



AMERICAN
COLLEGE *of*
CARDIOLOGY
FOUNDATION



American
Heart
Association®

**ACCF/AHA Pocket Guideline
November 2011**

**Management
of Patients With
Peripheral Artery
Disease
(Lower Extremity,
Renal, Mesenteric, and
Abdominal Aortic)**

**Adapted from the 2005
ACCF/AHA Guideline and the
2011 ACCF/AHA Focused Update**

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

© 2011 by the American College of Cardiology Foundation and the American Heart Association, Inc.

The following material was adapted from the 2011 ACCF/AHA focused update of the guideline for the management of patients with peripheral artery disease *J Am Coll Cardiol* 2011; 58:2020-2045 and the 2005 ACC/AHA guidelines for the management of the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic) *J Am Coll Cardiol* 2006;47:1239-312. This pocket guideline is available on the World Wide Web sites of the American College of Cardiology (cardiosource.org) and the American Heart Association (my.americanheart.org).

For copies of this document, please contact Elsevier Inc. Reprint Department, e-mail: reprints@elsevier.com; phone: 212-633-3813; fax: 212-633-3820.

Permissions: Multiple copies, modification, alteration, enhancement, and/or distribution of this document are not permitted without the express permission of the American College of Cardiology Foundation. Please contact Elsevier's permission department at healthpermissions@elsevier.com.

Contents

1. Introduction	3	
2. Patient History and Physical Examination: Fundamental Principles	6	
3. Evaluation and Treatment of Patients With, or at Risk for, PAD	9	
4. Lower Extremity Arterial Disease	11	Lower Extremity
A. Claudication.....	11	
B. Critical Limb Ischemia (UPDATED).....	25	
C. Acute Limb Ischemia.....	28	
D. Surveillance for Patients After Lower Extremity Revascularization.....	30	
E. Ankle-Brachial Index, Toe-Brachial Index, and Segmental Pressure Examination (UPDATED).....	31	
F. Smoking Cessation (UPDATED).....	33	
G. Antithrombotic and Antiplatelet Therapy (UPDATED).....	33	
5. Renal Arterial Disease	35	Renal
A. Clinical Indications.....	35	
B. Diagnostic Methods.....	38	
C. Indications for Revascularization of Patients with Hemodynamically Significant RAS.....	39	
D. Treatment Methods: Medical, Endovascular, and Surgical.....	42	
6. Mesenteric Arterial Disease	45	Mesenteric
A. Acute Intestinal Ischemia.....	45	
B. Acute Nonocclusive Intestinal Ischemia.....	46	
C. Chronic Intestinal Ischemia.....	48	
7. Aneurysms of the Abdominal Aorta, Its Branch Vessels, and the Lower Extremities	49	Abdominal
A. Abdominal Aortic Aneurysms.....	49	
B. Management Overview of Prevention of Aortic Aneurysm Rupture (UPDATED).....	53	
C. Visceral Arterial Aneurysms.....	55	
D. Lower Extremity Arterial Aneurysms.....	57	
E. Femoral Artery Pseudoaneurysms.....	59	

1. Introduction

This pocket guide provides rapid prompts for appropriate patient management, which is outlined in much greater detail in the full-text guidelines. It is not intended as a replacement for understanding the caveats and rationales that are stated carefully in the full-text guidelines. Users should consult the full-text guideline for more information.

The term peripheral artery disease (PAD) broadly encompasses the vascular diseases caused primarily by atherosclerosis and thromboembolic pathophysiologic processes that alter the normal structure and function of the aorta, its visceral arterial branches, and the arteries of the lower extremity. PAD is the preferred clinical term and should be used to denote stenotic, occlusive and aneurysmal diseases of the aorta and its branch arteries, exclusive of the coronary arteries.

The scope of these pocket guidelines (updated for 2011) is limited to disorders of the lower extremity arteries, renal and mesenteric arteries, and disorders of the abdominal aorta. The purpose of these guidelines is to 1) aid in the recognition, diagnosis, and treatment of PAD of the lower extremities, and 2) highlight the prevalence, impact on quality-of-life, cardiovascular ischemic risk, and increased risk of critical limb ischemia (CLI) associated with PAD. Inasmuch as the burden of PAD is widespread, these guidelines are intended to assist all clinicians who might provide care for such patients, including primary care clinicians, vascular and cardiovascular specialists, trainees in the primary care and vascular specialties, as well as nurses, physical therapists, and rehabilitative personnel.

All recommendations provided in this document follow the format of previous American College of Cardiology Foundation/American Heart Association guidelines (*Table 1*). Recommendations that remain unchanged used the Class of Recommendation/Level of Evidence table from the 2005 guideline.

Table 1. Applying Classification of Recommendations and Level of Evidence[†]

		SIZE OF TREATMENT EFFECT	
		CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/administered	CLASS IIa <i>Benefit >> Risk</i> <i>Additional studies with focused objectives needed</i> IT IS REASONABLE to perform procedure/administer treatment
ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	<ul style="list-style-type: none"> ■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses 	<ul style="list-style-type: none"> ■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses
	LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	<ul style="list-style-type: none"> ■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> ■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies
	LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	<ul style="list-style-type: none"> ■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care 	<ul style="list-style-type: none"> ■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care
	Suggested phrases for writing recommendations	should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated
Comparative effectiveness phrases[†]	treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B	



Class IIb <i>Benefit ≥ Risk</i> <i>Additional studies with broad objectives needed; additional registry data would be helpful</i> Procedure/Treatment MAY BE CONSIDERED	Class III No Benefit or Class III Harm									
<ul style="list-style-type: none"> ■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randomized trials or meta-analyses 	<table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th></th> <th style="text-align: center;">Procedure/ Test</th> <th style="text-align: center;">Treatment</th> </tr> </thead> <tbody> <tr> <td style="text-align: center;">COR III: No Benefit</td> <td style="text-align: center;">Not Helpful</td> <td style="text-align: center;">No Proven Benefit</td> </tr> <tr> <td style="text-align: center;">COR III: Harm</td> <td style="text-align: center;">Excess Cost w/o Benefit or Harmful</td> <td style="text-align: center;">Harmful to Patients</td> </tr> </tbody> </table> <ul style="list-style-type: none"> ■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses 		Procedure/ Test	Treatment	COR III: No Benefit	Not Helpful	No Proven Benefit	COR III: Harm	Excess Cost w/o Benefit or Harmful	Harmful to Patients
	Procedure/ Test	Treatment								
COR III: No Benefit	Not Helpful	No Proven Benefit								
COR III: Harm	Excess Cost w/o Benefit or Harmful	Harmful to Patients								
<ul style="list-style-type: none"> ■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> ■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies 									
<ul style="list-style-type: none"> ■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard-of-care 	<ul style="list-style-type: none"> ■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard-of-care 									

*A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

may/might be considered
may/might be reasonable
usefulness/effectiveness is
unknown/unclear/uncertain
or not well established

COR III:
No Benefit

is not recommended
is not indicated
should not be performed/
administered/
other
is not useful/
beneficial/
effective

COR III:
Harm

potentially harmful
causes harm
associated with excess morbidity/
mortality
should not be performed/
administered/
done

2. Patient History and Physical Examination: Fundamental Principles

Identifying individuals at risk for lower extremity PAD is a fundamental part of the vascular review of systems (*Table 2, Figure 1*).

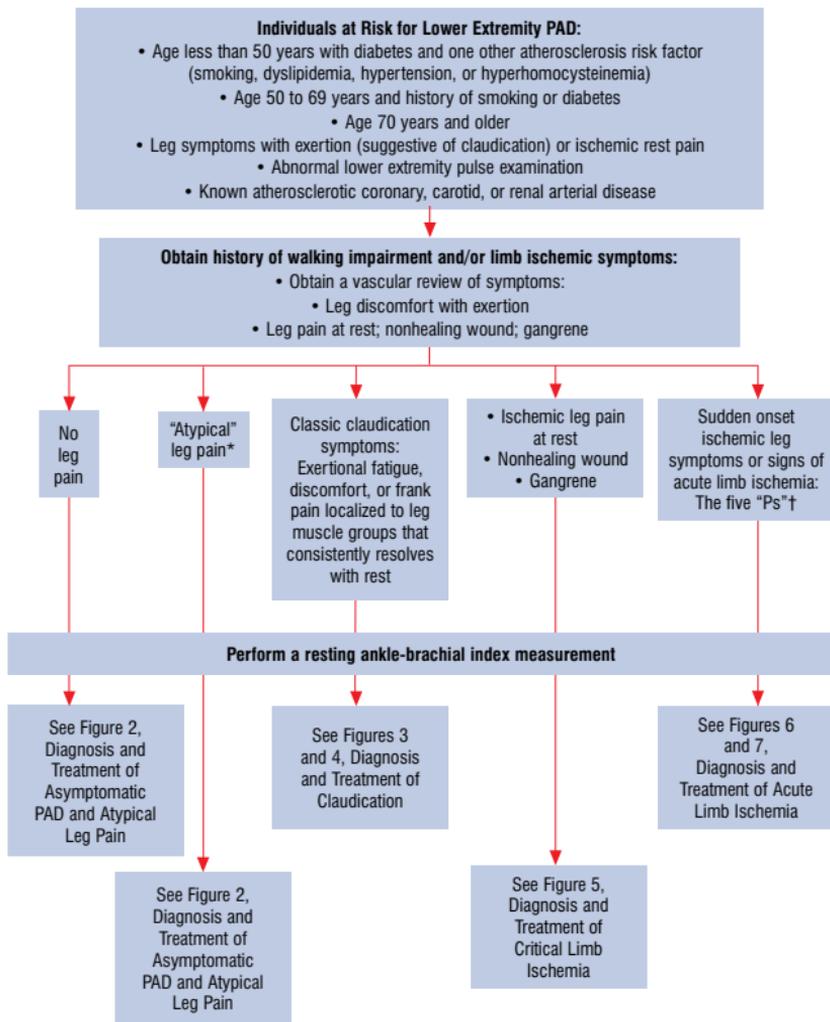
Table 2. Individuals at Risk for Lower Extremity Peripheral Arterial Disease

