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

March 2006

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I. Introduction

“Peripheral arterial disease” (PAD) is the preferred clinical term that should be used to denote stenotic, occlusive, and aneurysmal diseases of the aorta and its branch arteries, exclusive of the coronary arteries. These practice guidelines are intended to assist physicians in clinical decision making by describing a range of generally acceptable approaches for the diagnosis and management of patients with PAD. The scope of these guidelines is limited to include disorders of the abdominal aorta, the renal and mesenteric arteries, and the lower extremity arteries. However, it is important to recognize that patients with PAD are likely to have coexistent cardiac and cerebrovascular disease such that there is an elevated risk of myocardial infarction and stroke ischemic events and an associated increased mortality from coronary heart disease and stroke. Thus, attention to the entire cardiovascular system and achievement of risk factor reduction goals is exceedingly important.

These guidelines attempt to define practices that meet the needs of most patients in most circumstances. The ultimate judgment regarding the care
of an individual patient must be made by the physician and patient in light of all circumstances presented by that patient.

This pocket guide provides a brief synopsis of information provided in the full-text guidelines. It does not contain all of the recommendations found in the executive summary or full-text guidelines. The content herein is tailored toward the primary care clinician (family physician, internist, nurse practitioner, and physician’s assistant), cardiovascular physicians and vascular specialists, as well as trainees. For additional, more technical detail, the user should refer to the full-text guidelines for extensive information, rationale, recommendations and caveats, which are carefully presented.

Classifications of recommendations and levels of evidence are expressed in the ACC/AHA format as follows.
Classification of Recommendations

Class I

Conditions for which there is evidence for and/or general agreement that a given procedure or treatment is beneficial, useful, and effective.

Class II

Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa

Weight of evidence/opinion is in favor of usefulness/efficacy.

Class IIb

Usefulness/efficacy is less well established by evidence/opinion.

Class III

Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

Level of Evidence

Level of Evidence A

Data derived from multiple randomized clinical trials or meta-analyses.

Level of Evidence B

Data derived from a single randomized trial or nonrandomized studies.

Level of Evidence C

Only consensus opinion of experts, case studies, or standard-of-care.

Table 1 delineates the classification of recommendations and level of evidence.
### Table 1. Applying Classification of Recommendations and Level of Evidence in ACC/AHA Format

<table>
<thead>
<tr>
<th>ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT</th>
<th>SIZE OF TREATMENT EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>LEVEL A  &lt;br&gt;Multiple (3-5) population risk strata evaluated* &lt;br&gt;General consistency of direction and magnitude of effect</td>
<td>CLASS I  &lt;br&gt;Benefit &gt;&gt;&gt; Risk  &lt;br&gt;Procedure/Treatment SHOULD be performed/administered  &lt;br&gt;  - Recommendation that procedure or treatment is useful/effective  &lt;br&gt;  - Sufficient evidence from multiple randomized trials or meta-analyses</td>
</tr>
<tr>
<td>LEVEL B  &lt;br&gt;Limited (2-3) population risk strata evaluated*</td>
<td>Recommendation that procedure or treatment is useful/effective  &lt;br&gt;  - Limited evidence from single randomized trial or nonrandomized studies</td>
</tr>
<tr>
<td>LEVEL C  &lt;br&gt;Very limited (1-2) population risk strata evaluated*</td>
<td>Recommendation that procedure or treatment is useful/effective  &lt;br&gt;  - Only expert opinion, case studies, or standard-of-care</td>
</tr>
</tbody>
</table>

Suggested phrases for writing recommendations should  <br>is recommended  <br>is indicated  <br>is useful/effective/beneficial  <br>is reasonable  <br>can be useful/effective/beneficial  <br>is probably recommended or indicated  

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as gender, age, history of diabetes, history of prior MI, history of heart failure, and prior aspirin use.
<table>
<thead>
<tr>
<th>CLASS IIb</th>
<th>CLASS III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benefit ≥ Risk</td>
<td>Risk ≥ Benefit</td>
</tr>
<tr>
<td>Additional studies with broad objectives needed; additional registry data would be helpful</td>
<td>No additional studies needed</td>
</tr>
<tr>
<td>Procedure/Treatment MAY BE CONSIDERED</td>
<td>Procedure/Treatment should NOT be performed/administered SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL</td>
</tr>
</tbody>
</table>

