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ACC/AHA PRACTICE GUIDELINES

ACC/AHA 2005 Guidelines for the Management of Patients With Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic): Executive Summary

A Collaborative Report From the American Association for Vascular Surgery/Society for Vascular Surgery,* Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease)

Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation

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writing panel are asked to provide disclosure statements of all such relationships that might be perceived as real or potential conflicts of interest. These statements are reviewed by the parent task force, reported orally to all members of the writing panel at the first meeting, and updated as changes occur. The relationship with industry information for writing committee members, as well as peer reviewers of the document, is located in an appendix of the full-text guideline, which is available on the ACC, AHA, SCAI, SVMB, SVS, SIR, and VDF (see "Copies" for Web addresses).

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I. INTRODUCTION

These guidelines address the diagnosis and management of atherosclerotic, aneurysmal, and thromboembolic peripheral arterial diseases (PADs). The clinical manifestations of PAD are a major cause of acute and chronic illness, are associated with decrements in functional capacity and quality of life, cause limb amputation, and increase the risk of death. Whereas the term "peripheral arterial disease" encompasses a large series of disorders that affect arterial beds exclusive of the coronary arteries, this writing committee chose to limit the scope of the work of this document to include the disorders of the abdominal aorta, renal and mesenteric arteries, and lower extremity arteries. The purposes of the full guidelines are to (a) aid in the recognition, diagnosis, and treatment of PAD of the aorta and lower extremities, addressing its prevalence, impact on quality of life, cardiovascular ischemic risk, and risk of critical limb ischemia (CLI); (b) aid in the recognition, diagnosis, and treatment of renal and visceral arterial diseases; and (c) improve the detection and treatment of abdominal and branch artery aneurysms. Clinical management guidelines for other arterial beds (e.g., the thoracic aorta, carotid and vertebral arteries, and upper-extremity arteries) have been excluded from the current guidelines to focus on the infradiaphragmatic arterial system and in recognition of the robust evidence base that exists for the aortic, visceral, and lower extremity arteries.

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The reader should note that the text, recommendations, and tables included in this executive summary represent a succinct summary of the more extensive evidence base, critical evaluation, tables, and references that are included in the full-text document. Readers are strongly encouraged to refer to this source document to gain full access to the 65 tables, more than 1300 references, and supporting text assembled by the writing committee. The full-text document can be accessed at http://www.acc.org/clinical/guidelines/pad/index.pdf.

A classification of recommendation and level of evidence have been assigned to each recommendation. Classifications of recommendations and levels of evidence are expressed in the American College of Cardiology (ACC)/American Heart Association (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. (Please refer to Table 1 in the full-text guidelines for more details.)

A. Definitions

For the purposes of these guidelines, the term "peripheral arterial disease" broadly encompasses the vascular diseases caused primarily by atherosclerosis and thromboembolic pathophysiological processes that alter the normal structure and function of the aorta, its visceral arterial branches, and the arteries of the lower extremity. Peripheral arterial disease 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.

B. Vascular History and Physical Examination

RECOMMENDATIONS

Class I

1. **Individuals at risk for lower extremity PAD (see Section 2.1.1 of the full-text guidelines) should undergo a vascular review of symptoms to assess walking impairment, claudication, ischemic rest pain, and/or the presence of nonhealing wounds.** ***(Level of Evidence: C)***
2. **Individuals at risk for lower extremity PAD (see Section 2.1.1 of the full-text guidelines) should undergo comprehensive pulse examination and inspection of the feet.** ***(Level of Evidence: C)***
3. **Individuals over 50 years of age should be asked if they have a family history of a first-order relative with an abdominal aortic aneurysm.** ***(Level of Evidence: C)***

The full-text guidelines offer suggestions for creation of a vascular review of systems as outlined below.

Key components of the vascular review of systems (not usually included in the review of systems of the extremities) and family history include the following:

- Any exertional limitation of the lower extremity muscles or any history of walking impairment. The characteristics of this limitation may be described as fatigue, aching, numbness, or pain. The primary site(s) of discomfort in the buttock, thigh, calf, or foot should be recorded, along with the relation of such discomfort to rest or exertion.
- 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.

The Vascular Physical Examination

Key components of the vascular physical examination are as follows:

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

II. LOWER EXTREMITY PAD

A. Epidemiology

1. Risk Factors. The major cause of lower extremity PAD is atherosclerosis. Risk factors for atherosclerosis such as cigarette smoking, diabetes, dyslipidemia, hypertension, and hyperhomocysteinemia increase the likelihood of developing lower extremity PAD (Fig. 1).

2. Prevalence. Lower extremity PAD is a common syndrome that affects a large proportion of most adult populations worldwide (1,2). Peripheral arterial disease can be present in subclinical forms that can be detected by use of sensitive vascular imaging techniques, which may reveal early manifestations of arterial disease before it is detected by either limb-pressure measurements or clinical symptoms. When so defined, as, for example, by measurement of the intimal-medial thickness (IMT) in the carotid or femoral artery, early forms of PAD are easily detected in populations at risk (3). Claudication, a symptomatic expression of lower extremity PAD, defines a significantly smaller subset of the total population with the disease.

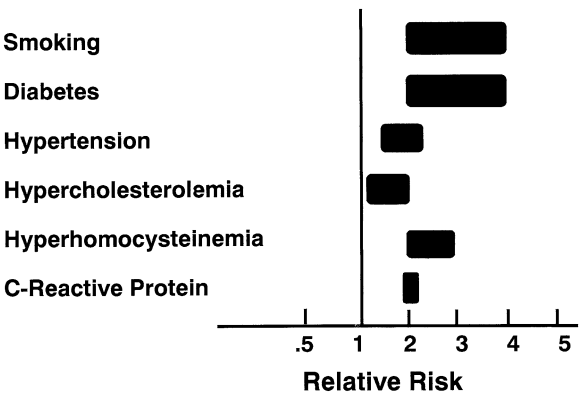

Figure 1. Risk of developing lower extremity peripheral arterial disease. The range for each risk factor is estimated from epidemiologic studies (see text). The relative risks take into consideration current smokers vs. former smokers and nonsmokers; the presence vs. the absence of diabetes and hypertension; and the highest vs. the lowest quartile of homocysteine and C-reactive protein. The estimate for hypercholesterolemia is based on a 10% risk for each 10 mg/dl rise in total cholesterol. 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 (16).

B. Prognosis and Natural History

1. Coprevalence of Coronary Arterial Disease and Carotid Disease. The prognosis of patients with lower extremity PAD is characterized by an increased risk for cardiovascular ischemic events due to concomitant coronary artery disease and cerebrovascular disease (1,4). These cardiovascular ischemic events are more frequent than ischemic limb events in any lower extremity PAD cohort, whether individuals present without symptoms or with atypical leg pain, classic claudication, or CLI (Fig. 2) (5).

C. Other Causes of Lower Extremity PAD

Peripheral arterial disease has a diversity of causes beyond atherosclerosis. Aneurysms may be associated with atherosclerosis, may be due to underlying hereditary (familial) reasons, or may be of acquired (e.g., due to smoking or trauma) origin. Renal arterial disease may be due to atherosclerosis, fibromuscular dysplasia, or arteritides. Lower extremity PAD may be caused by atherosclerotic, thromboembolic, inflammatory, or aneurysmal disease; by trauma, adventitial cysts, or entrapment syndromes; or by congenital abnormalities. Establishment of an accurate diagnosis is necessary if individual patients are to receive ideal pharmacological, endovascular, surgical, or rehabilitative interventions.

D. Clinical Presentation

1. Asymptomatic.

RECOMMENDATIONS

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) so that therapeutic interventions known to diminish their increased risk of myocardial infarction (MI), stroke, and death may be offered.** ***(Level of Evidence: B)***
3. **Smoking cessation, lipid lowering, and diabetes and hypertension treatment according to current national treatment guidelines are recommended for individuals with asymptomatic lower extremity PAD.** ***(Level of Evidence: B)***
4. **Antiplatelet therapy is indicated for individuals with asymptomatic lower extremity PAD to reduce the risk of adverse cardiovascular ischemic events.** ***(Level of Evidence: C)***

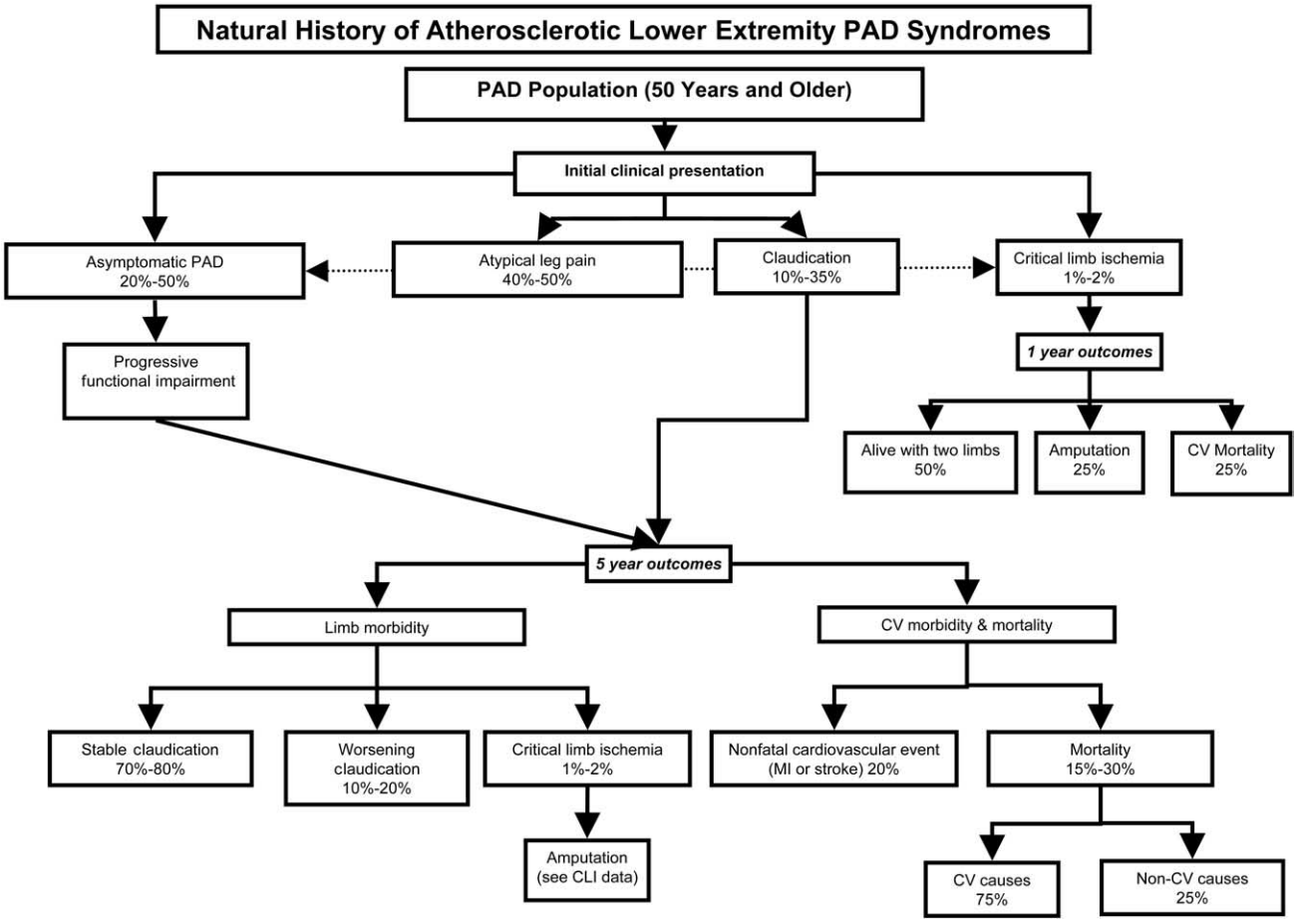

Figure 2. The natural history of atherosclerotic lower extremity peripheral arterial disease (PAD). Individuals with atherosclerotic lower extremity PAD may be: (a) asymptomatic (without identified ischemic leg symptoms, albeit with a functional impairment); (b) present with leg symptoms (classic claudication or atypical leg symptoms); or (c) present with critical limb ischemia. All individuals with PAD face a risk of progressive limb ischemic symptoms, as well as a high short-term cardiovascular ischemic event rate and increased mortality. These event rates are most clearly defined for individuals with claudication or critical limb ischemia (CLI), and less well defined for individuals with asymptomatic PAD. CV = cardiovascular; MI = myocardial infarction. Adapted with permission from Weitz JL et al. Circulation 1996;94:3026–49 (5).