- Age less than 50 years, with diabetes and one other atherosclerosis risk factor (smoking, dyslipidemia, hypertension, or hyperhomocysteinemia)
- Age 50 to 69 and a history of smoking and diabetes
- Age 70 or older
- Leg symptoms with exertion (suggestive of claudication) or ischemic rest pain
- Abnormal lower extremity pulse examination
- Known atherosclerotic coronary, carotid, or renal artery disease

Key Components of the Vascular Review of Systems

- Any *exertional* limitation of the lower extremity muscles or any history of walking impairment (described as fatigue, aching, numbness, or pain, occurring in the buttock, thigh, calf, or foot).
- Any poorly healing or nonhealing wounds of the legs or feet.
- Any pain at rest localized to the lower leg or foot, and its association with the upright or recumbent positions.
- Postprandial abdominal pain that reproducibly is provoked by eating, and is associated with weight loss.
- Family history of a first degree relative with an abdominal aortic aneurysm (AAA).

Figure 1. Steps Toward the Diagnosis of PAD



**"Atypical" leg pain is defined by lower extremity discomfort that is exertional, but that does not consistently resolve with rest, consistently limit exercise at a reproducible distance, or meet all "Rose questionnaire" criteria.

†The five "Ps" are defined by the clinical symptoms and signs that suggest potential limb jeopardy: pain, pulselessness, pallor, paresthasias, and paralysis (with polar being a sixth "P").

PAD indicates peripheral arterial disease.

Key Components of the Vascular Physical Examination

- Measurement of blood pressure in both arms and notation of any inter-arm asymmetry.
- Palpation of the carotid pulses, and notation of the carotid upstroke and amplitude, and presence of bruits.
- Auscultation of the abdomen and flank for bruits.
- Palpation of the abdomen and notation of the presence of the aortic pulsation and its maximal diameter.
- Palpation of pulses at the brachial, radial, ulnar, femoral, popliteal, dorsalis pedis, and posterior tibial sites. Perform Allen's test when knowledge of hand perfusion is needed.
- Auscultation of both femoral arteries for the presence of bruits
- Pulse intensity should be assessed and should be recorded numerically as follows:
 - 0, absent
 - 1, diminished
 - 2, normal
 - 3, bounding
- The shoes and socks should be removed, the feet inspected, the color, temperature, and integrity of the skin and intertriginous areas evaluated, and presence of ulcerations recorded.
- Additional findings suggestive of severe PAD, including distal hair loss, trophic skin changes, and hypertrophic nails, should be sought and recorded.

3. Evaluation and Treatment of Patients With, or at Risk for, PAD

The noninvasive vascular laboratory provides a powerful set of tools that can objectively assess the status of lower extremity arterial disease and facilitate the creation of a therapeutic plan.

Although there are many diagnostic vascular tests available, the clinical presentation of each patient can usually be linked to specific and efficient testing strategies (*Table 3*).

Table 3. Typical Noninvasive Vascular Laboratory Tests for Lower Extremity PAD Patients by Clinical Presentation

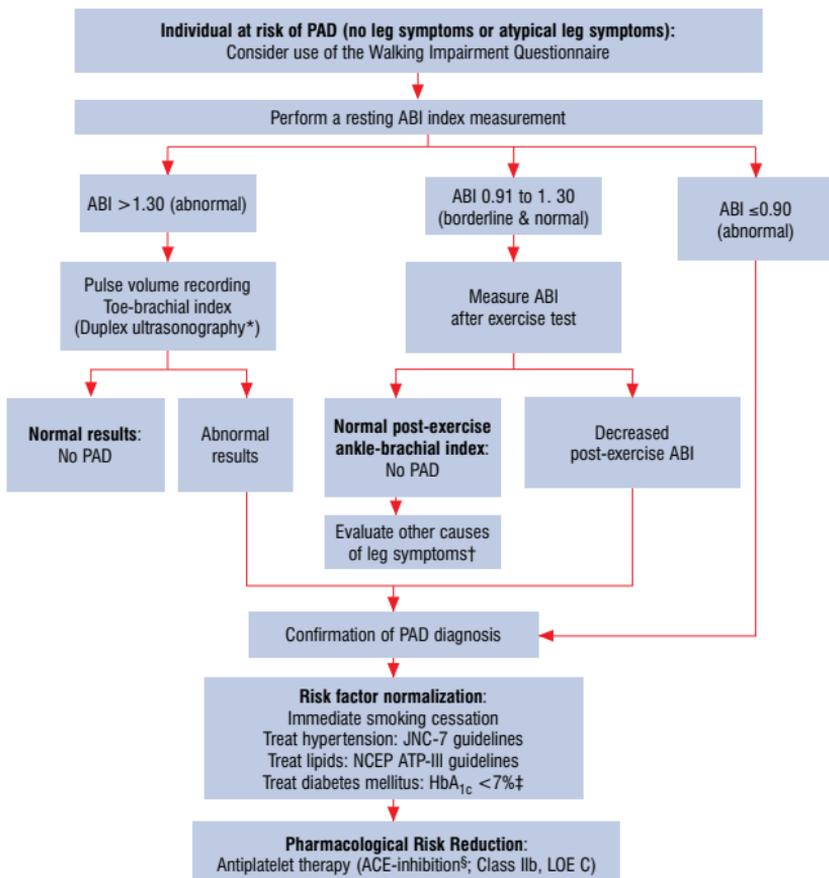
Clinical Presentation	Noninvasive Vascular Test
Asymptomatic lower extremity PAD	ABI
Claudication	ABI, PVR, or segmental pressures Duplex ultrasound Exercise test with ABI to assess functional status
Possible pseudoclaudication	Exercise test with ABI
Postoperative vein graft follow-up	Duplex ultrasound
Femoral pseudoaneurysm; iliac or popliteal aneurysm	Duplex ultrasound
Suspected aortic aneurysm; serial AAA follow-up	Abdominal ultrasound, CTA, or MRA
Candidate for revascularization	Duplex ultrasound, MRA, or CTA

AAA indicates abdominal aortic aneurysm; ABI, ankle-brachial index; CTA, computed tomography angiography; MRA, magnetic resonance angiography; PAD, peripheral artery disease and PVR, pulmonary vascular resistance.

Recommendations for Evaluation and Treatment of Individuals at Risk for PAD or With Asymptomatic PAD

- 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 systems for adults 50 years and older who have atherosclerosis risk factors, or for adults 70 years and older. (*Level of Evidence: C*)

Figure 2. Diagnosis and Treatment of Asymptomatic PAD and Atypical Leg Pain



*Duplex ultrasonography should generally be reserved for use in symptomatic patients in whom anatomic diagnostic data is required for care. †Other causes of leg pain may include: lumbar disk disease, sciatica, radiculopathy; muscle strain; neuropathy; compartment syndrome. ‡It is not yet proven that treatment of diabetes mellitus will significantly reduce PAD-specific (limb ischemic) endpoints. Primary treatment of diabetes mellitus should be continued according to established guidelines. §The benefit of ACE inhibition in individuals without claudication has not been specifically documented in prospective clinical trials, but has been extrapolated from other "at risk" populations. ACE indicates angiotensin-converting enzyme; ABI, ankle-brachial index; HgbA_{1c}, hemoglobin A_{1c}; JNC-7, Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; LOE, level of evidence; NCEP ATP III, National Cholesterol Education Program Adult Treatment Panel III; PAD, peripheral arterial disease. Adapted from Hiatt WR. Medical treatment of peripheral arterial disease and claudication. *N Engl J Med* 2001;344:1608–1621. Copyright © 2001 Massachusetts Medical Society. All rights reserved.

2. Individuals with asymptomatic lower extremity PAD should be identified by examination and/or measurement of the ankle-brachial index (ABI, see *Figure 2*) in order to offer therapeutic interventions known to diminish their increased risk of MI, stroke, and death. (*Level of Evidence: B*)

3. Smoking cessation, lipid lowering, diabetes and hypertension treatment according to current national treatment guidelines is 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*)

4. Lower Extremity Arterial Disease

A. Claudication

Claudication is defined as fatigue, discomfort, or pain that occurs in specific limb muscle groups during effort due to exercise-induced ischemia (*Figures 3 and 4*).