- Recommendation’s usefulness/efficacy less well established
- Greater conflicting evidence from multiple randomized trials or meta-analyses
- Recommendation’s usefulness/efficacy less well established
- Greater conflicting evidence from single randomized trial or nonrandomized studies
- Recommendation’s usefulness/efficacy less well established
- Only diverging expert opinion, case studies, or standard-of-care
- Recommendation that procedure or treatment is not useful/effective and may be harmful
- Sufficient evidence from multiple randomized trials or meta-analyses
- Recommendation that procedure or treatment is not useful/effective and may be harmful
- Limited evidence from single randomized trial or nonrandomized studies
- Recommendation that procedure or treatment is not useful/effective and may be harmful
- Only expert opinion, case studies, or standard-of-care

may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established is not recommended is not indicated should not is not useful/effective/beneficial may be harmful
II. 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 (see 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 years and history of smoking or diabetes
- Age 70 years and 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
**Figure 1. Steps Toward the Diagnosis of PAD**

**Individuals at Risk for Lower Extremity PAD:**
- Age less than 50 years with diabetes and one other atherosclerosis risk factor (smoking, dyslipidemia, hypertension, or hyperhomocysteinemia)
- Age 50 to 69 years and history of smoking or diabetes
- Age 70 years and older
- Leg symptoms with exertion (suggestive of claudication) or ischemic rest pain
- Abnormal lower extremity pulse examination
- Known atherosclerotic coronary, carotid, or renal arterial disease

**Obtain history of walking impairment and/or limb ischemic symptoms:**
- Leg discomfort with exertion
- Leg pain at rest; nonhealing wound; gangrene

**Perform a resting ankle-brachial index measurement**

- **No leg pain**
- **“Atypical” leg pain***
- **Classic claudication symptoms:** Exertional fatigue, discomfort, or frank pain localized to leg muscle groups that consistently resolves with rest
- **Ischemic leg pain at rest**
- **Nonhealing wound**
- **Gangrene**

**Sudden onset ischemic leg symptoms or signs of acute limb ischemia: The five “Ps”†**

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*“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, paresthesias, and paralysis (with polar being a sixth “P”).

PAD = peripheral arterial 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 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).
Key Components of the Vascular Physical Examination

- Measurement of blood pressure in both arms and notation of any interarm asymmetry.

- Palpation of carotid pulses, recording of carotid upstroke and amplitude and presence of bruits.

- Auscultation of abdomen and flank for bruits.

- Palpation of the abdomen and recording of the presence of the aortic pulsation and 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 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 the 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.
III. 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 (see Table 3).

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

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

AAA = abdominal aortic aneurysm; ABI = ankle-brachial index; CTA = computed tomographic angiography; MRA = magnetic resonance angiography; PAD = peripheral arterial disease; PVR = pulse volume recording.

Class I 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 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, see Figure 2) so that therapeutic interventions known to diminish their increased risk of myocardial infarction, stroke, and death may be offered. *(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)*
Figure 2. Diagnosis and Treatment of Asymptomatic PAD and Atypical Leg Pain

Individual at risk of PAD (no leg symptoms or atypical leg symptoms):
Consider use of the Walking Impairment Questionnaire

Perform a resting ankle-brachial index measurement

ABI greater than 1.30 (abnormal)

Pulse volume recording Toe-brachial index (Duplex ultrasonography*)

Normal results: No peripheral arterial disease
Abnormal results

ABI 0.91 to 1.30 (borderline & normal)

Measure ankle-brachial index after exercise test

Normal post-exercise ankle-brachial index; No peripheral arterial disease

Decreased post-exercise ankle-brachial index

Evaluate other causes of leg symptoms†

Confirmation of PAD diagnosis

Risk factor normalization:
- Immediate smoking cessation
- Treat hypertension: JNC-7 guidelines
- Treat lipids: NCEP ATP III guidelines
- Treat diabetes mellitus: HbA1c less than 7%‡

Pharmacological Risk Reduction:
- Antiplatelet therapy (ACE inhibition,§
Class Iib, LOE C)