Class IIa

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

Class IIb

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

Current data document that lower extremity PAD is common, that the traditional term "asymptomatic" may inaccurately imply that limb function is normal, and that lower extremity PAD is invariably and independently associated with impaired lower extremity functioning (6–8). Thus, most individuals with lower extremity PAD do not have classic (typical) claudication but may have more subtle impairments of lower extremity function.

Table 1. 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

Individuals with asymptomatic lower extremity PAD are characterized by a risk factor profile comparable to that of those with symptomatic lower extremity PAD (9,10). The high prevalence of diabetes, a history of past or current smoking, hypertension, and/or hypercholesterolemia place such individuals at a markedly increased risk of atherosclerotic ischemic events, including MI and stroke (9,11) and higher degrees of internal carotid artery stenosis (12,13). Given these data, current U.S. national hypertension, lipid, and antiplatelet treatment guidelines include all patients with lower extremity PAD, regardless of symptom status, as a "high-risk" category. All patients with lower extremity PAD should achieve risk reduction and specific treatment targets comparable to those of individuals with established coronary artery disease (14,15).

The responsibility for the detection of lower extremity PAD should be with the primary care provider. Programs of lower extremity PAD detection, whether applied in office practice or in community-based detection programs, should ideally utilize the epidemiological database to apply the detection tool to a population "at risk." The most cost-effective tool for lower extremity PAD detection is the ABI, which has been used in numerous field surveys and cross-sectional practice surveys, as cited in the full-text guidelines.

2. Claudication.

RECOMMENDATIONS

Class I

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

Class III

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

Vascular claudication due to lower extremity PAD is produced consistently by exercise and is relieved with rest and is therefore traditionally referred to as "intermittent" claudication, or simply "claudication." The severity of the symptoms can be classified according to either the Fontaine or Rutherford categories (Table 2).

Vascular claudication must be distinguished from other disorders that cause exertional leg pain, which has been called "pseudoclaudication." Distinguishing features of these various causes of leg pain are summarized in Table 3 (16).

The ABI should be measured in all patients with claudication. For individuals who present with classic claudication and in whom the ABI is borderline or normal (0.91 to 1.30) or supranormal (greater than 1.30), alternative diagnostic strategies should be used (including the toe-brachial index, segmental pressure examination, or duplex ultrasound), to confirm the lower extremity PAD diagnosis (see Section 2.5 of the full-text guidelines).

Table 2. Classification of Peripheral Arterial Disease: Fontaine's Stages and Rutherford's Categories

Fontaine		Rutherford		
Stage	**Clinical**	**Grade**	**Category**	**Clinical**
I	Asymptomatic	0	0	Asymptomatic
IIa	Mild claudication	I	1	Mild claudication
IIb	Moderate-severe claudication	I	2	Moderate claudication
		I	3	Severe claudication
III	Ischemic rest pain	II	4	Ischemic rest pain
IV	Ulceration or gangrene	III	5	Minor tissue loss
		IV	6	Ulceration or gangrene

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Table 3. Differential Diagnosis of Intermittent Claudication

Condition	Location of Pain or Discomfort	Characteristic Discomfort	Onset Relative to Exercise	Effect of Rest	Effect of Body Position	Other Characteristics
Intermittent claudication	Buttock, thigh, or calf muscles and rarely the foot	Cramping, aching, fatigue, weakness, or frank pain	After same degree of exercise	Quickly relieved	None	Reproducible
Nerve root compression (e.g., herniated disc)	Radiates down leg, usually posteriorly	Sharp lancinating pain	Soon, if not immediately after onset	Not quickly relieved (also often present at rest)	Relief may be aided by adjusting back position	History of back problems
Spinal stenosis	Hip, thigh, buttocks (follows dermatome)	Motor weakness more prominent than pain	After walking or standing for variable lengths of time	Relieved by stopping only if position changed	Relief by lumbar spine flexion (sitting or stooping forward)	Frequent history of back problems, provoked by intra-abdominal pressure
Arthritic, inflammatory processes	Foot, arch	Aching pain	After variable degree of exercise	Not quickly relieved (and may be present at rest)	May be relieved by not bearing weight	Variable, may relate to activity level
Hip arthritis	Hip, thigh, buttocks	Aching discomfort, usually localized to hip and gluteal region	After variable degree of exercise	Not quickly relieved (and may be present at rest)	More comfortable sitting, weight taken off legs	Variable, may relate to activity level, weather changes
Symptomatic Baker's cyst	Behind knee, down calf	Swelling, soreness, tenderness	With exercise	Present at rest	None	Not intermittent
Venous claudication	Entire leg, but usually worse in thigh and groin	Tight, bursting pain	After walking	Subsides slowly	Relief speeded by elevation	History of iliofemoral deep vein thrombosis, signs of venous congestion, edema
Chronic compartment syndrome	Calf muscles	Tight, bursting pain	After much exercise (e.g., jogging)	Subsides very slowly	Relief speeded by elevation	Typically occurs in heavy muscled athletes

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Most individuals with claudication benefit from a comprehensive medical approach that includes risk factor modification, antiplatelet therapy, exercise rehabilitation, and use of claudication medications (see Sections 2.6.1 and 2.6.2). In these individuals, more complex imaging studies are not required for effective management. Decisions regarding revascularization of individuals with claudication should be based on the severity of symptoms, a significant disability as assessed by the patient, failure of medical therapies, lack of significant comorbid conditions, vascular anatomy suitable for the planned revascularization, and a favorable risk-benefit ratio. These recommendations are summarized in Table 4 (16). Patients selected for possible revascularization may then undergo additional imaging studies as required to determine whether their arterial anatomy is suitable for percutaneous or surgical revascularization.

3. Critical Limb Ischemia.

RECOMMENDATIONS

Class I

1. **Patients with CLI should undergo expedited evaluation and treatment of factors that are known to increase the risk of amputation (see text).** ***(Level of Evidence: C)***

Table 4. Indications for Revascularization in Intermittent Claudication

Before offering a patient with intermittent claudication the option of any invasive revascularization therapy, whether endovascular or surgical, the following considerations must be taken into account:

- A predicted or observed lack of adequate response to exercise therapy and claudication pharmacotherapies
- Presence of a severe disability, either being unable to perform normal work or having very serious impairment of other activities important to the patient
- Absence of other disease that would limit exercise even if the claudication was improved (e.g., angina or chronic respiratory disease)
- The individual's anticipated natural history and prognosis
- The morphology of the lesion (must be such that the appropriate intervention would have low risk and a high probability of initial and long-term success)

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2. **Patients with CLI in whom open surgical repair is anticipated should undergo assessment of cardiovascular risk.** ***(Level of Evidence: B)***
3. **Patients with a prior history of CLI or who have undergone successful treatment for CLI should be evaluated at least twice annually by a vascular specialist owing to the relatively high incidence of recurrence.** ***(Level of Evidence: C)***
4. **Patients at risk of CLI (ABI less than 0.4 in a nondiabetic individual, or any diabetic individual with known lower extremity PAD) should undergo regular inspection of the feet to detect objective signs of CLI.** ***(Level of Evidence: B)***
5. **The feet should be examined directly, with shoes and socks removed, at regular intervals after successful treatment of CLI.** ***(Level of Evidence: C)***
6. **Patients with CLI and features to suggest atheroembolization should be evaluated for aneurysmal disease (e.g., abdominal aortic, popliteal, or common femoral aneurysms).** ***(Level of Evidence: B)***
7. **Systemic antibiotics should be initiated promptly in patients with CLI, skin ulcerations, and evidence of limb infection.** ***(Level of Evidence: B)***
8. **Patients with CLI and skin breakdown should be referred to healthcare providers with specialized expertise in wound care.** ***(Level of Evidence: B)***
9. **Patients at risk for CLI (those with diabetes, neuropathy, chronic renal failure, or infection) who develop acute limb symptoms represent potential vascular emergencies and should be assessed immediately and treated by a specialist competent in treating vascular disease.** ***(Level of Evidence: C)***
10. **Patients at risk for or who have been treated for CLI should receive verbal and written instructions regarding self-surveillance for potential recurrence.** ***(Level of Evidence: C)***

Critical limb ischemia is defined by most vascular clinicians in patients who present with lower extremity ischemic rest pain, ulceration, or gangrene. In these individuals, the untreated natural history of severe PAD would lead to major limb amputation within 6 months. The Rutherford clinical categories (described earlier) are used to classify the degree of ischemia and salvageability of the limb. Critical limb ischemia is also a component of the Fontaine clinical classification system (Table 2).

Patients with CLI usually present with limb pain at rest, with or without trophic skin changes or tissue loss. The discomfort is often worse when the patient is supine (e.g., in bed) and may lessen when the limb is maintained in the dependent position. Typically, narcotic medications are required for analgesia. Those factors that are known to increase the risk of limb loss in patients with CLI are delineated in Table 5.

It is fundamentally important for the clinician to determine the time course of development of the ischemia. If the clinical history and physical examination suggest relatively rapid progression, then early or "semiurgent" revascularization may be required to prevent further deterioration and irreversible tissue loss. A vascular history should also be obtained. This should include evaluation for arterial disease in other territories, assessment of global risk factors for atherosclerosis, and clarification of any specific precipitating factors or events (e.g., trauma or infection) that may have caused initial skin ulceration. The objectives for the diagnostic evaluation of patients with CLI are summarized in Table 6. Specific investigations that are helpful in evaluating patients with CLI are summarized in Table 7.

Distinctions should be made between ulcers that are arterial and those that are venous or neurotrophic (Tables 8, 9, and 10).

The evaluation of patients presenting with CLI should include a complete blood count, chemistries (including blood glucose and renal function tests), electrocardiogram, and ABI. In the absence of "noncompressible vessels," measurement of an absolute systolic blood pressure 50 mm Hg or lower at the ankle and 30 mm Hg at the toe will often imply that amputation may be required in the absence of successful revascularization (16,17). Individuals with CLI

Table 5. Factors That Increase Risk of Limb Loss in Patients With Critical Limb Ischemia

Factors that reduce blood flow to the microvascular bed:

- Diabetes
- Severe renal failure
- Severely decreased cardiac output (severe heart failure or shock)
- Vasospastic diseases or concomitant conditions (e.g., Raynaud's phenomenon, prolonged cold exposure)
- Smoking and tobacco use

Factors that increase demand for blood flow to the microvascular bed:

- Infection (e.g., cellulitis, osteomyelitis)
- Skin breakdown or traumatic injury

Table 6. Objectives for Diagnostic Evaluation of Patients With Critical Limb Ischemia

The diagnostic evaluation of patients with critical limb ischemia should be directed toward the following objectives:
- Objective confirmation of the diagnosis
- Localization of the responsible lesion(s) and a gauge of relative severity
- Assessment of the hemodynamic requirements for successful revascularization (vis-à-vis proximal versus combined revascularization of multilevel disease)
- Assessment of individual patient endovascular or operative risk

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who present with clinical features to suggest atheroembolization should be evaluated for more proximal aneurysmal disease (e.g., abdominal aortic, popliteal, or common femoral aneurysms). Atheroembolism is suggested by the onset of signs and symptoms of CLI after recent endovascular catheter manipulation, the onset of associated systemic fatigue or muscle discomfort, symmetrical bilateral limb symptoms, livido reticularis, or rising creatinine values.