General Management of Patients With 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 post-exercise if the resting index is normal. (*Level of Evidence: B*)

3. Before undergoing an evaluation for revascularization, 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, orthopedic limitations). (*Level of Evidence: C*)

4. 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*)

5. 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*)

-
- 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*)

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

(Level of Evidence: B)

4. The effectiveness of propionyl-L-carnitine or ginkgo biloba as therapy to improve walking distance in patients with intermittent claudication is not well established. *(Level of Evidence: B)*

Class III

1. Oral vasodilator prostaglandins such as beraprost and iloprost are not effective medications to 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)

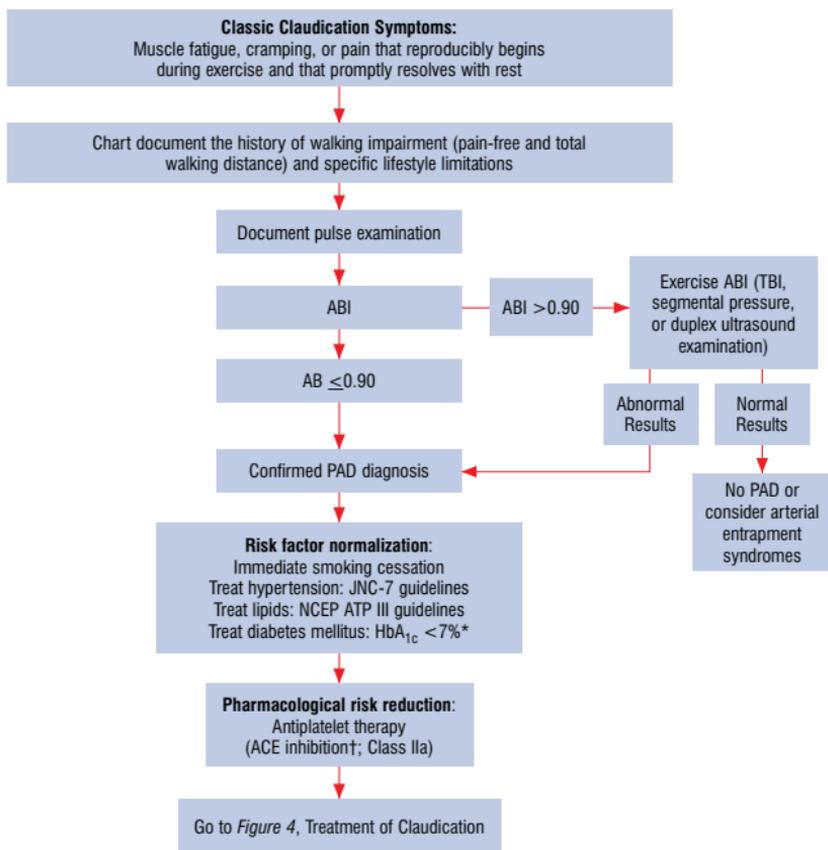
3. Chelation (e.g., Ethylenediaminetetraacetic acid) is not indicated for treatment of intermittent claudication and may have harmful adverse effects. *(Level of*

Evidence: A)

The key elements of a therapeutic claudication exercise program for patients with claudication are summarized in *Table 4, page 19*. For diagnosis and treatment of critical and acute limb ischemia, see *Figures 5, 6 and 7*.



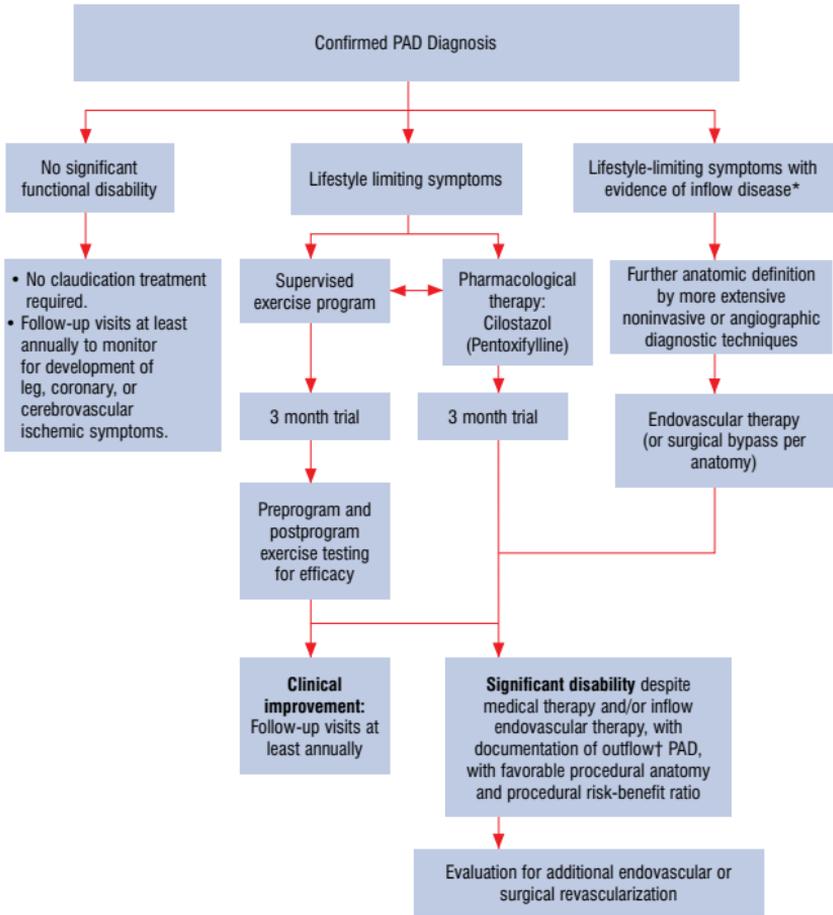
Figure 3. Diagnosis of Claudication and Systemic Risk Treatment



*It is not yet proven that treatment of diabetes mellitus will significantly reduce PAD-specific (limb ischemic) end points. Primary treatment of diabetes mellitus should be continued according to established guidelines. †The benefit of ACE inhibition in individuals without claudication has not been specifically documented in prospective clinical trials but has been extrapolated from other at-risk populations.

ABI indicates ankle-brachial index; ACE, angiotensin-converting enzyme; HgbA_{1c}, hemoglobin A_{1c}; JNC-7, Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; LOE, level of evidence; NCEP ATP III, National Cholesterol Education Program Adult Treatment Panel III; PAD, peripheral arterial disease; TBI, toe-brachial index.

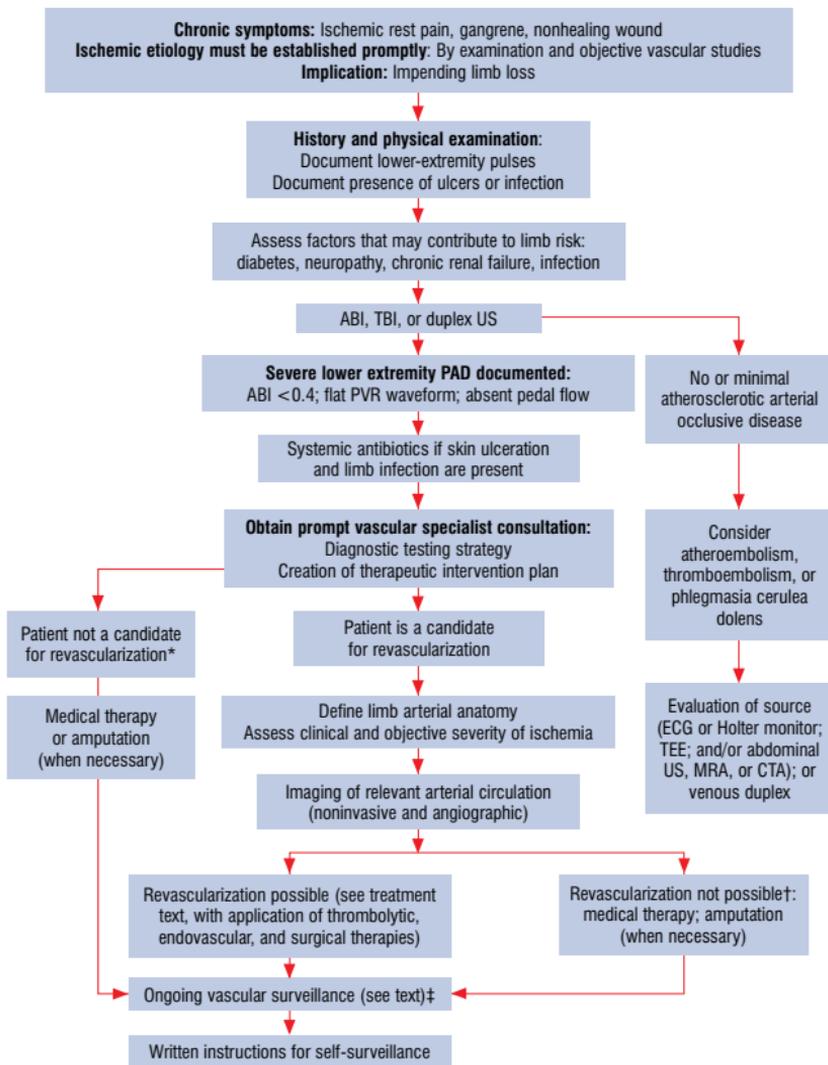
Figure 4. Treatment of Claudication



*Inflow disease should be suspected in individuals with gluteal or thigh claudication and femoral pulse diminution or bruit, and should be confirmed by noninvasive vascular laboratory diagnostic evidence of aorto-iliac stenoses.

