexplained congestive heart failure or sudden, unexplained pulmonary edema. (*Level of Evidence: B*)

#### CLASS IIa

1. Percutaneous revascularization is reasonable for patients with hemodynamically significant RAS and unstable angina. (*Level of Evidence: B*)

### 3.3.3. Endovascular Treatment for RAS

#### CLASS I

1. Renal stent placement is indicated for ostial atherosclerotic RAS lesions that meet the clinical criteria for intervention. (*Level of Evidence: B*)
2. Balloon angioplasty with bailout stent placement if necessary is recommended for fibromuscular dysplasia lesions. (*Level of Evidence: B*)

### 3.3.4. Surgery for RAS

#### CLASS I

1. Vascular surgical reconstruction is indicated for patients with fibromuscular dysplastic RAS with clinical indications for interventions (same as for percutaneous transluminal angioplasty), especially those exhibiting complex disease that extends into the segmental arteries and those having macroaneurysms. (*Level of Evidence: B*)
2. Vascular surgical reconstruction is indicated for patients with atherosclerotic RAS and clinical indications for intervention, especially those with multiple small renal arteries or early primary branching of the main renal artery. (*Level of Evidence: B*)
3. Vascular surgical reconstruction is indicated for patients with atherosclerotic RAS in combination with pararenal aortic reconstructions (in treatment of aortic aneurysms or severe aortoiliac occlusive disease). (*Level of Evidence: C*)

## 4. Mesenteric Arterial Disease: Recommendations

### 4.1. Acute Intestinal Ischemia

#### 4.1.1. Acute Intestinal Ischemia Caused by Arterial Obstruction

##### 4.1.1.1. DIAGNOSIS

###### CLASS I

1. Patients with acute abdominal pain out of proportion to physical findings and who have a history of cardiovascular disease should be suspected of having acute intestinal ischemia. (*Level of Evidence: B*)
2. Patients who develop acute abdominal pain after arterial interventions in which catheters traverse the visceral aorta or any proximal arteries or who have arrhythmias (such as atrial fibrillation) or recent MI should be suspected of having acute intestinal ischemia. (*Level of Evidence: C*)

###### CLASS III

1. In contrast to chronic intestinal ischemia, duplex sonography of the abdomen is not an appropriate diagnostic tool for suspected acute intestinal ischemia. (*Level of Evidence: C*)

##### 4.1.1.2. SURGICAL TREATMENT

###### CLASS I

1. Surgical treatment of acute obstructive intestinal ischemia includes revascularization, resection of necrotic bowel, and, when appropriate, a “second look” operation 24 to 48 hours after the revascularization. (*Level of Evidence: B*)

##### 4.1.1.3. ENDOVASCULAR TREATMENT

###### CLASS IIb

1. Percutaneous interventions (including transcatheter lytic therapy, balloon angioplasty, and stenting) are appropriate in selected patients with acute intestinal ischemia caused by arterial obstructions. Patients so treated may still require laparotomy. (*Level of Evidence: C*)

#### 4.1.2. Acute Nonocclusive Intestinal Ischemia

##### 4.1.2.1. ETIOLOGY

###### CLASS I

1. Nonocclusive intestinal ischemia should be suspected in patients with low flow states or shock, especially cardiogenic shock, who develop abdominal pain. (*Level of Evidence: B*)
2. Nonocclusive intestinal ischemia should be suspected in patients receiving vasoconstrictor substances and medications (e.g., cocaine, ergots, vasopressin, or norepinephrine) who develop abdominal pain. (*Level of Evidence: B*)
3. Nonocclusive intestinal ischemia should be suspected in patients who develop abdominal pain after coarctation repair or after surgical revascularization for intestinal ischemia caused by arterial obstruction. (*Level of Evidence: B*)

##### 4.1.2.2. DIAGNOSIS

###### CLASS I

1. Arteriography is indicated in patients suspected of having nonocclusive intestinal ischemia whose condition does not improve

rapidly with treatment of their underlying disease. (*Level of Evidence: B*)