Treatment of CLI is dependent on increasing blood flow to the affected extremity to relieve pain, heal ischemic ulcerations, and avoid limb loss. Individuals with minimal or no skin breakdown or in whom comorbid conditions prevent consideration of revascularization can occasionally be treated by medical therapies in the absence of revascularization. Medical care strategies have included the use of antiplatelet agents, anticoagulant medications, intravenous prostanoids, rheologic agents, and maintenance of the limb in a dependent position. However, none of these clinical interventions has been adequately evaluated or proven in prospective clinical trials to offer predictable improvements in limb outcomes. Recent investigation of angiogenic therapies, via administration of gene or protein, to enhance collateral blood flow has offered promise as a potential strategy to treat CLI; however, these are not yet proven interventions and are not available as established therapies.

In the absence of revascularization, most patients with CLI are expected to require amputation within 6 months. Therefore, timely referral to a vascular specialist is indicated. Detailed arterial mapping requires vascular expertise to (a) identify the cause of the ischemia and (b) define the options available for revascularization.

4. Acute Limb Ischemia.

RECOMMENDATIONS

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)***

Acute limb ischemia arises when a rapid or sudden decrease in limb perfusion threatens tissue viability. This form of CLI may be the first manifestation of arterial disease in a previously asymptomatic patient or may occur as an acute event that causes symptomatic deterioration in a patient with antecedent lower extremity PAD and intermittent claudication. Although attempts have been made to define various levels of ischemia (18), it is frequently not possible to precisely delineate the status of the patient with an acutely ischemic limb, because many of the classification schemes are based on subjective clinical criteria and not discrete end points. Table 11 displays the Society for Vascular Surgery/International Society for Cardiovascular Surgery classification scheme and provides the most useful clinical method to describe this entity.

The hallmark clinical symptoms and physical examination signs of acute limb ischemia include the 5 "Ps" that suggest limb jeopardy: pain, paralysis, paresthesias, pulseless, and pallor. Some clinicians would also include a sixth "P," polar, to indicate a cold extremity. However, arterial embolism can occur without symptoms, whereas thrombosis can produce sudden, severe limb ischemia. The clinical diagnosis of arterial embolism is suggested by (a) the sudden onset or sudden worsening of symptoms, (b) a known embolic source, (c) the absence of antecedent claudication or other manifestations of obstructive arterial disease, or (d) the presence of normal arterial pulses and Doppler systolic blood pressures in the contralateral limb. Acute limb ischemia is a situation that

Table 7. Investigations for Evaluating Patients With Critical Limb Ischemia (CLI)

To achieve the objectives listed in Table 6, the following investigations should be used in patients with CLI:
- Clinical history and examination, including the coronary and cerebral circulation
- Hematologic and biochemical tests: complete blood count, platelet count, fasting blood glucose, hemoglobin A_{1c}, creatinine, fasting lipid profile, and urinalysis (for glycosuria and proteinuria)
- Resting electrocardiogram
- Ankle or toe pressure measurement or other objective measures for the severity of ischemia
- Imaging of the lower-limb arteries in patients considered for endovascular or surgical intervention
- Duplex scan of the carotid arteries should be considered in selected patients at high risk (defined as individuals with cerebrovascular ischemic symptoms or in whom the risk of carotid revascularization is less than the short-term risk of stroke)
- A more detailed coronary assessment may be performed in selected patients in whom coronary ischemic symptoms would otherwise merit such an assessment if CLI were not present (such coronary assessments should usually not impede associated CLI care)

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Table 8. Differential Diagnosis of Common Foot and Leg Ulcers

Origin	Cause	Location	Pain	Appearance
Main arteries	Atherosclerotic lower extremity PAD, Buerger's disease, acute arterial occlusion	Toes, foot	Severe	Irregular, pink base
Venous	Venous disease	Malleolar	Mild	Irregular, pink base
Skin infarct	Systemic disease, embolism, hypertension	Lower third of leg	Severe	Small after infarction, often multiple
Neurotrophic	Neuropathy	Foot sole	None	Often deep, infected

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PAD = peripheral arterial disease.

requires prompt diagnosis and treatment to preserve the limb and prevent the systemic illness or death that might result from the metabolic abnormalities associated with tissue necrosis. Although the technical ability to recanalize or revascularize occluded arteries that perfuse ischemic tissues has improved significantly, the pathophysiology of the local and systemic clinical sequelae associated with reperfusion of an ischemic limb is only partially understood. Revascularization of an ischemic extremity may be complicated by reperfusion injury to the damaged tissues and precipitate systemic responses, including cardiac, renal, and pulmonary dysfunction.

5. Prior Limb Arterial Revascularization.

RECOMMENDATIONS

Class I

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

Class IIa

1. **Long-term patency of infrainguinal bypass grafts may be considered for evaluation in a surveillance program, which may include conducting exercise ABIs and other arterial imaging studies at regular intervals (see duplex ultrasound recommendations, Section 2.5.5 of the full-text guidelines).** ***(Level of Evidence: B)***
2. **Long-term patency of endovascular sites may be evaluated in a surveillance program, which may include conducting exercise ABIs and other arterial imaging studies at regular intervals (see duplex ultrasound recommendations, Section 2.5.5 of the full-text guidelines).** ***(Level of Evidence: B)***

Table 9. Foot Physical Examination and Differential Diagnosis of Neuropathic and Neuroischemic Ulcers

Neuropathic Ulcer	Neuroischemic Ulcer
Painless	Painful
Normal pulses	Absent pulses
Typically punched-out appearance	Irregular margins
Often located on sole or edge of foot or metatarsal head	Commonly located on toes
Presence of calluses	Calluses absent or infrequent
Loss of sensation, reflexes, and vibration sense	Variable sensory findings
Increase in blood flow (arteriovenous shunting)	Decrease in blood flow
Dilated veins	Collapsed veins
Dry, warm foot	Cold foot
Bone deformities	No bony deformities
Red appearance	Pale, cyanotic

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Table 10. Etiologic Classification of Foot and Leg Ulcers

- Venous obstruction and insufficiency
- Arterial etiologies
  - Larger arteries
    - Atherosclerotic lower extremity PAD
    - Thromboemboli, atheroemboli
    - Thromboangiitis obliterans
  - Microcirculatory
    - Diabetic microangiopathy
    - Vasculitis
  - Collagen vascular diseases
- Neuropathic
- Diabetes mellitus
- Infectious
  - Leprosy
  - Mycotic
- Hematologic
  - Sickle cell anemia
  - Polycythemia
  - Leukemia
  - Thalassemia
  - Thrombocytosis
- Malignancy
  - Squamous cell carcinoma
  - Kaposi's sarcoma
  - Secondary metastases
  - Lymphosarcoma, mycosis fungoides
- Miscellaneous
  - Gout
  - Pyoderma gangrenosum
  - Necrobiosis lipoidica
  - Vitamin B_{12} deficiency
- Drugs
- Artifactual or factitious

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PAD = peripheral arterial disease; PVR = pulse volume recording.

Table 11. Clinical Categories of Acute Limb Ischemia

Category	Description/Prognosis	Sensory Loss	Muscle Weakness	Arterial Doppler Signals	Venous Doppler Signals
Viable	Not immediately threatened	None	None	Audible	Audible
Threatened marginally	Salvageable if promptly treated	Minimal (toes) or none	None	(Often) inaudible	Audible
Threatened immediately	Salvageable with immediate revascularization	More than toes; associated with rest pain	Mild, moderate	(Usually) inaudible	Audible
Irreversible	Major tissue loss or permanent nerve damage	Profound, anesthetic	Profound paralysis (rigor)	Inaudible	Inaudible

Reprinted from Katzen BT. Clinical diagnosis and prognosis of acute limb ischemia. Rev Cardiovasc Med 2002;3 (Suppl 2):S2–S6 (18a).

Despite increasing short-term success rates for both endovascular and surgical revascularization procedures, the possibility of recurrence remains throughout the lifetime of the patient. Early revascularization interventions for recurrent hemodynamic compromise are preferred, because delay in detection or treatment can lead to higher morbidity and poorer outcome (19–24). Participation in a follow-up surveillance program is imperative for patients undergoing both percutaneous and surgical revascularization. There are inadequate data to permit creation of consensus-based standards to define exact time intervals for surveillance visits after each type of revascularization procedure. In the absence of evidence-based standards, the clinical timeframe has customarily been based on the judgment of the vascular specialist, by evaluating the specific level and type of revascularization procedure and taking into account specific patient characteristics (Tables 12, 13, and 14).

Recommendations have been made that follow-up of autogenous vein bypass grafts be performed with duplex ultrasonography at intervals of 1, 3, 6, 12, 18, and 24 months after surgery and then yearly thereafter (16). Prompt evaluation with invasive techniques (angiography) is then indicated when noninvasive methods suggest hemodynamically significant lesions (e.g., greater than 50% stenosis) (25,26). Some patients with failing lower extremity grafts due to stenosis documented by duplex ultrasound may proceed to have operative repair without angiography. The benefit of surveillance with duplex ultrasound is less well established for prosthetic grafts.

Table 12. Surveillance Program for Aortoiliac and Infrainguinal Transluminal Angioplasty

Patients undergoing aortoiliac and infrainguinal transluminal angioplasty for lower extremity revascularization should be entered into a surveillance program, which consists of:
- Interval history (new symptoms)
- Vascular examination of the leg with palpation of proximal and outflow vessel pulses
- Resting and, if possible, postexercise ABI recording

Surveillance programs should be performed in the immediate post-PTA period and at intervals for at least 2 years

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ABI = ankle-brachial index; PTA = percutaneous transluminal angioplasty.

Postprocedure surveillance after percutaneous or endovascular procedures is less well studied, and standards are less well established. Regular visits, with assessment of interval change in symptoms, vascular examination, and ABI measurement, is considered the standard of care. Postexercise ABI determinations may be useful in some individuals. These modalities are clearly useful for patients in whom there is evidence of recurrent narrowing at the interventional site. Similarly, distal or small-caliber endovascular sites (with or without stenting) at high risk of restenosis may merit more careful noninvasive evaluation. Whereas the role of surveillance duplex imaging of autogenous and prosthetic grafts has been evaluated (see full-text guidelines), the utility and role of duplex ultrasound and other noninvasive diagnostic modalities (magnetic resonance angiography [MRA] and computed tomographic angiography [CTA]) for such routine surveillance of endovascular sites have yet to be determined.

There is no uniformly accepted threshold for repeat angiography and intervention in the patient with evidence of recurrent stenosis. Patients who have recurrent symptoms in association with evidence of hemodynamic compromise require restudy and repeat intervention. Likewise, evidence of rapidly progressive restenosis, even in the absence of symptoms, should provide a clue that may identify individuals who might benefit from future invasive management. For grafts as well as native vessels, a stenosis of less than 50% appears to be associated with

Table 13. 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

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ABI = ankle-brachial index.