†Outflow disease represents femoropopliteal and infrapopliteal stenoses, (the presence of occlusive lesions in the lower extremity arterial tree below the inguinal ligament from the common femoral artery to the pedal vessels).

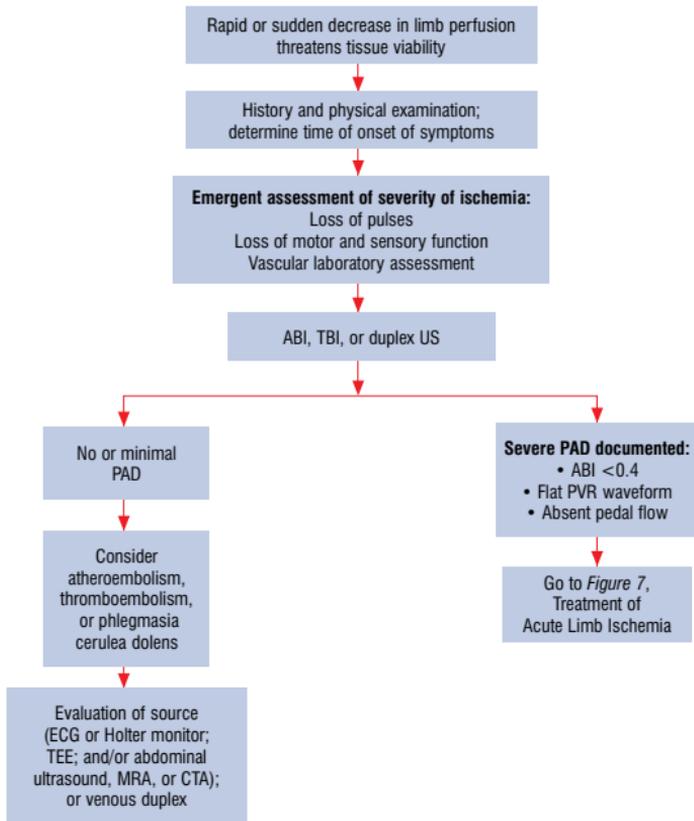
Figure 5. Diagnosis and Treatment of Critical Limb Ischemia



*Based on patient comorbidities. †Based on anatomy or lack of conduit. ‡Risk factor normalization: immediate smoking cessation, treat hypertension per the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure guidelines; treat lipids per National Cholesterol Education Program Adult Treatment Panel III guidelines; treat diabetes mellitus (HgbA_{1c} [hemoglobin A_{1c}] <7%; Class IIa). It is not yet proven that treatment of diabetes mellitus will significantly reduce PAD-specific (limb ischemic) endpoints. Primary treatment of diabetes mellitus should be continued according to established guidelines.

ABI indicates ankle-brachial index; CTA, computed tomographic angiography; ECG, electrocardiogram; MRA, magnetic resonance angiography; PAD, peripheral arterial disease; PVR, pulse volume recording; TBI, toe-brachial index; TEE, transesophageal echocardiography; US, ultrasonography.

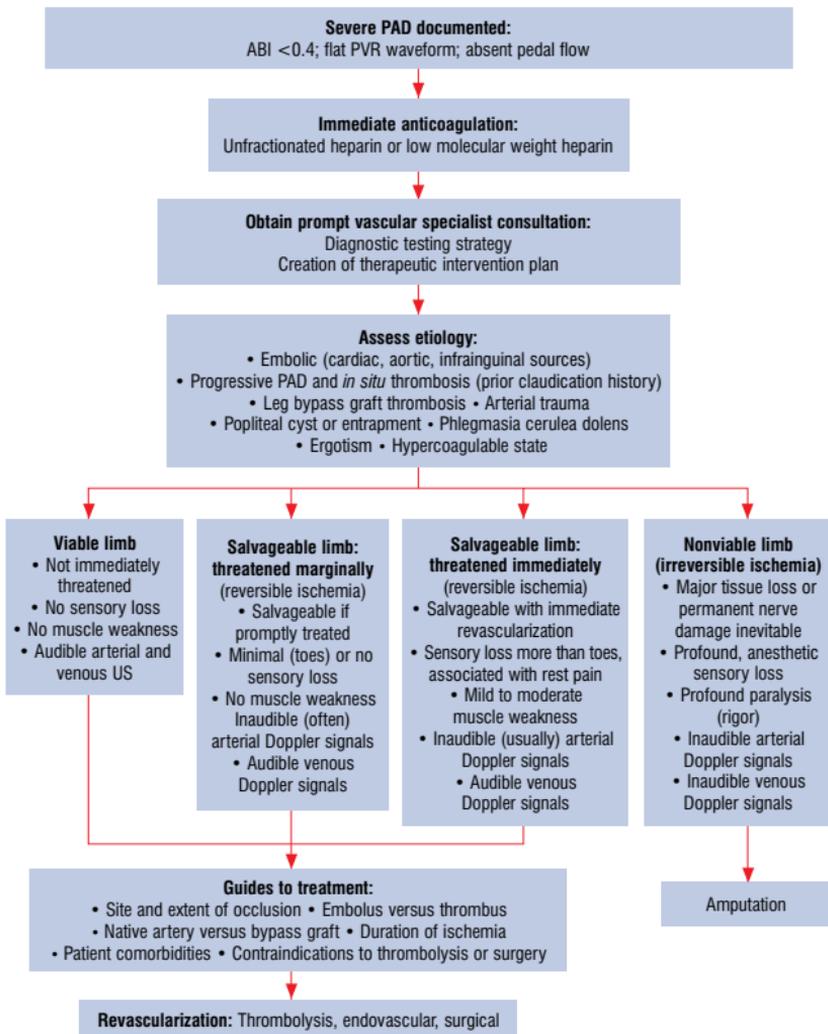
Figure 6. Diagnosis of Acute Limb Ischemia



ABI indicates ankle-brachial index; CTA, computed tomographic angiography; ECG, electrocardiogram; MRA, magnetic resonance angiography; PAD, peripheral arterial disease; PVR, pulse volume recording; TBI, toe-brachial index; TEE, transesophageal echocardiography; US, ultrasonography.

Adapted from *J Vasc Surg*, 26, Rutherford RB, Baker JD, Ernst C, et al., Recommended standards for reports dealing with lower extremity ischemia: revised version, 517–38, Copyright 1997, with permission from Elsevier.

Figure 7. Treatment of Claudication



*Inflow disease should be suspected in individuals with gluteal or thigh claudication and femoral pulse diminution or bruit and should be confirmed by noninvasive vascular laboratory diagnostic evidence of aortoiliac stenoses. †Outflow disease represents femoropopliteal and infrapopliteal stenoses (the presence of occlusive lesions in the lower extremity arterial tree below the inguinal ligament from the common femoral artery to the pedal vessels).

ABI indicates ankle-brachial index; PAD, peripheral arterial disease; PVR, pulse volume recording; US, ultrasonography.

Adapted from *J Vasc Surg*, 26, Rutherford RB, Baker JD, Ernst C, et al., Recommended standards for reports dealing with lower extremity ischemia: revised version, 517–38, Copyright 1997, with permission from Elsevier.

Table 4. Key Elements of a Therapeutic Claudication Exercise Training Program (Lower Extremity PAD Rehabilitation)

PRIMARY CLINICIAN ROLE

- Establish the PAD diagnosis using the ABI measurement or other objective vascular laboratory evaluations
- Determine that claudication is the major symptom limiting exercise
- Discuss risk/benefit of claudication therapeutic alternatives, including pharmacological, percutaneous, and surgical interventions
- Initiate systemic atherosclerosis risk modification
- Perform treadmill stress testing
- Provide formal referral to a claudication exercise rehabilitation program

EXERCISE GUIDELINES FOR CLAUDICATION

- *Warm-up and cool-down period of 5 to 10 minutes each*

Types of Exercise

- Treadmill and track walking are the most effective exercise for claudication
- Resistance training has conferred benefit to individuals with other forms of cardiovascular disease, and its use, as tolerated, for general fitness is complementary to but not a substitute for walking

Intensity

- The initial workload of the treadmill is set to a speed and grade that elicit claudication symptoms within 3 to 5 minutes
- Patients walk at this workload until they achieve claudication of moderate severity, which is then followed by a brief period of standing or sitting rest to permit symptoms to resolve

Duration

- The exercise-rest-exercise pattern should be repeated throughout the exercise session
- The initial duration will usually include 35 minutes of intermittent walking and should be increased by 5 minutes each session until 50 minutes of intermittent walking can be accomplished

Frequency

- Treadmill or track walking 3 to 5 times per week

ROLE OF DIRECT SUPERVISION

- As patients improve their walking ability, the exercise workload should be increased by modifying the treadmill grade or speed (or both) to ensure that there is always the stimulus of claudication pain during the workout
- As patients increase their walking ability, there is the possibility that cardiac signs and symptoms may appear (e.g., dysrhythmia, angina, or ST-segment depression). These events should prompt physician re-evaluation

*These general guidelines should be individualized and based on the results of treadmill stress testing and the clinical status of the patient. A full discussion of the exercise precautions for persons with concomitant diseases can be found elsewhere for diabetes.