*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 peripheral arterial disease (PAD)-specific (limb ischemic) end points. Primary treatment of diabetes mellitus should be continued according to established guidelines.
§The benefit of angiotensin-converting enzyme (ACE) inhibition in individuals without claudication has not been specifically documented in prospective clinical trials, but has been extrapolated from other “at risk” populations.

\[\text{ABI} = \text{ankle-brachial index; } \]
\[\text{HgbA1c} = \text{hemoglobin A1c; } \]
\[\text{JNC-7} = \text{Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; } \]
\[\text{LOE} = \text{level of evidence; } \]
\[\text{NCEP ATP III} = \text{National Cholesterol Education Program Adult Treatment Panel III.} \]

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.
IV. 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 (see 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 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)*

*continued on page 18*
**Figure 3. Diagnosis of Claudication and Systemic Risk Treatment**

**Classic Claudication Symptoms:**
Muscle fatigue, cramping, or pain that reproducibly begins during exercise and that promptly resolves with rest

Chart document the history of walking impairment (pain-free and total walking distance) and specific lifestyle limitations

Document pulse examination

**ABI**

**Exercise ABI**
(TBI, segmental pressure, or duplex ultrasound examination)

**Risk factor normalization:**
- Immediate smoking cessation
- Treat hypertension: JNC-7 guidelines
- Treat lipids: NCEP ATP III guidelines
- Treat diabetes mellitus: HbA1c less than 7%*

**Abnormal results**
**Normal results**

**Confirmed PAD diagnosis**

**Pharmacological risk reduction:**
Antiplatelet therapy (ACE inhibition;† Class IIa)

**Go to Figure 4, Treatment of Claudication**

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*It is not yet proven that treatment of diabetes mellitus will significantly reduce peripheral arterial disease (PAD)-specific (limb ischemic) end points. Primary treatment of diabetes mellitus should be continued according to established guidelines.

†The benefit of angiotensin-converting enzyme (ACE) inhibition in individuals without claudication has not been specifically documented in prospective clinical trials but has been extrapolated from other at-risk populations.

Figure 4. Treatment of Claudication

Confirmed PAD Diagnosis

No significant functional disability

- No claudication treatment required.
- Follow-up visits at least annually to monitor for development of leg, coronary, or cerebrovascular ischemic symptoms.

Lifestyle-limiting symptoms

Supervised exercise program

Three-month trial

Preprogram and postprogram exercise testing for efficacy

Clinical improvement: Follow-up visits at least annually

Lifestyle-limiting symptoms with evidence of inflow disease*

Pharmacological therapy: Cilostazol (Pentoxifylline)

Three-month trial

Significant disability despite medical therapy and/or inflow endovascular therapy, with documentation of outflow† PAD, with favorable procedural anatomy and procedural risk-benefit ratio

Evaluation for additional endovascular or surgical revascularization

Further anatomic definition by more extensive noninvasive or angiographic diagnostic techniques

Endovascular therapy or surgical bypass per anatomy

*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).

PAD = peripheral arterial disease.
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)*

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**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 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)

The key elements of a therapeutic claudication exercise program for patients with claudication are summarized in Table 4, page 23. For diagnosis and treatment of critical and acute limb ischemia see Figures 5, 6, and 7.
**Figure 5. Diagnosis and Treatment of Critical Limb Ischemia**

**Chronic symptoms:** Ischemic rest pain, gangrene, nonhealing wound

**Ischemic etiology must be established promptly:**
By examination and objective vascular studies

**Implication:** Impending limb loss

History and physical examination:
- Document lower extremity pulses
- Document presence of ulcers or infection

Assess factors that may contribute to limb risk: diabetes, neuropathy, chronic renal failure, infection

**Obtain prompt vascular specialist consultation:**
- Diagnostic testing strategy
- Creation of therapeutic intervention plan

Patient is a candidate for revascularization

- Define limb arterial anatomy
- Assess clinical and objective severity of ischemia

Imaging of relevant arterial circulation (noninvasive and angiographic)

Revascularization possible (see treatment text, with application of thrombolytic, endovascular, and surgical therapies)

Revascularization not possible†:
- Medical therapy; amputation (when necessary)

Ongoing vascular surveillance (see text)‡

Written instructions for self-surveillance

Patient is not a candidate for revascularization*

Medical therapy or amputation (when necessary)

Severe lower extremity PAD documented:
- ABI less than 0.4; flat PVR waveform; absent pedal flow

Systemic antibiotics if skin ulceration and limb infection are present

**ABI** = ankle-brachial index;
**CTA** = computed tomographic angiography;
**ECG** = electrocardiogram;
**MRA** = magnetic resonance angiography;
**PVR** = pulse volume recording;
**TBI** = toe-brachial index;
**TEE** = transesophageal echocardiography;
**US** = ultrasonography.