##### 4.1.2.3. TREATMENT

###### CLASS I

1. Treatment of the underlying shock state is the most important initial step in treatment of nonocclusive intestinal ischemia. (*Level of Evidence: C*)
2. Laparotomy and resection of nonviable bowel is indicated in patients with nonocclusive intestinal ischemia who have persistent symptoms despite treatment. (*Level of Evidence: B*)

###### CLASS IIa

1. Transcatheter administration of vasodilator medications into the area of vasospasm is indicated in patients with nonocclusive intestinal ischemia who do not respond to systemic supportive treatment and in patients with intestinal ischemia due to cocaine or ergot poisoning. (*Level of Evidence: B*)

### 4.2. Chronic Intestinal Ischemia

#### 4.2.1. Diagnosis

###### CLASS I

1. Chronic intestinal ischemia should be suspected in patients with abdominal pain and weight loss without other explanation, especially those with cardiovascular disease. (*Level of Evidence: B*)
2. Duplex ultrasound, CTA, and gadolinium-enhanced MRA are useful initial tests for supporting the clinical diagnosis of chronic intestinal ischemia. (*Level of Evidence: B*)
3. Diagnostic angiography, including lateral aortography, should be obtained in patients suspected of having chronic intestinal ischemia for whom noninvasive imaging is unavailable or indeterminate. (*Level of Evidence: B*)

#### 4.2.2. Endovascular Treatment for Chronic Intestinal Ischemia

###### CLASS I

1. Percutaneous endovascular treatment of intestinal arterial stenosis is indicated in patients with chronic intestinal ischemia. (*Level of Evidence: B*)

#### 4.2.3. Surgical Treatment

###### CLASS I

1. Surgical treatment of chronic intestinal ischemia is indicated in patients with chronic intestinal ischemia. (*Level of Evidence: B*)

###### CLASS IIb

1. Revascularization of asymptomatic intestinal arterial obstructions may be considered for patients undergoing aortic/renal artery surgery for other indications. (*Level of Evidence: B*)

###### CLASS III

1. Surgical revascularization is not indicated for patients with asymptomatic intestinal arterial obstructions, except in patients undergoing aortic/renal artery surgery for other indications. (*Level of Evidence: B*)



## 5. Aneurysms of the Abdominal Aorta, Its Branch Vessels, and the Lower Extremities: Recommendations

### 5.1. Abdominal Aortic and Iliac Aneurysms

#### 5.1.1. Etiology

##### 5.1.1.1. ATHEROSCLEROTIC RISK FACTORS

###### CLASS I

1. In patients with AAAs, blood pressure and fasting serum lipid values should be monitored and controlled as recommended for patients with atherosclerotic disease. *(Level of Evidence: C)*
2. Patients with aneurysms or a family history of aneurysms should be advised to stop smoking and be offered smoking cessation interventions, including behavior modification, nicotine replacement, or bupropion. *(Level of Evidence: B)*

#### 5.1.2. Natural History

##### 5.1.2.1. AORTIC ANEURYSM RUPTURE

###### CLASS I

1. Patients with infrarenal or juxtarenal AAAs measuring 5.5 cm or larger should undergo repair to eliminate the risk of rupture. *(Level of Evidence: B)*
2. Patients with infrarenal or juxtarenal AAAs measuring 4.0 to 5.4 cm in diameter should be monitored by ultrasound or computed tomographic scans every 6 to 12 months to detect expansion. *(Level of Evidence: A)*

###### CLASS IIa

1. Repair can be beneficial in patients with infrarenal or juxtarenal AAAs 5.0 to 5.4 cm in diameter. *(Level of Evidence: B)*
2. Repair is probably indicated in patients with suprarenal or type IV thoracoabdominal aortic aneurysms larger than 5.5 to 6.0 cm. *(Level of Evidence: B)*
3. In patients with AAAs smaller than 4.0 cm in diameter, monitoring by ultrasound examination every 2 to 3 years is reasonable. *(Level of Evidence: B)*