ABI indicates ankle-brachial index; PAD, peripheral arterial disease.

Adapted with permission from Stewart KJ, Hiatt WR, Regensteiner JG, Hirsch AT. Medical progress: exercise training for claudication. *N Engl J Med* 2002;347:1941–51. Copyright © 2002 Massachusetts Medical Society. All Rights Reserved.

Endovascular Treatment of Claudication

Class I

1. Endovascular procedures are indicated for individuals with a vocational or lifestyle disability due to intermittent claudication 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 benefit/risk ratio (e.g., focal aorto-iliac occlusive disease). (*Level of Evidence: A*)
2. Endovascular intervention is recommended as the preferred revascularization technique for TransAtlantic Inter-Society Consensus type A (see *Tables 5 and 6* and *Figure 8*) 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 prior to intervention. (*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*)

Table 5. Morphological Stratification of Iliac Lesions

TASC type A iliac lesions:	1. Single stenosis <3 cm of the CIA or EIA (unilateral/bilateral)
TASC type B iliac lesions:	2. Single stenosis 3 to 10 cm in length, not extending into the CFA 3. Total of 2 stenoses <5 cm long in the CIA and/or EIA and not extending into the CFA 4. Unilateral CIA occlusion
TASC type C iliac lesions:	5. Bilateral 5- to 10-cm-long stenosis of the CIA and/or EIA, not extending into the CFA 6. Unilateral EIA occlusion not extending into the CFA 7. Unilateral EIA stenosis extending into the CFA 8. Bilateral CIA occlusion
TASC type D iliac lesions:	9. Diffuse, multiple unilateral stenoses involving the CIA, EIA, and CFA (usually > 10 cm long) 10. Unilateral occlusion involving both the CIA and EIA 11. Bilateral EIA occlusions 12. Diffuse disease involving the aorta and both iliac arteries 13. Iliac stenoses in a patient with an abdominal aortic aneurysm or other lesion requiring aortic or iliac surgery

Endovascular procedure is the treatment of choice for type A lesions, and surgery is the procedure of choice for type D lesions. CFA indicates common femoral artery; CIA, common iliac artery; EIA, external iliac artery; TASC, TransAtlantic Inter-Society Consensus.

Adapted from *J Vasc Surg*, 31, Dormandy JA, Rutherford RB, for the TransAtlantic Inter-Society Consensus (TASC) Working Group, Management of peripheral arterial disease (PAD), S1–S296, Copyright 2000, with permission from Elsevier.

Table 6. Morphological Stratification of Femoropopliteal Lesions

TASC type A femoropopliteal lesions: 1. Single stenosis <3 cm of the superficial femoral artery or popliteal artery

TASC type B femoropopliteal lesions: 2. Single stenosis 3 to 10 cm in length, not involving the distal popliteal artery
3. Heavily calcified stenoses up to 3 cm in length
4. Multiple lesions, each <3 cm (stenoses or occlusions)
5. Single or multiple lesions in the absence of continuous tibial runoff to improve inflow for distal surgical bypass

TASC type C femoropopliteal lesions: 6. Single stenosis or occlusion longer than 5 cm
7. Multiple stenoses or occlusions, each 3 to 5 cm in length, with or without heavy calcification

TASC type D femoropopliteal lesions: 8. Complete common femoral artery or superficial femoral artery occlusions or complete popliteal and proximal trifurcation occlusions

Endovascular procedure is the treatment of choice for type A lesions, and surgery is the procedure of choice for type D lesions. More evidence is needed to make firm recommendations about the best treatment for type B and C lesions.

TASC indicates TransAtlantic Inter-Society Consensus.

Adapted from *J Vasc Surg*, 31, Dormandy JA, Rutherford RB, for the TransAtlantic Inter-Society Consensus (TASC) Working Group, Management of peripheral arterial disease (PAD), S1–S296, Copyright 2000, with permission from Elsevier.

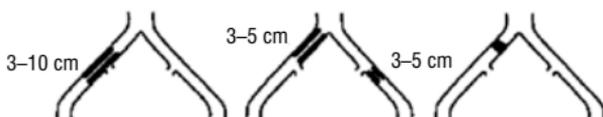


Figure 8. Summary of Preferred Options in Interventional Management of Iliac Lesions

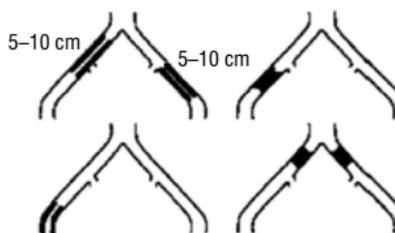
Type A Endovascular treatment of choice



Type B Currently, endovascular treatment is more often used but insufficient evidence for recommendation



Type C Currently, surgical treatment is more often used but insufficient evidence for recommendation



Type D Surgical treatment of choice



Reprinted from *J Vasc Surg*, 31, Dormandy JA, Rutherford RB, for the TransAtlantic Inter-Society Consensus (TASC) Working Group, Management of peripheral arterial disease (PAD), S1–S296, Copyright 2000, with permission from Elsevier.

Surgical Treatment of Claudication

- 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*)
 2. 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*)
-

- 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*)

B. Critical Limb Ischemia (UPDATED)

CLI is defined as limb pain occurring at rest or impending limb loss that is caused by severe compromise of blood flow to the

affected extremity. This includes patients with chronic ischemia rest pain, ulcers, or gangrene attributable to objectively proven arterial occlusive disease. See *Figure 5* for the diagnosis and treatment pathway for CLI.

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

-
- Class IIa**
1. For patients with limb-threatening lower extremity ischemia and an estimated life expectancy of 2 years or less or 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. 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)*

Thrombolysis for Acute and Chronic Limb Ischemia

Class I 1. Catheter-based thrombolysis is an effective and beneficial therapy and is indicated for patients with acute limb ischemia 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 artery occlusion. *(Level of Evidence: B)*

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

Surgery for CLI

Class I 1. For individuals with combined inflow and outflow disease with critical 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. *(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)*

C. Acute Limb Ischemia

Acute limb ischemia is defined as a rapid or sudden decrease in limb perfusion that threatens limb viability (see Figure 6). The five “Ps” suggest limb jeopardy: pain, paralysis, paresthesias, pulselessness, and pallor (with polar being a sixth “P”). See Figure 7 for the acute limb ischemia treatment pathway.

Management of Patients With Acute Limb Ischemia

Class I 1. Patients with acute limb ischemia and a salvageable extremity should undergo an emergency 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*)

D. Surveillance for Patients After Lower Extremity Revascularization

Patients who have undergone revascularization procedures require long-term care and vascular follow-up to detect recurrence of disease at revascularized sites, as well as development of new disease at remote sites.

-
- Class I**
1. Long-term patency of infrainguinal bypass grafts should be evaluated in a surveillance program (*Table 7*), which should include an interval vascular history, resting ABIs, physical examination, and a duplex ultrasound at regular intervals if venous conduit has been used. (*Level of Evidence: B*)
 2. Duplex ultrasound is recommended for routine surveillance following femoral-popliteal or femoral-tibial-pedal bypass using venous conduit. Minimum surveillance intervals are approximately 3 months, 6 months, 12 months, and then yearly following graft placement. (*Level of Evidence: A*)



Class IIa

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

Table 7. Surveillance Program for Infrainguinal Vein Bypass Grafts

Patients undergoing vein bypass graft placement in the lower extremity for the treatment of claudication or limb-threatening ischemia should be entered into a surveillance program. This program should consist of:

- Interval history (new symptoms)
- Vascular examination of the leg with palpation of proximal, graft, and outflow vessel pulses
- Periodic measurement of resting and, if possible, postexercise ABIs
- Duplex scanning of the entire length of the graft, with calculation of peak systolic velocities and velocity ratios across all identified lesions

Surveillance programs should be performed in the immediate postoperative period and at regular intervals for at least 2 years

- Femoral-popliteal and femoral-tibial venous conduit bypass at approximately 3, 6, and 12 months and annually

Modified from Dormandy JA, Rutherford RB. Management of peripheral artery disease (PAD). TASC Working Group. TransAtlantic Inter-Society Consensus (TASC). *J Vasc Surg.* 2000 Jan;31(1 pt 2):S1-S296.



E. Ankle-Brachial Index, Toe-Brachial Index, and Segmental Pressure Examination (UPDATED)

- Class I**
1. 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 years 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. 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*)

F. Smoking Cessation (UPDATED)

Class I

1. Patients who are smokers or former smokers should be asked about status of tobacco use at every visit. *(Level of Evidence: B)*
2. 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. 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. For all patients in the absence of contraindication, 1 or more of the following pharmacological therapies should be offered: varenicline, bupropion, and nicotine replacement therapy*. *(Level of Evidence: A)*

*<http://www.fda.gov/Safety/MedWatch/SafetyInformation/SafetyAlertsforHumanMedicalProducts/ucm259469.htm>



G. Antithrombotic and Antiplatelet Therapy (UPDATED)

Class I

1. 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. 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. 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. 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. 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. 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 at high perceived cardiovascular risk. *(Level of Evidence: B)*

**Class III:
No Benefit** 1. 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)*

5. Renal Arterial Disease

Renal artery stenosis (RAS) is both a common and progressive disease in patients with atherosclerosis and a relatively uncommon cause of hypertension.