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*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 (HgbA1c [hemoglobin A1c] less than 7%; Class Iia). It is not yet proven that treatment of diabetes mellitus will significantly reduce peripheral arterial disease (PAD)-specific (limb ischemic) end points. Primary treatment of diabetes mellitus should be continued according to established guidelines.
Lower Extremity

Rapid or sudden decrease in limb perfusion threatens tissue viability

History and physical examination; determine time of onset of symptoms

Emergent assessment of severity of ischemia:
- Loss of pulses
- Loss of motor and sensory function
- Vascular laboratory assessment

ABI, TBI, or duplex US

No or minimal atherosclerotic arterial occlusive disease

Consider atheroembolism, thromboembolism, or phlegmasia cerulea dolens

Evaluation of source (ECG or Holter monitor; TEE; and/or abdominal US, MRA, or CTA); or venous duplex

Severe PAD documented:
- ABI less than 0.4
- Flat PVR waveform
- Absent pedal flow

Go to Figure 7, Treatment of Acute Limb Ischemia

No or minimal PAD

Consider atheroembolism, thromboembolism, or phlegmasia cerulea dolens

Evaluation of source (ECG or Holter monitor; TEE; and/or abdominal US, MRA, or CTA); or venous duplex

ABI = 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.
Severe PAD documented:
ABI less than 0.4; flat PVR waveform; absent pedal flow

Immediate anticoagulation:
Unfractionated heparin or low molecular weight heparin

Obtain prompt vascular specialist consultation:
Diagnostic testing strategy
Creation of therapeutic intervention plan

Assess etiology:
- Embolic (cardiac, aortic, infrainguinal sources)
- Progressive PAD and in situ thrombosis (prior claudication history)
- Leg bypass graft thrombosis
- Arterial trauma
- Popliteal cyst or entrapment
- Phlegmasia cerulea dolens
- Ergotism
- Hypercoagulable state

Viable limb
- Not immediately threatened
- No sensory loss
- No muscle weakness
- Audible arterial and venous US

Salvageable limb: threatened marginally (reversible ischemia)
- Salvageable if promptly treated
- Minimal (toes) or no sensory loss
- No muscle weakness
- Inaudible (often) arterial Doppler signals
- Audible venous Doppler signals

Salvageable limb: threatened immediately (reversible ischemia)
- Salvageable with immediate revascularization
- Sensory loss more than toes, associated with rest pain
- Mild to moderate muscle weakness
- Inaudible (usually) arterial Doppler signals
- Audible venous Doppler signals

Salvageable limb: threatened marginally (irreversible ischemia)
- Major tissue loss or permanent nerve damage inevitable
- Profound, anesthetic sensory loss
- Profound paralysis (rigor)
- Inaudible arterial Doppler signals
- Inaudible venous Doppler signals

Nonviable limb
- Amputation

Guides to treatment:
- Site and extent of occlusion
- Embolus versus thrombus
- Native artery versus bypass graft
- Duration of ischemia
- Patient comorbidities
- Contraindications to thrombolysis or surgery

Revascularization: Thrombolysis, endovascular, surgical

ABI = 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.
### 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

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PAD = peripheral arterial disease; ABI = ankle-brachial index.

# Endovascular Treatment of Claudication

## Class I

1. Endovascular procedures are indicated for individuals with a vocational or lifestyle-limiting 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 risk-benefit ratio (e.g., focal aortoiliac 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*, next page, and *Figure 8*, page 28) 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)*

## 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 greater than 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 less than 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 less than 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 more than 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 = common femoral artery; CIA = common iliac artery; EIA = external iliac artery; TASC = TransAtlantic Inter-Society Consensus.

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Table 6. Morphological Stratification of Femoropopliteal Lesions

**TASC type A femoropopliteal lesions:**

1. Single stenosis less than 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 less than 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* = TransAtlantic Inter-Society Consensus.