###### CLASS III

1. Intervention is not recommended for asymptomatic infrarenal or juxtarenal AAAs if they measure less than 5.0 cm in diameter in men or less than 4.5 cm in diameter in women. *(Level of Evidence: A)*

#### 5.1.3. Diagnosis

##### 5.1.3.1. SYMPTOMATIC AORTIC OR ILIAC ANEURYSMS

###### CLASS I

1. In patients with the clinical triad of abdominal and/or back pain, a pulsatile abdominal mass, and hypotension, immediate surgical evaluation is indicated. *(Level of Evidence: B)*
2. In patients with symptomatic aortic aneurysms, repair is indicated regardless of diameter. *(Level of Evidence: C)*

##### 5.1.3.2. SCREENING HIGH-RISK POPULATIONS

###### CLASS I

1. Men 60 years of age or older who are either the siblings or offspring of patients with AAAs should undergo physical examination and ultrasound screening for detection of aortic aneurysms. *(Level of Evidence: B)*

###### CLASS IIa

1. Men who are 65 to 75 years of age who have ever smoked should undergo a physical examination and 1-time ultrasound screening for detection of AAAs. *(Level of Evidence: B)*

#### 5.1.4. Observational Management

##### 5.1.4.1. BLOOD PRESSURE CONTROL AND BETA-BLOCKADE

###### CLASS I

1. Perioperative administration of beta-adrenergic blocking agents, in the absence of contraindications, is indicated to reduce the risk of adverse cardiac events and mortality in patients with coronary artery disease undergoing surgical repair of atherosclerotic aortic aneurysms. *(Level of Evidence: A)*

###### CLASS IIb

1. Beta-adrenergic blocking agents may be considered to reduce the rate of aneurysm expansion in patients with aortic aneurysms. *(Level of Evidence: B)*

#### 5.1.5. Prevention of Aortic Aneurysm Rupture

##### 5.1.5.1. MANAGEMENT OVERVIEW

###### CLASS I

1. **2011 Updated Recommendation:** Open or endovascular repair of infrarenal AAAs and/or common iliac aneurysms is indicated in patients who are good surgical candidates. *(Level of Evidence: A)*
2. **2011 Updated Recommendation:** Periodic long-term surveillance imaging should be performed to monitor for an endoleak, to document shrinkage or stability of the excluded aneurysm sac, and to determine the need for further intervention in patients who have undergone endovascular repair of infrarenal aortic and/or iliac aneurysms. *(Level of Evidence: A)*

###### CLASS IIa

1. **2011 New Recommendation:** Open aneurysm repair is reasonable to perform in patients who are good surgical candidates but who cannot comply with the periodic long-term surveillance required after endovascular repair. *(Level of Evidence: C)*

###### CLASS IIb

1. **2011 New Recommendation:** Endovascular repair of infrarenal aortic aneurysms in patients who are at high surgical or anesthetic risk as determined by the presence of coexisting severe cardiac, pulmonary, and/or renal disease is of uncertain effectiveness. *(Level of Evidence: B)*

### 5.2. Visceral Artery Aneurysms

###### CLASS I

1. Open repair or catheter-based intervention is indicated for visceral aneurysms measuring 2.0 cm in diameter or larger in women of childbearing age who are not pregnant and in patients of either gender undergoing liver transplantation. *(Level of Evidence: B)*

###### CLASS IIa

1. Open repair or catheter-based intervention is probably indicated for visceral aneurysms 2.0 cm in diameter or larger in women beyond childbearing age and in men. *(Level of Evidence: B)*

### 5.3. Lower Extremity Aneurysms

#### 5.3.1. Natural History

###### CLASS I

1. In patients with femoral or popliteal aneurysms, ultrasound (or computed tomography or magnetic resonance) imaging is rec-

ommended to exclude contralateral femoral or popliteal aneurysms and AAA. (Level of Evidence: B)