A. Clinical Indications

Class I

1. The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with:
 - the onset of hypertension before the age of 30 years. (*Level of Evidence: B*)
 - 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*)
 - the following characteristics: (*Level of Evidence: C*)
 - accelerated hypertension (sudden and persistent worsening of previously controlled hypertension);
 - hypertension resistant to treatment (use of at least 2 antihypertensive medications of different classes, including a diuretic)
 - malignant hypertension (with end-organ damage, i.e., acute renal failure, congestive heart failure, visual or neurological disturbance, and/or advanced (grade III to IV) retinopathy).
 - new azotemia or worsening renal function after the administration of an angiotensin converting

enzyme (ACE) inhibitor or an angiotensin receptor blocking agent. (*Level of Evidence: B*)

- an unexplained atrophic kidney or a discrepancy in size between the 2 kidneys of greater than 1.5 cm. (*Level of Evidence: B*)
 - sudden, unexplained pulmonary edema (especially in azotemic patients). (*Level of Evidence: B*)
-

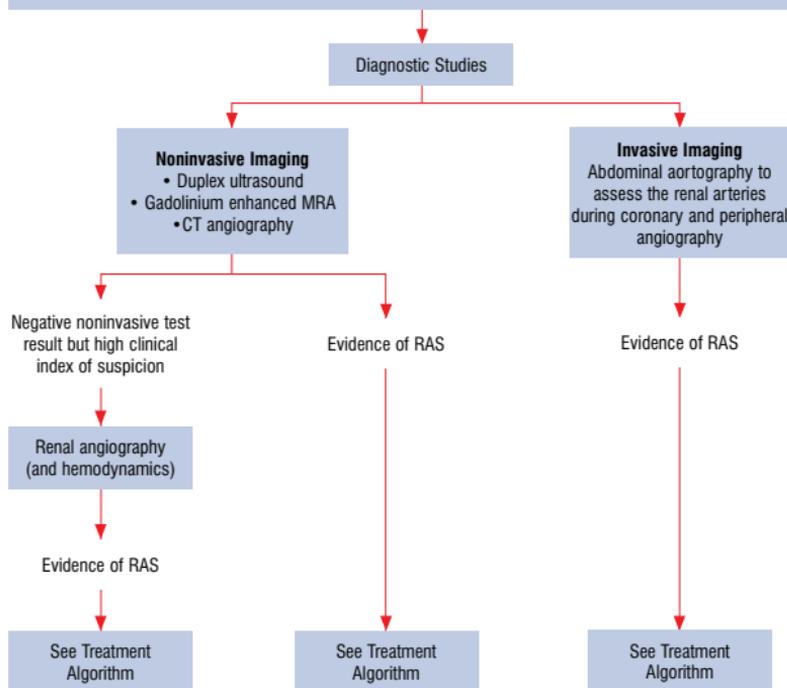
Class IIa 1. The performance of diagnostic studies to identify clinically significant RAS is reasonable in patients with unexplained renal dysfunction, including individuals starting renal replacement therapy (dialysis or renal transplantation). (*Level of Evidence: B*)

Class IIb 1. The performance of arteriography to identify significant RAS may be reasonable in patients with multivessel coronary artery disease and none of the clinical clues (*Figure 9*) (at the time of coronary angiography) 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 (see definition in *Figure 9* footnote).

Figure 9. Clinical Clues to the Diagnosis of Renal Artery Stenosis

1. Onset of hypertension before the age of 30 years or severe hypertension after the age of 55.* (Class I; LOE B)
2. Accelerated, resistant, or malignant hypertension.* (Class I; LOE C)
3. Development of new azotemia or worsening renal function after administration of an ACE inhibitor or ARB agent. (Class I; LOE B)
4. Unexplained atrophic kidney or size discrepancy between kidneys of greater than 1.5 cm.† (Class I; LOE B)
5. Sudden, unexplained pulmonary edema. (Class I; LOE B)
6. Unexplained renal dysfunction, including individuals starting renal replacement therapy. (Class IIa; LOE B)
7. Multi-vessel coronary artery disease. (Class IIb; LOE B)
8. Unexplained congestive heart failure. (Class IIb; LOE C)
9. Refractory angina. (Class IIb; LOE C)



*For definition of hypertension, please see Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003; 289:2560–72 † For example, atrophic kidney due to chronic pyelonephritis is not an indication for renal artery stenosis (RAS) evaluation.

ACE indicates angiotensin-converting enzyme; ARB, angiotensin receptor blocking agent; CT, computed tomography; LOE, level of evidence; MRA, magnetic resonance angiography.

B. Diagnostic Methods

- Class I**
1. Duplex ultrasound sonography is recommended as a screening test to establish the diagnosis of RAS.
(Level of Evidence: B)
 2. Computed tomography angiography (in individuals with normal renal function) is recommended as a screening test to establish the diagnosis of RAS.
(Level of Evidence: B)
 3. Magnetic resonance angiography 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. The 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 following captopril administration) is not

recommended as a useful screening test to establish the diagnosis of RAS. (*Level of Evidence: B*)

C. Indications for Revascularization of Patients with Hemodynamically Significant RAS

A treatment algorithm based on the current evidence is provided in *Figure 10*.

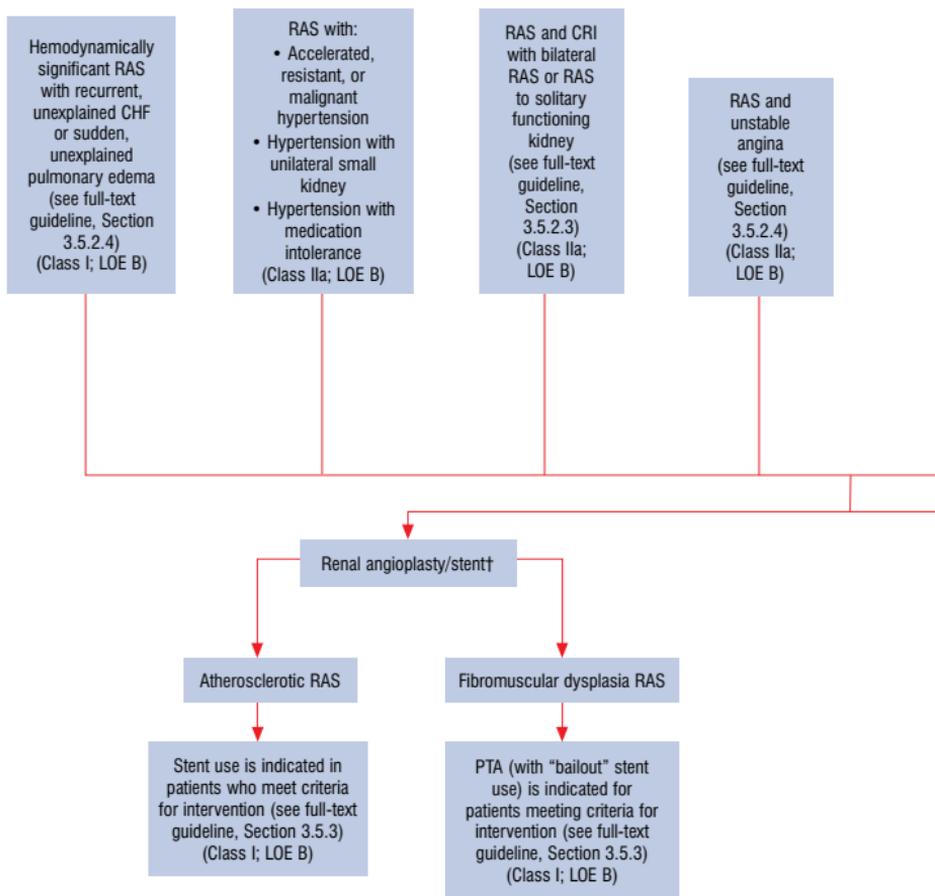
Asymptomatic Stenosis

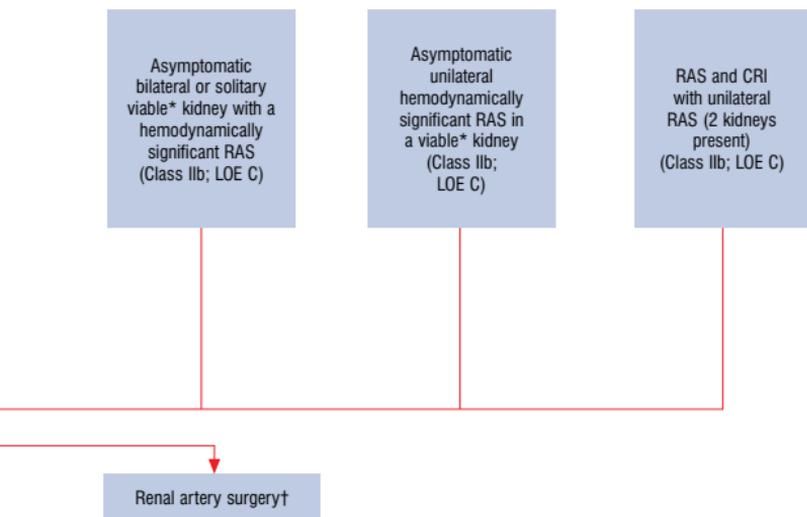
- Class IIb**
1. Percutaneous revascularization may be considered for treatment of an asymptomatic bilateral or a solitary viable kidney with a hemodynamically significant RAS. (*Level of Evidence: C*)
 2. The usefulness of percutaneous revascularization of an asymptomatic unilateral hemodynamically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

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*)

Figure 10. Indications for Renal Revascularization





*Viable means kidney linear length greater than 7 cm. †It is recognized that renal artery surgery has proven efficacy in alleviating RAS due to atherosclerosis and fibromuscular dysplasia. Currently, however, its role is often reserved for individuals in whom less invasive percutaneous RAS interventions are not feasible.