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

Type A
Endovascular Treatment of Choice

<3cm <3cm

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

3-10 cm 3-5 cm 3-5 cm

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

5-10 cm 5-10 cm

Type D
Surgical treatment of choice

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**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

Critical limb ischemia (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 ischemic 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 Critical Limb Ischemia

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, intra-arterial pressure measurements across suprainguinal lesions should be measured before and after the administration of a vasodilator. (Level of Evidence: C)
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 arterial 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 Critical Limb Ischemia

**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 less than 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 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)*
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.

Recommendations

Class I

1. Long-term patency of infrainguinal bypass grafts should be evaluated in a surveillance program (see Table 7), 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)

2. Duplex ultrasound is recommended for routine surveillance following 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. 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

ABI = ankle-brachial index.

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V. 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:
  - accelerated hypertension (sudden and persistent worsening of previously controlled hypertension);
  - 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);
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)*

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 two kidneys of greater than 1.5 cm. *(Level of Evidence: B)*

sudden, unexplained pulmonary edema (especially in azotemic patients).
*(Level of Evidence: B)*

*continued next page*
**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 disease and none of the clinical clues (see Figure 9) 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 Section 3.5.2.4 of the full-text guidelines). *(Level of Evidence: C)*
**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)

![Diagram](image)


†For example, atrophic kidney due to chronic pyleonephritis is not an indication for renal artery stenosis (RAS) evaluation.

ACE = 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 tomographic 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. 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 base 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

Hemodynamically significant RAS with recurrent, unexplained CHF or sudden, unexplained pulmonary edema (see full-text guideline, Section 3.5.2.4) (Class I; LOE B)

RAS and CRI with bilateral RAS or RAS to solitary functioning kidney (see full-text guideline, Section 3.5.2.3) (Class IIa; LOE B)

Asymptomatic bilateral or solitary viable* kidney with a hemodynamically significant RAS (Class IIb; LOE C)

RAS and CRI with unilateral RAS (2 kidneys present) (Class IIb; LOE C)

RAS with:
- Accelerated, resistant, or malignant hypertension
- Hypertension with unilateral small kidney
- Hypertension with medication intolerance (Class IIa; LOE B)

RAS and unstable angina (see full-text guideline, Section 3.5.2.4) (Class IIa; LOE B)

Asymptomatic unilateral hemodynamically significant RAS in a viable* kidney (Class IIb; LOE C)

Renal angioplasty/stent†

Renal artery surgery†

Atherosclerotic RAS

Stent use is indicated in patients who meet criteria for intervention (see full-text guideline, Section 3.5.3) (Class I; LOE B)

Fibromuscular dysplasia RAS

PTA (with “bailout” stent use) is indicated for patients meeting criteria for intervention (see full-text guideline, Section 3.5.3) (Class I; LOE B)

*Viable means kidney linear length greater than 7 cm.
†It is recognized that renal artery surgery has proven efficacy in alleviating renal artery stenosis (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 = congestive heart failure; CRI = chronic renal insufficiency; LOE = level of evidence.
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 full-text guidelines). *(Level of Evidence: B)*

Class IIa
1. Percutaneous revascularization is reasonable for patients with hemodynamically significant RAS and unstable angina (see full-text guidelines, Section 3.5.2.4). *(Level of Evidence: B)*
D. Treatment Methods: Medical, Endovascular, and Surgical

Pharmacological Treatment of Individuals With Renal Artery Stenosis

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.

Recommendations for Pharmacological Treatment of Renal Artery Stenosis

Class I

1. ACE inhibitors are effective medications for treatment of hypertension associated with 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)*
Catheter-Based Interventions for Renal Artery Stenosis

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 Renal Artery Stenosis

Class I

1. Vascular surgical reconstruction is indicated for patients with

   - fibromuscular dysplastic RAS with clinical indications for interventions (same as percutaneous transluminal angioplasty), especially those exhibiting complex disease that extends 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 pararenal aortic reconstructions (in treatment of aortic aneurysms or severe aortoiliac occlusive disease). (*Level of Evidence: C*)
VI. 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 myocardial infarction 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 for 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 after the revascularization. *(Level of Evidence: B)*