Table 14. Surveillance Program for Infrainguinal Prosthetic Grafts

Patients undergoing prosthetic femoropopliteal or femorotibial bypass for claudication or limb-threatening ischemia should be entered into a graft surveillance program that consists of:

- Interval history (new symptoms)
- Vascular examination of the leg with palpation of proximal and outflow vessel pulses
- Measurement of ABIs at rest and, if possible, after exercise testing

Surveillance programs should be performed in the immediate postoperative period and at regular intervals (timing of surveillance and efficacy have not been ideally defined) for at least 2 years

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ABI = ankle-brachial index.

favorable prognosis and patency, whereas a stenosis greater than 70% is a harbinger of poor long-term patency, and thus, reintervention may be warranted (27,28).

E. Diagnostic Methods

Patients with vascular disorders can usually be assured that an accurate anatomic diagnosis will be established with modern noninvasive vascular diagnostic techniques (e.g., ankle- and toe-brachial indices, segmental pressure measurements, pulse volume recordings, duplex ultrasound imaging, Doppler waveform analysis, and exercise testing). These tests will usually provide adequate information for creation of a therapeutic plan. When required, these physiological and anatomic data can be supplemented by use of MRA or CTA and selective use of invasive aortic and lower extremity angiographic techniques. Table 15 summarizes the evidence base that defines the benefits and limitations of each of these vascular diagnostic techniques. Table 16 summarizes typical use of noninvasive tests based on clinical presentation. For a detailed discussion of each of these techniques, see the full text of the guidelines.

1. Ankle-Brachial and Toe-Brachial Indices, Segmental Pressure Examination.

RECOMMENDATIONS

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 exertional leg symptoms, with nonhealing wounds, who are 70 years and older or who are 50 years and older with a history of smoking or diabetes.** ***(Level of Evidence: C)***
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)***

2. Pulse Volume Recording.

RECOMMENDATION

Class IIa

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

3. Continuous-Wave Doppler Ultrasound.

RECOMMENDATION

Class I

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

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

RECOMMENDATIONS

Class I

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

Table 15. Noninvasive and Invasive Vascular Diagnostic Tools: Benefits and Limitations

Diagnostic Tool	Benefits	Limitations
Ankle-brachial indices (ABIs)	• A quick and cost-effective way to establish or refute the lower extremity PAD diagnosis (see text)	• May not be accurate when systolic blood pressure cannot be abolished by inflation of an air-filled blood pressure cuff (noncompressible pedal arteries), as occurs in a small fraction of diabetic or very elderly individuals
Toe-brachial indices	• A quick and cost-effective way to establish or refute the lower extremity PAD diagnosis (see text) • Can measure digital perfusion when small-vessel arterial occlusive disease is present • Useful in individuals with noncompressible posterior tibial or dorsalis pedis arteries	• Requires small cuffs and careful technique to preserve accuracy
Segmental pressure examination	• Useful to establish or refute the PAD diagnosis (see text) • Useful to provide anatomic localization of lower extremity PAD when these data are required to create a therapeutic plan • Can provide data to predict limb survival, wound healing, and patient survival • Useful to monitor the efficacy of therapeutic interventions	• May not be accurate when systolic blood pressure cannot be measured by inflation of an air-filled blood pressure cuff owing to noncompressible pedal arteries, as occurs in a small fraction of diabetic or very elderly individuals
Pulse volume recording	• Useful to establish the diagnosis of PAD in vascular laboratories or office practice • Usefulness maintained in patients with noncompressible vessels (ABI value greater than 1.3) • Helpful in predicting the outcome in CLI and risk of amputation • Can be used to monitor limb perfusion after revascularization procedures	• Qualitative, not quantitative, measure of perfusion • May not be accurate in more distal segments • Less accurate than other noninvasive tests in providing arterial anatomic localization of PAD • May be abnormal in patients with low cardiac stroke volume
Continuous-wave Doppler ultrasound	• Useful to assess lower extremity PAD anatomy, severity, and progression • Can provide localizing information in patients with poorly compressible arteries • Can provide quantitative data after successful lower extremity revascularization	• "Pulse normalization" downstream from stenoses can diminish test sensitivity • Test specificity greater for patent superficial femoral arteries than for aortoiliac occlusive disease • Does not provide visualization of arterial anatomy • Limited accuracy in tortuous, overlapping, or densely calcified arterial segments, and insensitive for iliac arteries (in context of obesity, bowel gas, and vessel tortuosity)
Duplex ultrasound	• Can establish the lower extremity PAD diagnosis, establish anatomic localization, and define severity of focal lower extremity arterial stenoses • Useful tool to provide graft surveillance after femoral popliteal or femoral tibial or pedal surgical bypass with venous (but not prosthetic) conduit • Can be useful to select candidates for endovascular or surgical revascularization	• Accuracy is diminished in proximal aortoiliac arterial segments in some individuals (e.g., due to obesity or the presence of bowel gas) • Dense arterial calcification can limit diagnostic accuracy • Sensitivity is diminished for detecting stenoses downstream from a proximal stenosis • Diminished predictive value in surveillance of prosthetic bypass grafts
Toe-tip exercise testing, with pre-exercise and postexercise ABIs	• Useful to diagnose lower extremity PAD when resting ABI values are normal • Can be performed in the absence of a treadmill, with increased convenience and low cost	• Provides qualitative (rather than quantitative) exercise diagnostic results • Lower workload may not elicit symptoms in all individuals with claudication
Treadmill exercise testing, with and without pre-exercise and postexercise ABIs	• Helps differentiate claudication from pseudoclaudication in individuals with exertional leg symptoms • Useful to diagnose lower extremity PAD when resting ABI values are normal • Objectively documents the magnitude of symptom limitation in patients with claudication, especially when used with a standardized treadmill protocol	• Requires use of a motorized treadmill, with or without continuous electrocardiogram monitoring, as well as staff familiar with exercise testing protocols

Continued on next page

Table 15. Continued

Diagnostic Tool	Benefits	Limitations
Treadmill exercise testing, with and without pre-exercise and postexercise ABIs *(continued)*	• Demonstrates the safety of exercise and provides data to individualize exercise prescriptions in individuals with claudication before initiation of a formal program of therapeutic exercise training • Useful to measure the objective functional response to claudication therapeutic interventions	
Magnetic resonance angiography (MRA)	• Useful to assess PAD anatomy and presence of significant stenoses • Useful to select patients who are candidates for endovascular or surgical revascularization	• Tends to overestimate the degree of stenosis • May be inaccurate in arteries treated with metal stents • Cannot be used in patients with contraindications to the magnetic resonance technique (e.g., pacemakers, defibrillators, intracranial metallic stents, clips, coils, and other devices)
Computed tomographic angiography (CTA)	• Useful to assess PAD anatomy and presence of significant stenoses • Useful to select patients who are candidates for endovascular or surgical revascularization • Helpful to provide associated soft tissue diagnostic information that may be associated with PAD presentation (e.g., aneurysms, popliteal entrapment, and cystic adventitial disease) • Patients with contraindications to MRA (e.g., pacemakers or defibrillators) may be safely imaged • Metal clips, stents, and metallic prostheses do not cause significant CTA artifacts • Scan times are significantly faster than for MRA	• Single-detector computed tomography lacks accuracy for detection of stenosis • Spatial resolution lower than digital subtraction angiography • Venous opacification can obscure arterial filling • Asymmetrical opacification of the legs may obscure arterial phase in some vessels • Accuracy and effectiveness not as well determined as MRA • Treatment plans based on CTA have not been compared with those of catheter angiography • Requires iodinated contrast and ionizing radiation (although radiation exposure is less than with catheter angiography) • Because CTA requires administration of iodinated contrast, use is limited in individuals with established renal dysfunction
Contrast angiography	• Definitive method for anatomic evaluation of PAD when revascularization is planned	• Invasive evaluation is associated with risk of bleeding, infection, vascular access complications (e.g., dissection or hematoma), atheroembolization, contrast allergy, and contrast nephropathy • May provide limited visualization of tibial-pedal vessels in patients with CLI with poor inflow to the leg • Below-knee vessels may be difficult to identify by digital subtraction angiography • Multiple projections may be necessary to visualize eccentric lesions

Tools are listed in order from least to most invasive and from least to most costly.
CLI = critical limb ischemia; PAD = peripheral arterial disease.

determine functional capacity, assess nonvascular exercise limitations, and demonstrate the safety of exercise. ***(Level of Evidence: B)***

Class IIb

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

5. Duplex Ultrasound.

RECOMMENDATIONS

Class I

1. **Duplex ultrasound of the extremities is useful to diagnose anatomic location and degree of stenosis of PAD.** ***(Level of Evidence: A)***
2. **Duplex ultrasound is recommended for routine surveillance after femoral-popliteal or femoral-tibial-pedal bypass with a venous conduit. Minimum surveillance intervals are approximately 3, 6, and 12 months, and then yearly after graft placement.** ***(Level of Evidence: A)***

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Table 16. 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

Adapted from *Primary Cardiology*, 2nd ed., Braunwald E, Goldman L, eds., "Recognition and management of peripheral arterial disease," Hirsch AT, 659–71, Philadelphia, PA: WB Saunders, Copyright 2003, with permission from Elsevier (30a).

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 IIa

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

Class IIb

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

6. Computed Tomographic Angiography.

RECOMMENDATIONS

Class IIb

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

7. Magnetic Resonance Angiography.

RECOMMENDATIONS

Class I

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

Class IIb

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

8. Contrast Angiography.

RECOMMENDATIONS

Class I

1. **Contrast angiography provides detailed information about arterial anatomy and is recommended for evaluation of patients with lower extremity PAD when revascularization is contemplated.** ***(Level of Evidence: B)***
2. **A history of contrast reaction should be documented before the performance of contrast angiography and appropriate pretreatment administered before contrast is given.** ***(Level of Evidence: B)***
3. **Decisions regarding the potential utility of invasive therapeutic interventions (percutaneous or surgical) in patients with lower extremity PAD should be made with a complete anatomic assessment of the affected arterial territory, including imaging of the occlusive lesion, as well as arterial inflow and outflow with angiography or a combination of angiography and noninvasive vascular techniques.** ***(Level of Evidence: B)***
4. **Digital subtraction angiography is recommended for contrast angiographic studies because this technique allows for enhanced imaging capabilities compared with conventional unsubtracted contrast angiography.** ***(Level of Evidence: A)***
5. **Before performance of contrast angiography, a full history and complete vascular examination should be performed to optimize decisions regarding the**

access site, as well as to minimize contrast dose and catheter manipulation. *(Level of Evidence: C)*

6. **Selective or superselective catheter placement during lower extremity angiography is indicated because this can enhance imaging, reduce contrast dose, and improve sensitivity and specificity of the procedure. *(Level of Evidence: C)***
7. **The diagnostic lower extremity arteriogram should image the iliac, femoral, and tibial bifurcations in profile without vessel overlap. *(Level of Evidence: B)***
8. **When conducting a diagnostic lower extremity arteriogram in which the significance of an obstructive lesion is ambiguous, transstenotic pressure gradients and supplementary angulated views should be obtained. *(Level of Evidence: B)***
9. **Patients with baseline renal insufficiency should receive hydration before undergoing contrast angiography. *(Level of Evidence: B)***
10. **Follow-up clinical evaluation, including a physical examination and measurement of renal function, is recommended within 2 weeks after contrast angiography to detect the presence of delayed adverse effects such as atheroembolism, deterioration in renal function, or access site injury (e.g., pseudoaneurysm or arteriovenous fistula). *(Level of Evidence: C)***

Class IIa

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

F. Treatment

1. Cardiovascular Risk Reduction.

A. LIPID-LOWERING DRUGS.

RECOMMENDATIONS

Class I

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

Class IIa

1. **Treatment with a hydroxymethyl glutaryl coenzyme-A reductase inhibitor (statin) medication to achieve a target LDL cholesterol level of less than 70 mg per dl is reasonable for patients with lower extremity PAD at very high risk of ischemic events. *(Level of Evidence: B)***
2. **Treatment with a fibric acid derivative can be useful for patients with PAD and low high-density lipoprotein (HDL) cholesterol, normal LDL cholesterol, and elevated triglycerides. *(Level of Evidence: C)***

It is recommended that patients with PAD and LDL cholesterol of 100 mg per dl or greater be treated with a statin, but when risk is very high, an LDL cholesterol goal of less than 70 mg per dl is a therapeutic option (29,30). Among the factors that define very high risk in individuals with established PAD are (a) multiple major risk factors (especially diabetes), (b) severe and poorly controlled risk factors (especially continued cigarette smoking), (c) multiple risk factors of the metabolic syndrome (especially high triglycerides; i.e., greater than or equal to 200 mg per dl plus non-HDL cholesterol greater than or equal to 130 mg per dl with low HDL cholesterol [less than or equal to 40 mg per dl]), and (d) individuals with acute coronary syndromes. The efficacy of this treatment with fibric acid derivatives in patients with PAD is not known. In patients with coronary artery disease and low HDL cholesterol levels, one study found that gemfibrozil reduced the risk of nonfatal myocardial infarction or cardiovascular death by 22% (31).