CHF indicates congestive heart failure; CRI, chronic renal insufficiency; LOE, level of evidence, RAS, renal artery stenosis; PTA, percutaneous transluminal angioplasty.

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)*

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 (see text). *(Level of Evidence: B)*

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

D. Treatment Methods: Medical, Endovascular, and Surgical

Pharmacological Treatment of Individuals with RAS

Multiple studies have now shown that the ACE inhibitors and calcium channel blockers are effective in the treatment of hypertension in the presence of RAS. Pharmacological treatment of hypertension to

therapeutic goals, with any class of effective antihypertensive medication, should be considered an essential component of medical care for such individuals with RAS and hypertension.

Class I

1. ACE inhibitors are effective medications for treatment of hypertension associated with RAS.

(Level of Evidence: A)

2. ARBs are effective medications for treatment of hypertension associated with unilateral RAS. *(Level of Evidence: B)*

(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)

Catheter-Based Interventions 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 “bail-out” stent placement if necessary is recommended for fibromuscular dysplasia lesions. *(Level of Evidence: B)*

Surgery for RAS

Class I

1. Vascular surgical reconstruction is indicated for patients with:

- fibromuscular dysplastic renal artery stenosis with clinical indications for interventions (same as percutaneous transluminal angioplasty), especially those exhibiting complex disease extending into the segmental arteries and those having macroaneurysms. *(Level of Evidence: B)*
- 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)*
- atherosclerotic RAS in combination with para-renal aortic reconstructions (in treatment of aortic aneurysms or severe aorto-iliac occlusive disease). *(Level of Evidence: C)*



6. Mesenteric Arterial Disease

Acute intestinal ischemia may occur due to thromboembolism, a hypercoagulable state, arterial dissection, or nonocclusive low flow states. Chronic intestinal ischemia is virtually always due to arterial obstruction.

A. Acute Intestinal Ischemia

Diagnosis of Acute Intestinal Ischemia

- 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 have arrhythmias such as atrial fibrillation, or recent MIs, 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*)

Surgical Treatment of Acute Intestinal Ischemia

- 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 following the revascularization. *(Level of Evidence: B)*

Endovascular Treatment of Acute Intestinal Ischemia

- Class IIb** 1. Percutaneous interventions (including transcatheter lytic therapy, balloon angioplasty and/or 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)*

B. Acute Nonocclusive Intestinal Ischemia

- 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)*
 - receiving vasoconstrictor substances and medications (e.g., cocaine, ergot, vasopressin, norepinephrine, etc.) who develop abdominal pain. *(Level of Evidence: B)*

- who develop abdominal pain after coarctation repair, or after surgical revascularization for intestinal ischemia caused by arterial obstruction.

(Level of Evidence: B)

2. Arteriography is indicated in patients suspected of nonocclusive intestinal ischemia whose condition does not improve rapidly with treatment of their underlying disease. *(Level of Evidence: B)*

3. Treatment of the underlying shock state is the initial most important step in treatment of nonocclusive intestinal ischemia. *(Level of Evidence: C)*

4. 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, or in patients with intestinal ischemia due to cocaine or ergot poisoning. *(Level of Evidence: B)*

C. Chronic Intestinal Ischemia

Diagnosis of Chronic Intestinal Ischemia

- 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, computed tomography angiography, and gadolinium enhanced magnetic resonance angiography 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)*

Treatment of 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)*
 2. 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*)

7. Aneurysms of the Abdominal Aorta, Its Branch Vessels, and the Lower Extremities

Arterial aneurysms share many of the same atherosclerotic risk factors and pose similar threats to life, limb, and vital organ function as occlusive artery disease. The presence of most common aneurysms can be suspected on the basis of an attentive physical examination and subsequently confirmed by noninvasive, widely available imaging studies.

A. Abdominal Aortic Aneurysms

In general, an AAA is considered to be present when the minimum anteroposterior diameter of the aorta reaches 3.0 cm. Risk factors for AAA include advancing age, family history (particularly for first degree relatives), male gender, and tobacco use.

Screening High-Risk Populations for AAAs

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 one time ultrasound screening for detection of AAAs. (*Level of Evidence: B*)

General Patient Management

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*)

3. 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*)

4. In patients with symptomatic aortic aneurysms, repair is indicated regardless of diameter. (*Level of Evidence: C*)

5. 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*)

Treatment of AAAs

For an overview of the treatment and management of AAAs, see *Figure 11*.

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 computer tomography scans every 6 to 12 months to detect expansion. (*Level of Evidence: A*)
3. Open repair of infrarenal AAAs and/or common iliac aneurysms is indicated in patients who are good or average surgical candidates. (*Level of Evidence: B*)

4. For patients who have undergone endovascular repair of infrarenal aortic and/or iliac aneurysms, periodic long-term surveillance imaging should be performed to monitor for an endoleak, document shrinkage or stability of the excluded aneurysm sac, and to determine the need for further intervention. *(Level of Evidence: B)*

- Class IIa**
1. Repair can be beneficial in patients with infrarenal or juxtarenal abdominal aortic aneurysms 5.0 to 5.4 cm in diameter. *(Level of Evidence: B)*
 2. Repair is probably indicated in patients with suprarenal or Type IV thoraco-abdominal 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)*
 4. Endovascular repair of infrarenal aortic and/or common iliac aneurysms is reasonable in patients at high risk of complications from open operations because of cardiopulmonary or other associated diseases. *(Level of Evidence: B)*
-

- Class IIb**
1. Endovascular repair of infrarenal aortic and/or common iliac aneurysms may be considered in patients at low or average surgical risk. *(Level of Evidence: B)*

Class III 1. Intervention is not recommended for asymptomatic infrarenal or juxtarenal abdominal aortic aneurysms 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*)

B. Management Overview of Prevention of Aortic Aneurysm Rupture (UPDATED)

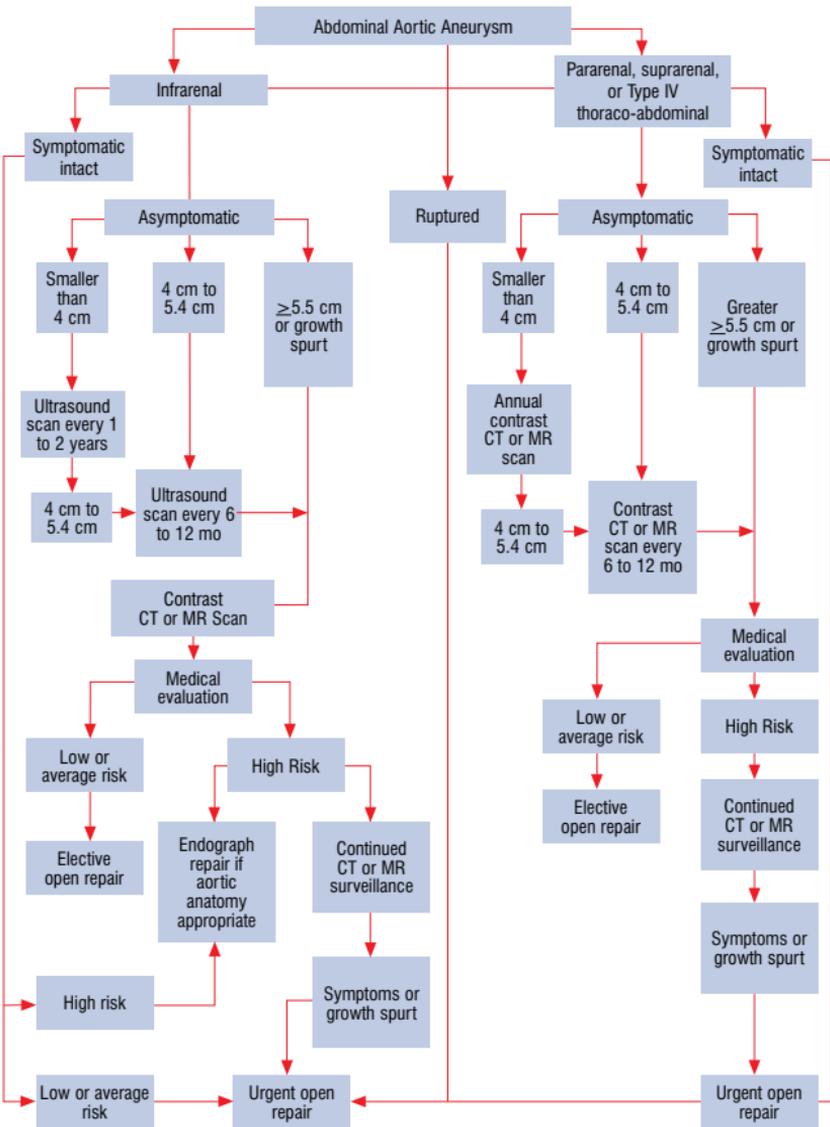
Class I 1. Open or endovascular repair of infrarenal AAAs and/or common iliac aneurysms is indicated in patients who are good surgical candidates. (*Level of Evidence: A*)