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**Endovascular Treatment for Acute Intestinal Ischemia**

**Class IIb**
1. Percutaneous interventions (including trans-catheter 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)*

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**B. Acute Nonocclusive Intestinal Ischemia**

**Diagnosis and Treatment of 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, ergots, vasopressin, or norepinephrine) 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)*

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**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 tomographic 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*)
VII. 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 arterial 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 Abdominal Aortic Aneurysms

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)*
**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 Abdominal Aortic Aneurysms**

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 computed tomography (CT) 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 two to three 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)*
Figure 11. Management of Abdominal Aortic Aneurysms

Abdominal Aortic Aneurysm

Symptomatic intact

Infrarenal

Asymptomatic

Smaller than 4 cm

Biennial ultrasound scan

4 to 5.4 cm

Ultrasound scan every 6 to 12 months

Greater than or equal to 5.5 cm or growth spurt

Contrast CT or MR scan

Medical evaluation

Low or average risk

Elective open repair

High risk

Endograft repair if aortic anatomy appropriate

Continued CT or MR surveillance

Symptoms or growth spurt

Pararenal, suprarenal, or type IV thoracoabdominal

Ruptured

Asymptomatic

Smaller than 4 cm

Annual contrast CT or MR scan

4 to 5.4 cm

Contrast CT or MR scan every 6 to 12 months

Greater than or equal to 5.5 cm or growth spurt

Symptomatic intact

Medical evaluation

Low or average risk

Elective open repair

High risk

Continued CT or MR surveillance

Symptoms or growth spurt

Urgent open repair

Contrast CT or MR scan every 6 to 12 months

CT = computed tomography; MR = magnetic resonance.
**Management Overview of Prevention of Aortic Aneurysm Rupture**

**Class I**

1. 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)*

2. 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: B)*

**Class IIa**

1. 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)*
B. Visceral Artery Aneurysms

Visceral artery aneurysms are insidious because they usually cannot be detected by physical examination and may be overlooked on radiographs or CT/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 multiparity.

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)
C. Lower Extremity Artery Aneurysms

In general, lower extremity artery aneurysms are considered to be significant when the minimum diameter reaches 3.0 cm (common femoral) to 2.0 cm (popliteal). The presence of a lower extremity artery aneurysm should lead to examination for the presence of an AAA (see Figure 12).

Unlike AAAs, the natural history of extremity artery aneurysms is not one of expansion and rupture but one of thromboembolism or thrombosis.

**Recommendations for Management of Lower Extremity Artery Aneurysms**

**Class I**

1. In patients with femoral or popliteal aneurysms, ultrasound (or CT or 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)*

*continued on page 60*
**Figure 12. Diagnostic and Treatment Algorithm for Popliteal Mass**

- **Popliteal mass**
- **Duplex scan**
  - **Vascular**
    - **Screen for incidental aortic aneurysm**
    - **Symptoms**
      - Yes: **CT or arteriogram for runoff**
      - No: **Diameter greater than 2 cm**
        - No: **Observe yearly duplex scan**
        - Yes: **Operate**
  - **Not vascular**
    - **Manage as per nonvascular diagnosis**
    - **Adequate runoff**
      - Yes: **Catheter-directed thrombolysis**
      - No: **Operate**

*CT* = computed tomography.
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)*

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**D. 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).*
Figure 13. Diagnostic and Treatment Algorithm for Femoral Pseudoaneurysm

- Suspected catheter-related femoral pseudoaneurysm
- Duplex scan confirms pseudoaneurysm
  - Asymptomatic pseudoaneurysm
    - Small (Less than 2 cm)
      - Observe Duplex scan in 1 month
    - Persistent pseudoaneurysm
  - Symptomatic pseudoaneurysm
    - Large and/or multi-chambered
    - Minor local discomfort
      - Skin erosion
      - AV fistula
      - Nerve compression
      - Expanding hematoma
  - Nonoperative intervention
    - Ultrasound-guided manual compression
    - Ultrasound-guided thrombin injection
      - Observe Duplex scan
      - Failed therapy
      - Operate

AV = arteriovenous.
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. Re-evaluation 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)*
Lower Extremity Aneurysms

History/Exam

Eval/Treat

Renal Mesenteric