B. ANTIHYPERTENSIVE DRUGS.

RECOMMENDATIONS

Class I

1. **Antihypertensive therapy should be administered to hypertensive patients with lower extremity PAD to achieve a goal of less than 140 mm Hg systolic over 90 mm Hg diastolic (nondiabetics) or less than 130 mm Hg systolic over 80 mm Hg diastolic (diabetics and individuals with chronic renal disease) to reduce the risk of MI, stroke, congestive heart failure, and cardiovascular death. *(Level of Evidence: A)***
2. **Beta-adrenergic blocking drugs are effective antihypertensive agents and are not contraindicated in patients with PAD. *(Level of Evidence: A)***

Class IIa

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

Class IIb

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

Treatment of high blood pressure is indicated to reduce the risk of cardiovascular events (32). Beta-blockers, which have been shown to reduce the risk of MI and death in

patients with coronary atherosclerosis (33), do not adversely affect walking capacity (34). Angiotensin-converting enzyme inhibitors reduce the risk of death and nonfatal cardiovascular events in patients with coronary artery disease and left ventricular dysfunction (35,36). The Heart Outcomes Prevention Evaluation (HOPE) trial found that in patients with symptomatic PAD, ramipril reduced the risk of MI, stroke, or vascular death by approximately 25%, a level of efficacy comparable to that achieved in the entire study population (37). There is currently no evidence base for the efficacy of ACE inhibitors in patients with asymptomatic PAD, and thus, the use of ACE-inhibitor medications to lower cardiovascular ischemic event rates in this population must be extrapolated from the data on symptomatic patients.

C. DIABETES THERAPIES.

RECOMMENDATIONS

Class I

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

Class IIa

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

Aggressive treatment of diabetes is known to decrease the risk for microvascular events such as nephropathy and retinopathy (38,39). Patients with lower extremity PAD and diabetes should be treated to reduce their glycosylated hemoglobin to less than 7%, per the American Diabetes Association recommendation (40). Frequent foot inspection by patients and physicians will enable early identification of foot lesions and ulcerations and facilitate prompt referral for treatment (41).

D. SMOKING CESSATION.

RECOMMENDATION

Class I

1. **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 should be offered comprehensive smoking cessation interventions, including behavior modification therapy, nicotine replacement therapy, or bupropion.** ***(Level of Evidence: B)***

Physician advice coupled with frequent follow-up achieves 1-year smoking cessation rates of approximately 5% compared with only 0.1% in those attempting to quit smoking without a physician's intervention (42). Pharmacological interventions such as nicotine replacement therapy and bupropion achieve 1-year smoking cessation rates of approximately 16% and 30%, respectively (43). Tobacco cessation interventions are particularly critical in individuals with thromboangiitis obliterans, because continued use is associated with a particularly adverse outcome (44).

E. HOMOCYSTEINE-LOWERING DRUGS.

RECOMMENDATION

Class IIb

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

F. ANTIPLATELET AND ANTITHROMBOTIC DRUGS.

RECOMMENDATIONS

Class I

1. **Antiplatelet therapy is indicated to reduce the risk of MI, stroke, or vascular death in individuals with atherosclerotic lower extremity PAD.** ***(Level of Evidence: A)***
2. **Aspirin, 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 atherosclerotic lower extremity PAD.** ***(Level of Evidence: A)***
3. **Clopidogrel (75 mg per day) is recommended as an effective alternative antiplatelet therapy to aspirin to reduce the risk of MI, stroke, or vascular death in individuals with atherosclerotic lower extremity PAD.** ***(Level of Evidence: B)***

Class III

1. **Oral anticoagulation therapy with warfarin is not indicated to reduce the risk of adverse cardiovascular ischemic events in individuals with atherosclerotic lower extremity PAD.** ***(Level of Evidence: C)***

In the Antithrombotic Trialists' Collaboration (ATC) (45), patients treated with antiplatelet therapy had a 32%

proportional reduction of cardiovascular events with 75 to 150 mg daily, 26% with 160 to 325 mg daily, and 19% with 500 to 1500 mg daily. There was a significantly smaller (13%) reduction in cardiovascular events in patients being treated with less than 75 mg of aspirin per day. In the CAPRIE trial (Clopidogrel Versus Aspirin in Patients at Risk of Ischemic Events), clopidogrel reduced the risk of MI, stroke, or vascular death by 23.8% compared with aspirin in patients with PAD (46). To date, there is no evidence to support the efficacy of combined aspirin and clopidogrel treatment versus a single antiplatelet agent in patients with lower extremity PAD. Information regarding the efficacy of oral anticoagulants (i.e., coumarin derivatives such as warfarin) in reducing adverse cardiovascular events in patients with atherosclerosis is derived primarily from studies of patients with coronary artery disease. Among patients with coronary artery disease, moderate- and high-intensity oral anticoagulation with coumarin derivatives reduces the risk of MI and death but with an increased rate of bleeding (47,48).

2. Claudication.

A. EXERCISE AND LOWER EXTREMITY PAD REHABILITATION.

RECOMMENDATIONS

Class I

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

Class IIb

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

Regular walking in a supervised claudication exercise program can be expected to result in an increase in the speed, distance, and duration walked, with decreased claudication symptoms at each workload or distance (49–55). Indeed, supervised exercise can induce increases in maximal walking ability that exceed those attained with drug therapies, which have been estimated to result in improvements in maximal walking distance of 20% to 25% with pentoxifylline and 40% to 60% with cilostazol (56,57). In a meta-analysis by Gardner and Poehlman (50), the greatest improvements in walking ability occurred when each exercise session lasted longer than 30 minutes, when sessions took place at least 3 times per week, when the exercise modality was walking to near-maximal pain, and when the program lasted 6 months or greater. The key elements of such a therapeutic claudication exercise program for patients with claudication are summarized in Table 17.

Table 17. Key Elements of a Therapeutic Claudication Exercise Training Program (Lower Extremity PAD Rehabilitation)

Primary Clinician Role
• Establish the PAD diagnosis using the ankle-brachial index 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 elicits 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.

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 © 2002 Massachusetts Medical Society. All rights reserved (62a). *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 (Ruderman N, Devlin JT, Schneider S, Kriska A. Handbook of Exercise in Diabetes. Alexandria, VA: American Diabetes Association, 2002) (62b), hypertension (ACSM's Guidelines for Exercise Testing and Prescription. In: Franklin BA, editor. Baltimore, MD: Lippincott, Williams, and Wilkins, 2000) (62c), and coronary artery disease (Guidelines for Cardiac Rehabilitation and Secondary Prevention/American Association of Cardiovascular and Pulmonary Rehabilitation. Champaign, IL: Human Kinetics, 1999) (62d).

PAD = peripheral arterial disease.

B. MEDICAL AND PHARMACOLOGICAL TREATMENT FOR CLAUDICATION.

Cilostazol.

RECOMMENDATIONS

Class I

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

Cilostazol improves maximal walking distance by 40% to 60% after 12 to 24 weeks of therapy (56–60). Cilostazol increases ABI modestly, but the hemodynamic effect cannot account for the improvement in claudication (56,57,59,61). A meta-analysis indicates that cilostazol also improves walking ability and health-related quality of life (62). Cilostazol administered at 100 mg twice daily is more effective than 50 mg twice daily (58,60). Although no trials have found a significant increase in major cardiovascular events in patients treated with cilostazol, this medication should not be used in individuals with heart failure because of its potential adverse effect in this population as a phosphodiesterase type 3 inhibitor.

Pentoxifylline.

RECOMMENDATIONS

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)***

Meta-analyses of randomized, placebo-controlled, double-blind clinical trials found that pentoxifylline causes a marginal but statistically significant improvement in pain-free and maximal walking distance (63,64); however, pentoxifylline does not increase the ABI at rest or after exercise (64). Pentoxifylline may be considered to treat patients with intermittent claudication; however, the anticipated outcome is likely to be of marginal clinical importance.

Other Proposed Medical Therapies.

RECOMMENDATIONS

Class IIb

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

Class III

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

C. ENDOVASCULAR TREATMENT FOR CLAUDICATION.

RECOMMENDATIONS

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 20 and 21 and Figure 8 of the full-text guidelines) iliac and femoropopliteal arterial lesions.** ***(Level of Evidence: B)***
3. **Translesional pressure gradients (with and without vasodilation) should be obtained to evaluate the significance of angiographic iliac arterial stenoses of 50% to 75% diameter before intervention.** ***(Level of Evidence: C)***

4. **Provisional stent placement is indicated for use in the iliac arteries as salvage therapy for a suboptimal or failed result from balloon dilation (e.g., persistent translesional gradient, residual diameter stenosis greater than 50%, or flow-limiting dissection).** ***(Level of Evidence: B)***
5. **Stenting is effective as primary therapy for common iliac artery stenosis and occlusions.** ***(Level of Evidence: B)***
6. **Stenting is effective as primary therapy in external iliac artery stenoses and occlusions.** ***(Level of Evidence: C)***

Class IIa

1. **Stents (and other adjunctive techniques such as lasers, cutting balloons, atherectomy devices, and thermal devices) can be useful in the femoral, popliteal, and tibial arteries as salvage therapy for a suboptimal or failed result from balloon dilation (e.g., persistent translesional gradient, residual diameter stenosis 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)***

Outcomes of percutaneous transluminal angioplasty (PTA) and stents to improve results for individuals with claudication depend on anatomic and clinical factors (Table 18). Durability of patency after PTA is greatest for lesions in the common iliac artery and decreases distally and with increasing length of the stenosis/occlusion, multiple and diffuse lesions, poor-quality runoff, diabetes, renal failure, smoking, and CLI (65–80). Overall outcomes of PTA and stenting of native vessels are summarized in Table 21 in the full-text guidelines. Percutaneous transluminal angioplasty of vein bypass graft stenoses has also been reported, with 1- to 3-year patency rates of the treated site of approximately 60% (81–83), comparable to surgical repair (81). Percutaneous transluminal angioplasty of multiple vein graft stenoses has a much lower 3-year patency rate of only 6% (82). Therefore, patient selection is key in obtaining satisfactory outcomes. For a full discussion of patient and lesion selection, see the full-text guidelines.

D. SURGERY FOR CLAUDICATION.

INDICATIONS.