2. Periodic long-term surveillance imaging should be performed to monitor for endoleak, confirm graft position, document shrinkage or stability of the excluded aneurysm sac, and 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. 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. Endovascular repair of infrarenal aortic aneurysms in patients who are at high surgical or anesthetic

Figure 11. Management of Abdominal Aortic Aneurysms



CT indicates computed tomography; IV, intravenous; MR, magnetic resonance imaging; mo, month; y, year.

risk as determined by the presence of coexisting severe cardiac, pulmonary, and/or renal disease is of uncertain effectiveness. (*Level of Evidence: B*)

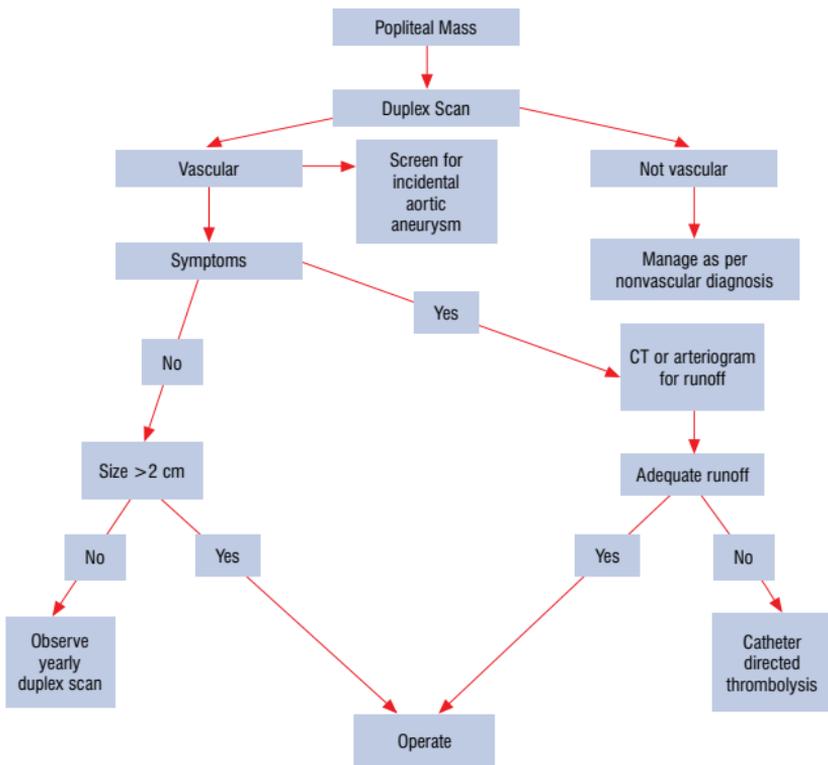
C. Visceral Arterial Aneurysms

Visceral artery aneurysms are insidious because they usually cannot be detected by physical examination and may be overlooked on radiographs or computed tomography/magnetic resonance scanning. Approximately half present with rupture, and the mortality rate is 25% or higher. Risk factors include portal hypertension, prior liver transplantation, and multiparous women.

Class I 1. Open repair or catheter-based intervention is indicated for visceral aneurysms measuring 2 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 cm in diameter or larger in women beyond childbearing age and in men. (*Level of Evidence: B*)

Figure 12. Diagnostic and Treatment Algorithm for Popliteal Mass



CT indicates computed tomography.

D. Lower Extremity Arterial Aneurysms

In general, lower extremity arterial aneurysms are considered to be significant when the minimum diameter reaches 3.0 cm (common femoral) to 2.0 (popliteal). The presence of a lower extremity arterial aneurysm should lead to examination for the presence of an AAA. (*Figure 12*). Unlike AAAs, the natural history of extremity artery aneurysms is not one of expansion and rupture but one of thromboembolism or thrombosis.

- Class I**
1. In patients with femoral or popliteal aneurysms, ultrasound (or computed tomography, magnetic resonance) imaging is recommended to exclude contralateral femoral or popliteal aneurysms and AAA. (*Level of Evidence: B*)
 2. Patients with a palpable popliteal mass should undergo an ultrasound examination to exclude popliteal aneurysm. (*Level of Evidence: B*)
 3. 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*)
 4. 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 and/or mechanical thrombectomy 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)*



E. Femoral Artery Pseudoaneurysms

Femoral artery pseudoaneurysms may occur after blunt trauma, access for catheter-based procedures, injury resulting from puncture for drug abuse, or disruption of a previous suture line (see Figure 13).

Catheter-Related Femoral Artery Pseudoaneurysms

Class I 1. Patients with suspected femoral pseudoaneurysms should be evaluated by duplex ultrasonography.

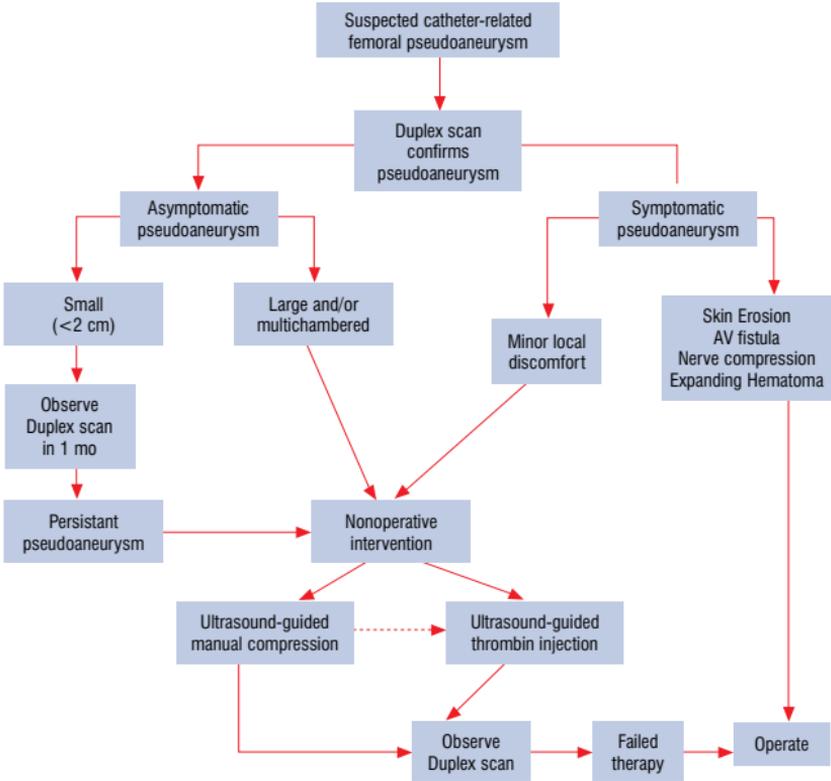
(Level of Evidence: B)

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)*

Figure 13. Diagnostic and Treatment Algorithm for Femoral Pseudoaneurysm



AV indicates arteriovenous; mo, month.

The ACCF/AHA would like to acknowledge and thank our volunteer writing committee members for their time and contributions in support of the missions of our organizations.

2011 PAD WRITING GROUP MEMBERS

Joshua A. Beckman, MD, FACC, FAHA
Laura K. Findeiss, MD
Jafar Golzarian, MD
Heather L. Gornik, MD, FACC, FAHA
Jonathan L. Halperin, MD, FACC, FAHA
Alan T. Hirsch, MD, FACC, *Vice Chair*
Michael R. Jaff, DO, FACC
Sanjay Misra, MD, *Vice Chair*
Gregory L. Moneta, MD, FACS
Jeffrey W. Olin, DO, FACC, FAHA
Thom W. Rooke, MD, FACC, *Chair*
Anton N. Sidawy, MD, MPH, FACS, *Vice Chair*
James C. Stanley, MD, FACS
Christopher J. White, MD, FACC, FAHA, FSCAI
John V. White, MD, FACS
R. Eugene Zierler, MD, FACS

2005 PAD WRITING COMMITTEE MEMBERS

Curtis W. Bakal, MD, MPH, FAHA
Mark A. Creager, MD, FACC, FAHA
Jonathan L. Halperin, MD, FACC, FAHA
Ziv J. Haskal, MD, FAHA, FSIR, *Vice Chair*
Norman R. Hertzner, MD, FACS, *Vice Chair*
Loren F. Hiratzka, MD, FACC, FAHA, FACS
Alan T. Hirsch, MD, FACC, *Chair*
William R.C. Murphy, MD, FACC, FACS
Jeffrey W. Olin, DO, FACC
Kenneth A. Rosenfield, MD, FACC
David Sacks, MD, FSIR
James C. Stanley, MD, FACS
Lloyd M. Taylor Jr, MD, FACS
Christopher J. White, MD, FACC, FAHA, FSCAI
John V. White, MD, FACS
Rodney A. White, MD, FACS