### 5.3.2. Management

#### CLASS I

1. Patients with a palpable popliteal mass should undergo an ultrasound examination to exclude popliteal aneurysm. (Level of Evidence: B)
2. Patients with popliteal aneurysms 2.0 cm in diameter or larger should undergo repair to reduce the risk of thromboembolic complications and limb loss. (Level of Evidence: B)
3. Patients with anastomotic pseudoaneurysms or symptomatic femoral artery aneurysms should undergo repair. (Level of Evidence: A)

#### CLASS IIa

1. Surveillance by annual ultrasound imaging is suggested for patients with asymptomatic femoral artery true aneurysms smaller than 3.0 cm in diameter. (Level of Evidence: C)
2. In patients with acute ischemia and popliteal artery aneurysms and absent runoff, catheter-directed thrombolysis or mechanical thrombectomy (or both) is suggested to restore distal runoff and resolve emboli. (Level of Evidence: B)

3. In patients with asymptomatic enlargement of the popliteal arteries twice the normal diameter for age and gender, annual ultrasound monitoring is reasonable. (Level of Evidence: C)
4. In patients with femoral or popliteal artery aneurysms, administration of antiplatelet medication may be beneficial. (Level of Evidence: C)

#### 5.3.2.1. CATHETER-RELATED FEMORAL ARTERY PSEUDOANEURYSMS

#### CLASS I

1. Patients with suspected femoral pseudoaneurysms should be evaluated by duplex ultrasonography. (Level of Evidence: B)
2. Initial treatment with ultrasound-guided compression or thrombin injection is recommended in patients with large and/or symptomatic femoral artery pseudoaneurysms. (Level of Evidence: B)

#### CLASS IIa

1. Surgical repair is reasonable in patients with femoral artery pseudoaneurysms 2.0 cm in diameter or larger that persist or recur after ultrasound-guided compression or thrombin injection. (Level of Evidence: B)
2. Reevaluation by ultrasound 1 month after the original injury can be useful in patients with asymptomatic femoral artery pseudoaneurysms smaller than 2.0 cm in diameter. (Level of Evidence: B)

## APPENDIX 1. AUTHOR RELATIONSHIPS WITH INDUSTRY (RELEVANT)—2005 ACC/AHA WRITING COMMITTEE TO DEVELOP GUIDELINES ON PERIPHERAL ARTERIAL DISEASE

Committee Member	Research Grant	Speakers Bureau/Honoraria	Stock Ownership	Consultant	Advisory Board
Curtis W. Bakal	None	None	None	None	<ul style="list-style-type: none"> <li>Abbott Labs</li> <li>Berlex Labs</li> </ul>
Mark A. Creager	<ul style="list-style-type: none"> <li>Eli Lilly</li> <li>Otsuka Pharmaceuticals</li> <li>Pfizer</li> <li>Vasogen</li> </ul>	<ul style="list-style-type: none"> <li>Bristol-Myers Squibb/Sanofi</li> <li>Otsuka Pharmaceuticals</li> </ul>	<ul style="list-style-type: none"> <li>Northport Domain</li> </ul>	None	<ul style="list-style-type: none"> <li>Bristol-Myers Squibb/Sanofi</li> <li>Genvec</li> <li>Geozyme</li> <li>Northport Domain</li> <li>Otsuka Pharmaceuticals</li> <li>Pfizer</li> <li>Vasogen</li> </ul>
Jonathan L. Halperin	None	<ul style="list-style-type: none"> <li>AstraZeneca</li> <li>Bristol-Myers Squibb/Sanofi</li> </ul>	None	<ul style="list-style-type: none"> <li>AstraZeneca</li> <li>Bayer AG</li> <li>Boehringer Ingelheim</li> <li>Bristol-Myers Squibb/Sanofi</li> </ul>	<ul style="list-style-type: none"> <li>AstraZeneca</li> </ul>
Ziv J. Haskal	<ul style="list-style-type: none"> <li>Bard/Impira</li> <li>Boston Scientific</li> <li>Cook</li> <li>Cordis Endovascular</li> <li>Genetech</li> <li>IntraTherapeutics</li> <li>W.L. Gore</li> </ul>	<ul style="list-style-type: none"> <li>TransVascular</li> <li>W.L. Gore</li> </ul>	None	<ul style="list-style-type: none"> <li>Bard/Impira</li> <li>Endosurgery</li> <li>Ethicon</li> <li>Omnisonics</li> <li>TransVascular</li> </ul>	<ul style="list-style-type: none"> <li>TransVascular</li> </ul>
Norman R. Hertzner	None	None	None	None	None
Loren F. Hiratzka	None	None	None	None	None