RECOMMENDATIONS

Class I

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

Class IIb

1. **Because the presence of more aggressive atherosclerotic occlusive disease is associated with less durable results in patients younger than 50 years of age, the effectiveness of surgical intervention in this population for intermittent claudication is unclear.** ***(Level of Evidence: B)***

Class III

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

Because claudication usually does not progress to limb-threatening ischemia, there is no automatic mandate to proceed to surgical intervention. Surgical intervention is usually reserved for individuals (a) who do not derive adequate functional benefit from nonsurgical therapies, (b) who have limb arterial anatomy that is favorable to obtaining a durable clinical result, and (c) in whom the cardiovascular risk of surgical revascularization is low. The 2 traditional functional indications for surgical intervention are exercise impairment sufficient to threaten the patient's employment or to require significant alterations in the patient's lifestyle after failure of nonsurgical or endovascular therapy. Patients who present with symptoms of claudication before 50 years of age may have a more virulent form of atherosclerosis and have a poorer response to vascular surgical interventions, frequently requiring graft revisions or replacements (87,88). Surgery for these younger patients should be avoided if possible.

Table 18. Overview of Primary Patency and Limb Salvage Rates After Endovascular Procedures for Peripheral Arterial Disease of the Lower Extremities

Procedure	Lesion Type	Severity of Disease	Reference	No. of Limbs	% 30-Day Mortality (95% CI)	% Major Complication (95% CI)	% Technical Success (95% CI)	% Primary Patency (95% CI)*				
								1 yr	2 yrs	3 yrs	4 yrs	5 yrs
Iliac PTA	80% Stenoses, 20% occlusion	67% Claudication, 33% critical ischemia	(84)	1473	1.0 (0–2.9)	4.3 (2.0–6.5)	91 (86–96)	74 (71–76)	66 (63–68)	61 (59–64)	58 (56–61)	—
	Stenoses	Claudication			ND	ND	96	79	72	68	65	—
	Stenoses	Critical ischemia			ND	ND	ND	72	61	56	53	—
	Occlusion	Claudication			ND	ND	80	66	60	57	54	—
	Occlusion	Critical ischemia			ND	ND	ND	60	51	47	44	—
Iliac stent	72% Stenoses, 28% occlusion	85% Claudication, 15% critical ischemia	(84)	901	0.8 (0.7–0.9)	5.2 (3.5–6.9)	96 (91–100)	86 (84–89)	79 (76–81)	75 (72–78)	74 (69–78)	—
	Stenoses	Claudication			ND	ND	100	91	84	80	77	—
	Stenoses	Critical ischemia			ND	ND	ND	87	76	70	67	—
	Occlusion	Claudication			ND	ND	80	72	67	64	61	—
	Occlusion	Critical ischemia			ND	ND	ND	69	60	56	53	—
Femoropopliteal PTA	64% Stenoses, 36% occlusion	65% Claudication, 35% critical ischemia	(85,86)	4800/1003†	0.9 (0.7–1.1)	8.1 (7.3–8.9)	89 (87–91)	59 (56–62)	54 (51–57)	52 (48–55)	49 (45–52)	45 (4–49)
	Stenoses	Claudication			ND	ND	95	79	75	74	71	68
	Stenoses	Critical ischemia			ND	ND	90	62	57	54	51	47
	Occlusion	Claudication			ND	ND	87	52	46	43	40	35
	Occlusion	Critical ischemia			ND	ND	75	26	21	18	15	12
Femoropopliteal stent	Stenoses and occlusions	80% Claudication, 20% critical ischemia	Meta-analysis‡	600	—	5.9 (1.7–10)	98 (97–100)	62 (48–80)	52 (33–83)	43 (22–86)	—	—
Infrapopliteal PTA	Stenoses and occlusions	14% Claudication, 86% critical ischemia	Meta-analysis§	1282	—	—	93 (90–96)	79 (68–90)	74 (65–83)	—	—	—

*All patency rates and limb savage rates include initial technical failures. †Mortality and complication rates are based on n equals 4800, patency rates are based on n equals 1003. ‡Based on a random-effects meta-analysis of the results from various sources, each weighted with the inverse of the variance (17–27). §Based on a random-effects meta-regression analysis of the results from various sources, each weighted with the inverse of the variance (28–46). Reprinted from Kandarpa et al. J Vasc Interv Radiol 2001;12:683–95 (84a).

CI = confidence interval; ND = no difference by subgroup can be demonstrated; PTA = percutaneous transluminal angioplasty.

Preoperative Evaluation.

RECOMMENDATION

Class I

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

Lower extremity PAD is associated with the presence of coronary artery disease and marks high short-term and long-term coronary ischemic risk, and therefore, a preoperative cardiovascular risk evaluation should be undertaken. The specific testing strategy that might be used for a specific patient is beyond the scope of this guideline. Perioperative ischemic risk is increased for all lower extremity vascular surgical procedures (inclusive of aortic, femoral, and infrapopliteal segments). This risk is further increased in those patients with an established history of ischemic heart disease, current angina, or an abnormal electrocardiogram and may be challenging to assess in those individuals in whom a sedentary lifestyle limits assessment of functional capacity. The preoperative cardiovascular risk evaluation is summarized in more detail in the ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery (89).

Correlation of Symptoms and Lesions.

Surgical Procedures. For individuals with claudication, initial revascularization strategies will usually rely on endovascular techniques, with surgical intervention reserved for individuals in whom arterial anatomy is not favorable for endovascular procedures. As noted in Section 2.6.2.4 of the full-text guidelines, comparable efficacy can often be achieved, with less risk imposed by endovascular intervention when both procedures are feasible (90–92). Once the decision to proceed with surgical intervention has been made and the site and severity of occlusive lesions have been defined through imaging studies, the type of revascularization must be chosen. In patients with combined inflow and outflow disease, inflow problems are corrected first. A significant improvement in inflow may diminish the symptoms of claudication to the extent that supervised exercise therapy or pharmacotherapies may be effective and, if distal revascularization is needed, reduce the likelihood of distal graft thrombosis from low flow.

Inflow Procedures: Aortoiliac Occlusive Disease.

RECOMMENDATIONS

Class I

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

Class IIb

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

Class III

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

There are numerous patterns of aortoiliac occlusive disease and procedures to surgically treat them (Table 19). Most commonly, patients demonstrate diffuse disease of the infrarenal aorta and iliac vessels, with the lesions of greatest hemodynamic consequence located in the iliac arteries. The most effective surgical procedure for the treatment for this pattern of atherosclerotic occlusive disease and the resultant buttock and thigh claudication is aortobifemoral bypass.

If the aortoiliac lesions are confined to the area of the aortic bifurcation, localized aortoiliac endarterectomy may be considered. A less invasive approach may be appropriate for patients with adequate aortic flow but stenoses or occlusions of both iliac vessels. Such patients may not be considered acceptable candidates for aortobifemoral bypass

Table 19. Vascular Surgical Procedures for Inflow Improvement

Inflow Procedure	Operative Mortality (%)	Expected Patency Rates (%)	References
Aortobifemoral bypass	3.3	87.5 (5 yrs)	(93)
Aortoiliac or aortofemoral bypass	1–2	85–90 (5 yrs)	(94–96)
Iliac endarterectomy	0	79–90 (5 yrs)	(97–99)
Femorofemoral bypass	6	71 (5 yrs)	(100)
Axillofemoral bypass	6	49–80 (3 yrs)	(101,102)
Axillofemoral-femoral bypass	4.9	63–67.7 (5 yrs)	(103,104)

because of comorbid cardiovascular disease. If endovascular treatment of 1 iliac artery is feasible and can achieve patency with this less invasive approach, a subsequent endarterectomy, unilateral iliofemoral bypass, or a femoral-femoral bypass can be constructed. In the absence of an inflow stenosis within the donor iliac arterial segment, this procedure can effectively provide flow to both lower extremities and eliminate the symptoms of claudication. Patients with severe infrarenal aortic atherosclerosis who are at high cardiovascular or surgical risk for open aortobifemoral bypass may be treated with axillofemoral-femoral bypass. Because of lower patency rates, such bypasses are reserved for those who have no alternatives for revascularization. Unilateral iliac stenoses or occlusions that cannot be effectively treated by angioplasty and stent placement can be treated by iliac artery endarterectomy, aortoiliac bypass, aortofemoral bypass, or iliofemoral bypass if the origin of the iliac artery is free of disease.

Outflow Procedures: Infrainguinal Disease.

RECOMMENDATIONS

Class I

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

Class IIa

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

Class IIb

1. **Femoral-tibial artery bypasses constructed with autogenous vein may be considered for the treatment of claudication in rare instances for certain patients (see text).** ***(Level of Evidence: B)***
2. **Because their use is associated with reduced patency rates, the effectiveness of the use of synthetic grafts to the popliteal artery above the knee is not well established.** ***(Level of Evidence: B)***

Class III

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

The most commonly performed infrainguinal bypass for the treatment of claudication is the femoral-popliteal artery bypass (Table 20). There are, however, specific factors that may modify the result of this procedure. The 2 major factors are the type of conduit and the site of anastomosis to the popliteal artery, whether above or below the knee.

Nearly all studies that have compared vein with prosthetic conduit for arterial reconstruction of the lower extremity have demonstrated the superior patency of vein. Four large, randomized, prospective studies summarized in Table 21 demonstrate findings consistent with the large body of evidence on the choice of graft material for the construction of bypasses to the above-knee popliteal artery (115–118). The superior rates of immediate and long-term patency at all time periods favor use of autogenous vein, whether in situ or reversed. In its absence, polytetrafluoroethylene or polyester fiber may be used with an expected lower but acceptable patency rate. The need for retreatment or revision is greater with synthetic material over time. With more distal anastomoses or the presence of hemodynamically significant tibial arterial occlusive disease and poor outflow, there is accelerated failure of prosthetic grafts. Therefore, the use of autogenous vein is also strongly favored for bypasses to the popliteal artery below the knee. Femoral tibial bypass grafting with autogenous vein should rarely be necessary for the treatment of intermittent claudication because of the increased risk of amputation associated with failure of

Table 20. Vascular Surgical Procedures for Outflow Improvement

Outflow Procedure	Operative Mortality (%)	Expected Patency Rate (%)	References
Fem-AK popliteal vein	1.3–6.3	66 (5 yrs)	(85,105,106)
Fem-AK popliteal prosthetic	1.3–6.3	50 (5 yrs)	(115–118)
Fem-BK popliteal vein	1.3–6.3	66 (5 yrs)	(105,106)
Fem-BK popliteal prosthetic	1.3–6.3	33 (5 yrs)	(85,105,106)
Fem-Tib vein	1.3–6.3	74–80 (5 yrs)	(107)
Fem-Tib prosthetic	1.3–6.3	25 (3 yrs)	(109)
Composite sequential bypass	0–4	28–40 (5 yrs)	(110,111)
Fem-Tib blind segment bypass	2.7–3.2	64–67 (2 yrs)	(112)
Profundaplasty	0–3	49–50 (3 yrs)	(113,114)

AK = above the knee; BK = below the knee; Fem = femoral; Tib = tibial.

Table 21. Patency of Bypass Grafts to the Above-Knee Popliteal Artery

				% Patency (yrs)				
First Author	Reference	Graft Material	n	2	5	Assisted 5	6	Assisted 6
Johnson	(117)	SVG	226	81	73			
		PTFE	265	69	39			
Klinkert	(118)	SVG	75		75.6	79.7		
		PTFE	76		51.9	57.2		
AbuRahma	(116)	SVG	43				76	83
		PTFE	43				68	68
Green	(115)	PTFE			43		68	
		Dacron		45		68		

n = number of patients; PTFE = polytetrafluoroethylene; SVG = saphenous vein graft.

such grafts (119,120). Bypasses to the tibial arteries with prosthetic material should be avoided at all costs for the treatment of the claudicant because of very high risks of graft failure and amputation.