Committee Member	Research Grant	Speakers Bureau/ Honoraria	Stock Ownership	Consultant	Advisory Board
Alan T. Hirsch	<ul style="list-style-type: none"> <li>Alteon</li> <li>AstraZeneca</li> <li>Bristol-Myers Squibb/Sanofi Aventis</li> <li>Kos Pharmaceuticals</li> <li>Otsuka America Pharmaceuticals</li> </ul>	<ul style="list-style-type: none"> <li>AstraZeneca</li> <li>Bristol-Myers Squibb/Sanofi Aventis Partnership</li> <li>Otsuka America Pharmaceuticals</li> <li>Pfizer</li> </ul>	None	<ul style="list-style-type: none"> <li>Sonosite</li> <li>Vasogen</li> </ul>	None
William R. C. Murphy	None	None	None	None	None
Jeffrey W. Olin	<ul style="list-style-type: none"> <li>Bristol-Myers Squibb/Sanofi Partnership</li> <li>Vasogen</li> </ul>	None	None	<ul style="list-style-type: none"> <li>Aventia</li> <li>Bristol-Myers Squibb/Sanofi Partnership</li> <li>Genzyme</li> <li>Otsuka</li> <li>Vasogen</li> </ul>	<ul style="list-style-type: none"> <li>Abbott</li> <li>Aventis</li> <li>Bristol-Myers Squibb/Sanofi Partnership</li> <li>Genzyme</li> </ul>
Jules B. Puschett	None	None	None	None	None
Kenneth A. Rosenfield	<ul style="list-style-type: none"> <li>Abbott</li> <li>Boston Scientific</li> <li>Cordis</li> <li>Guidant</li> </ul>	<ul style="list-style-type: none"> <li>Eli Lilly</li> </ul>	<ul style="list-style-type: none"> <li>CryoVascular</li> </ul>	<ul style="list-style-type: none"> <li>Abbott</li> <li>Boston Scientific</li> <li>Cordis</li> <li>CryoVascular</li> <li>Guidant</li> </ul>	<ul style="list-style-type: none"> <li>Abbott</li> <li>Boston Scientific</li> <li>Cordis</li> <li>Guidant</li> </ul>
David Sacks	None	None	<ul style="list-style-type: none"> <li>Angiotech</li> </ul>	None	None
James C. Stanley	None	None	None	None	None
Lloyd M. Taylor, Jr	None	None	None	None	None
Christopher J. White	None	<ul style="list-style-type: none"> <li>Eli Lilly</li> </ul>	None	None	None
John White	None	None	None	None	None
Rodney A. White	<ul style="list-style-type: none"> <li>AVE Bard</li> <li>Baxter</li> <li>Cordis J&amp;J</li> <li>EndoLogix</li> <li>EndoSonics</li> <li>Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>Multiple relationships with commercial entities that arise and are met as needed</li> </ul>	<ul style="list-style-type: none"> <li>Several biomedical companies</li> </ul>	None	None

This table represents the relationships of committee members with industry that were disclosed at the initial writing committee meeting in November 2002 and that were updated in conjunction with all meetings and conference calls of the writing committee. It does not necessarily reflect relationships with industry at the time of publication.