Follow-Up After Vascular Surgical Procedures.

RECOMMENDATIONS

Class I

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

3. Critical Limb Ischemia and Treatment for Limb Salvage.

A. MEDICAL AND PHARMACOLOGICAL TREATMENT FOR CLI.

RECOMMENDATIONS

Class III

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

Prostaglandins.

RECOMMENDATIONS

Class IIb

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

Class III

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

In the 8 short-term trials of parenteral administration of PGE-1 or prostacyclin in patients with CLI, the results have been inconsistent and for the most part have not demonstrated efficacy, as defined by amelioration of pain or healing of ulcers (16,121–127). In addition, there have been at least 11 randomized, placebo-controlled trials of intravenous PGE-1 or iloprost (16). The majority of studies have found that parenteral administration of either PGE-1 or iloprost reduced pain, as assessed by analgesic consumption, ulcer size, and/or amputation (16,128–136). One study evaluated the efficacy of oral iloprost in patients with CLI. Iloprost did not significantly affect the primary end point of amputation or death at 1 year (137).

Angiogenic Growth Factors.

RECOMMENDATION

Class IIb

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

B. ENDOVASCULAR TREATMENTS FOR CLI.

RECOMMENDATIONS

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)***

The optimal strategy for management of a patient with CLI must be determined on a case-by-case basis. Important issues to consider include the urgency of the clinical presentation, the presence of comorbidity, and the arterial anatomy. A significant improvement in inflow may diminish the symptoms of rest pain, but pulsatile flow to the foot is generally necessary for the treatment of ischemic ulcers or ischemic gangrene. Therefore, if infection, ischemic ulcers, or gangrenous lesions persist and the ABI is less than 0.8 after correction of inflow, an outflow procedure should be performed that bypasses all major distal stenoses and occlusions (138). The angiographic evaluation may also suggest the presence of arterial stenoses whose functional significance may not be clear. In this situation, measurement of trans-stenotic pressure gradients can guide therapy. However, in the presence of severe outflow disease, an inaccurately low pressure gradient may exist. Severe outflow disease may so limit arterial flow that gradients are not developed, and in this context, use of a pharmacological arterial vasodilator to augment flow may be useful.

C. THROMBOLYSIS FOR ACUTE AND CHRONIC LIMB ISCHEMIA.

RECOMMENDATIONS

Class I

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

Class IIa

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

Class IIb

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

Randomized controlled trials and registry reports indicate that the use of thrombolytic therapy for acute limb ischemia is effective as initial therapy (139–142). (The terms "thrombolysis" and "thrombolytic" are synonymous with the terms "fibrinolysis" and "fibrinolytic," as used in other ACC/AHA guidelines.) These randomized trials and case series suggest that the use of intra-arterial thrombolytic therapy for acute limb ischemia is reasonably effective and comparable to surgery. The advantage of thrombolytic therapy is that it offers a low-risk alternative to open surgery in complex patients with severe comorbidities. Other advantages of pursuing immediate angiography in patients with acute limb ischemia include delineation of the limb arterial anatomy with visualization of both inflow and runoff vessels. Finally, thrombolytic therapy has the advantage, compared with surgical embolectomy, of clearing intra-arterial thrombus from the distal runoff vessels, thereby potentially enhancing long-term patency. The choice of thrombolytic versus surgical revascularization depends on several factors (16). Patients with profound limb ischemia may not tolerate the time necessary to perform thrombolysis. Infrainguinal or distal arterial thrombolysis has worse outcomes than more proximal or iliofemoral lysis (141). Because of bleeding risks, thrombolysis may be contraindicated in some patients. Contraindications have been summarized, although these recommendations are based on common practice and are not necessarily supported by published studies. Because of comorbidities, surgery may be contraindicated in some patients. Nonrandomized trials and small series have also reported on the use of mechanical thrombectomy devices, which may avert the need for thrombolysis or permit the use of decreased doses of thrombolytic drugs (Table 22).

D. SURGERY FOR CLI.

RECOMMENDATIONS

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)***

Table 22. Mechanical Thrombectomy Devices for the Treatment of Peripheral Arterial Occlusions

Device and First Author (Reference)	Year	n	Conduit n (%)	Duration n	MTD Success,* n (%)	Adjunctive Procedures	Primary Patency (%)	Complications (%)
Oasis								
Hoptner (143)	1999	51	Native: 44 (86) Grafts: 7 (14)	All acute	6 (11.8)	Lysis: 5 PTA: 20 PAT: 15 SA: 3	1 month: 64 6 months: 54	Hemorrhage: 8 Emboli: 4.8 Acute occlusion: 3 Amputation: 17.7 Mortality: 8
AngioJet								
Muller-Hulsbeck (144)	2000	112	Native: 99 (86) Grafts: 16 (14)	All acute	79 (71)	Lysis: 20 PTA: 68 PAT: 11	6 months: 68 2 years: 60 3 years: 58	Embolization: 9.8 Dissection: 8 Perforation: 3.6 Amputation: 1.8 Mortality: 7
Kasirajan (145)	2001	83	Native: 52 (63) Grafts: 31 (37)	Acute: 62 Chronic: 21	Complete: 51 (61) Partial: 19 (23)	Lysis: 50 PTA: 47	3 months: 90 6 months: 78	Hemorrhage: 10.5 Emboli: 2.3 Dissection: 3.5 Perforation: 2.3 Amputation: 11.6 Mortality: 9.3
Silva (146)	1998	22	Native: 13 (59) Grafts: 9 (41)	All acute	21 (95)	PTA: 21	NA	Hemorrhage: 10 Embolism: 9 Dissection: 5 Occlusion: 18 Amputation: 5 Mortality: 14
Wagner (147)	1997	50	Native: 39 (78) Grafts: 11 (22)	All acute	26 (52)	Lysis: 15 PTA: 34 PAT: 9	1 year: 69	Hemorrhage: 6 Emboli: 6 Dissection: 6 Perforation: 6 Amputation: 8 Mortality: 0
Hydrolyser								
Reekers (148)	1996	28	Native: 11 (39) Grafts: 17 (61)	Acute: 23 Chronic: 5	23 (82)	Lysis: 11 PTA: 20 PAT: 2	1 month: 50	Embolization: 18 Hemorrhage: 0 Acute occlusion: 10 Amputation: 11 Mortality: 0
Henry (149)	1998	41	Native: 28 (68) Grafts: 8 (20) Other: 5	All acute	34 (83)	Lysis: 10 PTA: 29 PAT: 17	1 month: 73	Acute occlusion: 12 Emboli: 2.4 Amputation: 0 Mortality: 0 Mortality: 0
Amplatz								
Rilinger (150)	1997	40	All native	All acute	30 (75)	Lysis/PTA/SA: 9	NA	Hemorrhage: 2.5 Device failure: 7.5 Emboli: 0 Amputation: 5 Mortality: 0
Tadavarthy (151)	1994	14	Native: 2 (14) Grafts: 10 (71) Other: 2	Acute: 9 Chronic: 5	10 (71)	Lysis: 4 PTA/SA: 11	6 months: 43	Hemorrhage: 14.3 Emboli: 14 Device failure: 7 Amputation: 0 Mortality: 0
Gorich (152)	1998	18	All native	All acute	14 (78)	Lysis: 12 PAT: 9	NA	Hemorrhage: 6 Device failure: 6 Amputation: 6

*Definition of success varies among studies. Reprinted from Haskal ZJ. Mechanical thrombectomy devices for the treatment of peripheral arterial occlusions. Rev Cardiovasc Med 2002;3 Suppl 2:S45–S52 (152a).

MTD = mechanical thrombectomy device; n = number of patients; NA = not applicable; PAT = percutaneous aspiration thrombectomy; PTA = percutaneous transluminal angioplasty; SA = Simpson atherectomy.

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)***

The goal of surgical intervention in patients with CLI is the elimination of clinical manifestations of severe lower extremity PAD, whether rest pain, ischemic ulcers, or distal ischemic gangrene. A significant improvement in inflow may diminish the symptoms of rest pain, but pulsatile flow to the foot is generally necessary for the treatment of ischemic ulcers or ischemic gangrene. Therefore, if infection, ischemic ulcers, or gangrenous lesions persist and the ABI is less than 0.8 after correction of inflow, an outflow procedure should be performed that bypasses all major distal stenoses and occlusions (138). Surgery for the treatment of severe lower extremity ischemia (as for endovascular treatment) must be based on specific goals, such as the relief of rest pain or healing of ulcers, prior revascularization attempts, the type of procedure required to accomplish the goals, and the patient's overall ability to successfully recover from the effort. In patients with combined inflow and outflow disease, inflow problems must be corrected first.

Inflow Procedures: Aortoiliac Occlusive Disease.

RECOMMENDATIONS

Class I

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

The creation of an in situ or reversed greater saphenous vein bypass to a tibial vessel is the most commonly performed limb salvage procedure. This type of bypass can be performed under general or regional (or, more rarely, local) anesthesia and is generally well tolerated. There are, however, specific factors that may modify the result of this procedure, most notably, the type of conduit and the outflow tract beyond the distal anastomosis.

Nearly all studies that have compared vein to prosthetic conduit for arterial reconstruction of the lower extremity have demonstrated the superior patency of vein (115–118). In its absence, polytetrafluoroethylene or Dacron (polyester fiber) may be used with an expected lower but acceptable patency rate for above-knee bypasses. Patency of prosthetic grafts is significantly lower once the knee joint is crossed (85). When vein length is inadequate, a composite sequential graft that consists of a prosthetic graft to the above-knee popliteal artery and a jump graft of autogenous vein to the distal vessel may be used. If no other option exists, the use of a prosthetic with an adjunctive procedure, such as arteriovenous fistula or vein interposition or cuff, may improve patency, although this has not been proven. The least-diseased tibial or pedal artery with continuous flow to the foot should be used as the outflow vessel for the construction of a distal bypass, because equivalent results can be achieved with all tibial and even pedal arteries (153,154).

Outflow Procedures: Infrainguinal Disease.

RECOMMENDATIONS

Class I

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

Class IIa

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

There are numerous patterns of aortoiliac occlusive disease and procedures to surgically treat them. Most commonly, patients demonstrate diffuse disease of the infrarenal aorta and iliac vessels, with the lesions of greatest hemodynamic consequence located in the iliac arteries. The most effective surgical procedure for the treatment for this pattern of atherosclerotic occlusive disease is aortobifemoral bypass. Aortobifemoral grafting is associated with an operative mortality of 3.3% and a morbidity of 8.3% (93). Major morbidity is most commonly due to MI (0.8% to 5.2%) or renal failure (0% to 4.6%) (155). The expected patency of aortobifemoral bypass as the sole procedure for the treatment of CLI is excellent (93,155).

If the aortoiliac lesions are confined to the area of the aortic bifurcation, localized aortoiliac endarterectomy may be considered. This procedure is effective but is uncommonly performed because few patients have such a limited manifestation of atherosclerosis. Nonetheless, when the operation is indicated, the results demonstrate good patency, in the range of 48% to 77% at 10 years (156).

For patients with adequate aortic flow but stenoses or occlusions of both iliac vessels who are not considered acceptable candidates for aortobifemoral bypass, a somewhat less invasive approach may be appropriate. If 1 iliac artery can be made widely patent by angioplasty and stent placement, endarterectomy, or a unilateral iliofemoral bypass, a femoral-femoral bypass can be constructed. In the absence of an inflow stenosis within the donor iliac arterial segment, this procedure can effectively improve flow to both lower extremities. Unilateral iliac stenoses or occlusions that cannot be treated effectively by angioplasty and stent placement can be treated by iliac artery endarterectomy, aortoiliac bypass, aortofemoral bypass, or iliofemoral bypass if the origin of the iliac artery is free of disease.