## APPENDIX 2. AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (RELEVANT)—2011 ACC/AHA FOCUSED UPDATE OF THE GUIDELINE FOR THE MANAGEMENT OF PATIENTS WITH PERIPHERAL ARTERY DISEASE

Writing Group Member	Employment	Consultant	Speakers' Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusal (by section)*
Thom W. Rooke, Chair	Mayo Clinic—Professor of Medicine	None	None	None	None	None	None	None
Alan T. Hirsch, Vice Chair	University of Minnesota Medical School: Cardiovascular Division—Vascular Medicine Program: Director; Professor of Medicine: Epidemiology and Community Health	<ul style="list-style-type: none"> <li>eV3</li> </ul>	None	None	<ul style="list-style-type: none"> <li>Abbott Vascular</li> <li>BMS/sanofi-aventis</li> <li>Cytokinetics</li> <li>Sanofi-aventis</li> <li>ViroMed (PI)</li> </ul>	None	None	2.5.1 2.6.1.6 2.6.3
Sanjay Misra, Vice Chair	Mayo Clinic: Division of Vascular and Interventional Radiology—Associate Professor of Radiology	<ul style="list-style-type: none"> <li>Johnson &amp; Johnson</li> </ul>	None	None	None	None	None	2.6.3

Writing Group Member	Employment	Consultant	Speakers' Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusal (by section)*
Anton N. Sidawy, Vice Chair	George Washington University—Professor and Chairman, Department of Surgery	None	None	None	None	None	None	None
Joshua A. Beckman	Brigham and Women's Hospital Cardiovascular Division: Cardiovascular Fellowship	• Bristol-Myers Squibb • Sanofi-aventis	None	None	None	None	None	2.6.1.6
Laura K. Findeiss	University of California, Irvine: Chief, Division of Vascular and Interventional Radiology—Associate Professor of Radiology and Surgery	None	None	None	None	None	None	None
Jafar Golzarian	University of Minnesota Medical School—Professor of Radiology and Surgery	None	None	None	None	None	None	None
Gregory L. Moneta	Oregon Health & Science University—Chief and Professor of Vascular Surgery	None	None	None	None	None	None	None
Jeffrey W. Olin	Mount Sinai School of Medicine—Professor of Medicine and Director of the Vascular Medicine Program	• Genzyme	None	None	• BMS/sanofi-aventis • Colorado Prevention Center (DSMB)	None	• Defendant; pulmonary embolism; 2009	2.6.1.6
James C. Stanley	University of Michigan, Division of Vascular Surgery, University Hospital—Handleman Professor of Surgery	None	None	None	None	None	None	None
Christopher J. White	Ochsner Clinical Foundation: Department of Cardiology—Chairman	None	None	None	• Boston Scientific • Neovasc • St. Jude Medical	None	None	2.6.3 5.2.6
John V. White	Advocate Lutheran General Hospital—Chief of Surgery	None	None	None	None	None	None	None
R. Eugene Zierler	University of Washington—Professor of Surgery	None	None	None	None	None	None	None

This table represents the relationships of writing group members with industry and other entities that were determined to be relevant to this document. These relationships were reviewed and updated in conjunction with all meetings and/or conference calls of the writing group during the document development process.

The table does not necessarily reflect relationships with industry at the time of publication. A person is deemed to have a significant interest in a business if the interest represents ownership of >5% of the voting stock or share of the business entity, or ownership of \$10,000 of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person's gross income for the previous year. Relationships that exist with no financial benefit are also included for the purpose of transparency. Relationships in this table are modest unless otherwise noted.

According to the ACCF/AHA, a person has a relevant relationship IF: (a) The relationship or interest relates to the same or similar subject matter, intellectual property or asset, topic, or issue addressed in the document; or (b) the company/entity (with whom the relationship exists) makes a drug, drug class, or device addressed in the document, or makes a competing drug or device addressed in the document; or (c) the person or a member of the person's household, has a reasonable potential for financial, professional or other personal gain or loss as a result of the issues/content addressed in the document.

\*Writing group members are required to recuse themselves from voting on sections to which their specific relationships with industry and other entities may apply. Section numbers are from the 2011 Focused Update.

†Significant relationship.

‡No financial benefit.

DSMB indicates Data and Safety Monitoring Board; and PI, principal investigator.