The surgical treatment of unilateral iliac disease by aortoiliac, iliofemoral, or femoral-femoral bypass graft placement provides excellent results for the restoration of inflow into the lower extremity. Ipsilateral bypasses that originate from the aorta or proximal iliac artery have a 3-year patency rate in the range of 90% (95,157). Femoral-femoral bypass grafting yields a 3-year patency rate that ranges from 60% to 80% and a 5-year patency rate of 60% to 90% (158,159).

Patients with severe infrarenal aortic atherosclerosis who are at high cardiovascular or surgical risk for open aortobifemoral bypass may be treated with axillofemoral-femoral bypass. Because this graft is based on the axillary artery, preoperative assessment of bilateral arm blood pressures, duplex ultrasound flow assessments, and/or imaging of the aortic arch and great vessels to the origin of the donor vessel should be obtained.

Axillofemoral and axillobifemoral grafts are significantly inferior to aortobifemoral bypass grafts or aortoiliac endarterectomy for the treatment of severe diffuse aortoiliac disease. The 5-year patency rate for axillofemoral grafts ranges from 19% to 50% (160,161). The results of axillobifemoral bypass are somewhat better, with 5-year patency rates that range from 50% to 76% (162).

Postsurgical Care.

RECOMMENDATIONS

Class I

1. **Unless contraindicated, all patients undergoing revascularization for CLI should be placed on antiplatelet therapy (see Sections 2.4.2 and 2.6.1.6 in the full-text guidelines), and this treatment should be continued indefinitely.** ***(Level of Evidence: A)***
2. **Patients who have undergone placement of aortobifemoral bypass grafts should be followed up with periodic evaluations that record any return or progression of ischemic symptoms, the presence of femoral pulses, and ABIs.** ***(Level of Evidence: B)***
3. **If infection, ischemic ulcers, or gangrenous lesions persist and the ABI is less than 0.8 after correction of inflow, an outflow procedure should be performed that bypasses all major distal stenoses and occlusions.** ***(Level of Evidence: A)***
4. **Patients who have undergone placement of a lower extremity bypass with autogenous vein should undergo for at least 2 years periodic examinations that record any return or progression of ischemic symptoms; a physical examination, with concentration on pulse examination of the proximal, graft, and outflow vessels; and duplex imaging of the entire length of the graft, with measurement of peak systolic velocities and calculation of velocity ratios across all lesions.** ***(Level of Evidence: A)***
5. **Patients who have undergone placement of a synthetic lower extremity bypass graft should undergo periodic examinations that record any return of ischemic symptoms; a pulse examination of the proximal, graft, and outflow vessels; and assessment of ABIs at rest and after exercise for at least 2 years after implantation.** ***(Level of Evidence: A)***

To maximize the benefit of revascularization and to minimize the risk of cardiovascular ischemic events (MI and stroke), all postoperative patients with lower extremity PAD should receive cardiovascular risk-reduction therapy and an oral antiplatelet medication, usually aspirin or clopidogrel.

Optimally, risk-reduction therapies will be initiated preoperatively and continued for the patient's lifetime. There are minimal data to suggest that anticoagulation with warfarin

may prolong graft patency; most case series include small numbers of patients, and thus the overall database is inconclusive as yet (163,164). One retrospective analysis of patients who had undergone infrainguinal bypass suggested that the use of an ACE inhibitor might decrease mortality (165). To maintain optimal outcomes, patients should undergo periodic graft surveillance for at least 2 years after placement. For vein grafts, duplex imaging of the donor and recipient arteries, proximal and distal anastomoses, and the entire graft length is of benefit for the detection of grafts with reduced flow secondary to intraluminal lesions. Duplex imaging is of limited benefit for the detection of lesions within synthetic grafts. Therefore, the periodic recording of ABIs is sufficient.

G. Algorithms

1. Diagnostic and Treatment Pathways. The diagnosis of lower extremity PAD should be considered in individuals who are either "at risk" for lower extremity PAD and in those who present with lower extremity ischemic symptoms (Fig. 3). Specific clinical information should be used to identify these at-risk individuals who merit pursuit of an objective lower extremity PAD diagnosis by measurement of an ABI examination or by use of alternative PAD testing strategies (Figs. 3 to 5). Clinical data that should guide this assessment include the presence of atherosclerosis risk factors (especially age, smoking, and diabetes), clinical history (a history of atherosclerotic coronary artery, carotid artery, or renal artery disease and lower extremity symptoms), and an abnormal lower extremity pulse examination. Subsequent diagnostic testing and therapeutic interventions are dependent on the severity and acuity of the presenting limb symptoms (Figs. 4 to 8). Use of lower extremity symptoms and ABI data should then be used to initiate therapeutic interventions to decrease cardiovascular ischemic risk; diminish claudication symptoms; and promptly identify individuals with CLI or those who are at risk for amputation. Algorithms for the diagnosis and treatment of PAD are presented for individuals with PAD who are asymptomatic or who present with atypical symptoms (Fig. 4), claudication (Figs. 5 and 6), CLI (Fig. 7), and acute limb ischemia (Figs. 8 and 9).

III. RENAL ARTERIAL DISEASE

A. Prevalence and Natural History

Renal artery stenosis (RAS) is both a common and progressive disease in patients with atherosclerosis and a relatively uncommon cause of hypertension (166,167). From a limited epidemiological database, it is estimated that atherosclerotic RAS may affect as many as 6.8% of people aged 65 years and older (168). However, atherosclerotic RAS is common in cohorts that have clinically evident atherosclerosis in other arterial circulations. For example, 22% to 59% of patients with PAD have hemodynamically significant RAS (as defined by a stenosis greater than 50%) (169–179). In individuals with histories of proven MI, 12% of postmortem examinations demonstrate the presence of an RAS of 75% or greater. Despite the high prevalence of RAS in these atherosclerotic subgroups, it remains controversial as to which lesions are associated with important clinical sequelae.

B. Clinical Clues to the Diagnosis of RAS

RECOMMENDATIONS

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)***
2. **The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with the onset of severe hypertension [as defined in the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report (187)] after the age of 55 years.** ***(Level of Evidence: B)***
3. **The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with the following characteristics: (a) accelerated hypertension (sudden and persistent worsening of previously controlled hypertension); (b) resistant hypertension (defined as the failure to achieve goal blood pressure in patients who are adhering to full doses of an appropriate 3-drug regimen that includes a diuretic); or (c) malignant hypertension (hypertension with coexistent evidence of acute end-organ damage; i.e., acute renal failure, acutely decompensated congestive heart failure, new visual or neurological disturbance, and/or advanced [grade III to IV] retinopathy).** ***(Level of Evidence: C)***
4. **The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with new azotemia or worsening renal function after the administration of an ACE inhibitor or an angiotensin receptor blocking agent.** ***(Level of Evidence: B)***
5. **The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with an unexplained atrophic kidney or a discrepancy in size between the 2 kidneys of greater than 1.5 cm.** ***(Level of Evidence: B)***
6. **The performance of diagnostic studies to identify clinically significant RAS is indicated in patients with sudden, unexplained pulmonary edema (especially in azotemic patients).** ***(Level of Evidence: B)***

Class IIa

1. **The performance of diagnostic studies to identify clinically significant RAS is reasonable in patients with unexplained renal failure, including individuals**

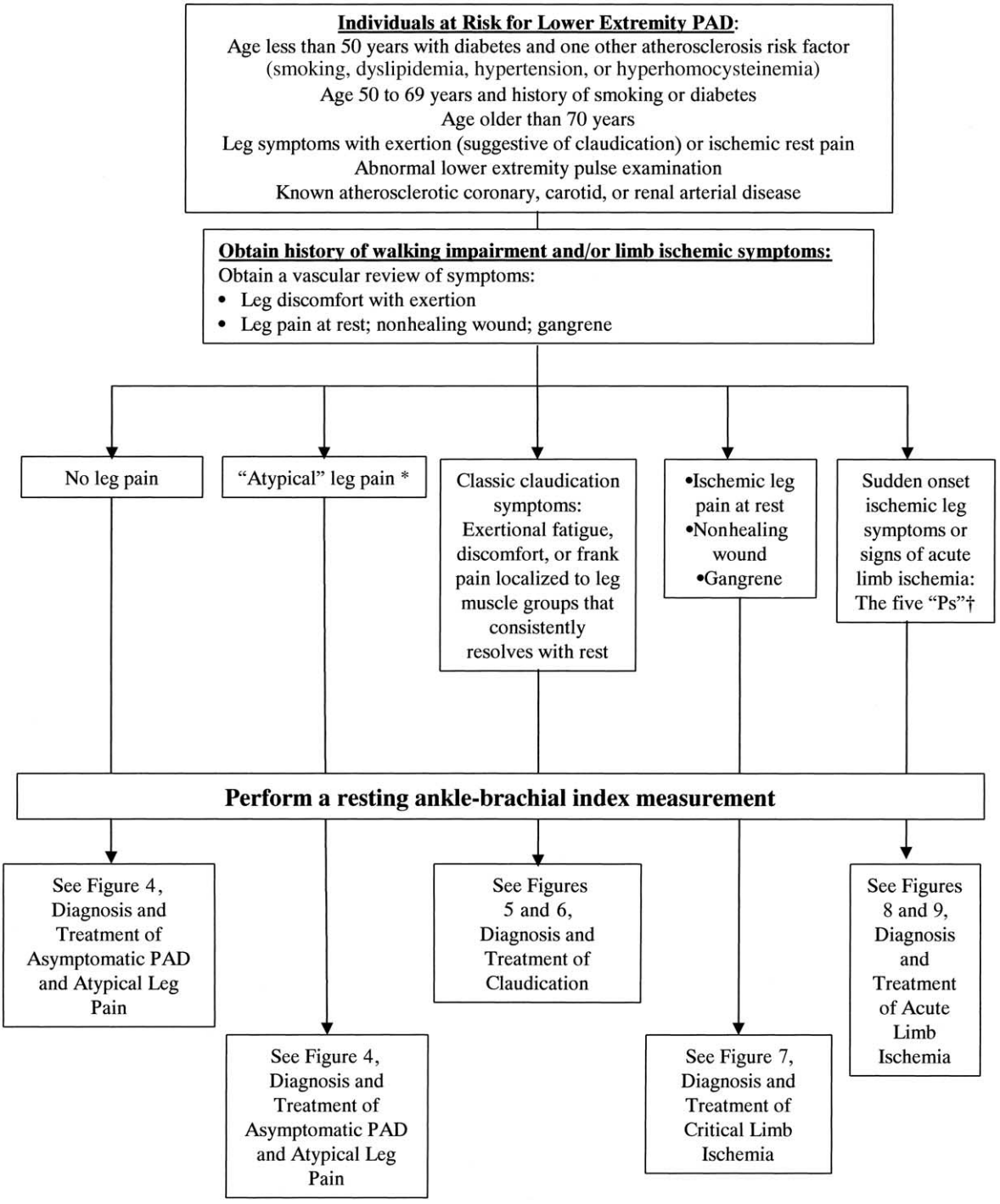

Figure 3. Steps toward the diagnosis of peripheral arterial disease (PAD). *"Atypical" leg pain is defined by lower extremity discomfort that is exertional, but that does not consistently resolve with rest, consistently limit exercise at a reproducible distance, or meet all "Rose questionnaire" criteria. †The five "Ps" are defined by the clinical symptoms and signs that suggest potential limb jeopardy: pain, pulselessness, pallor, paresthesias, and paralysis (with polar being a sixth "P").

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 (Fig. 10) 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)***

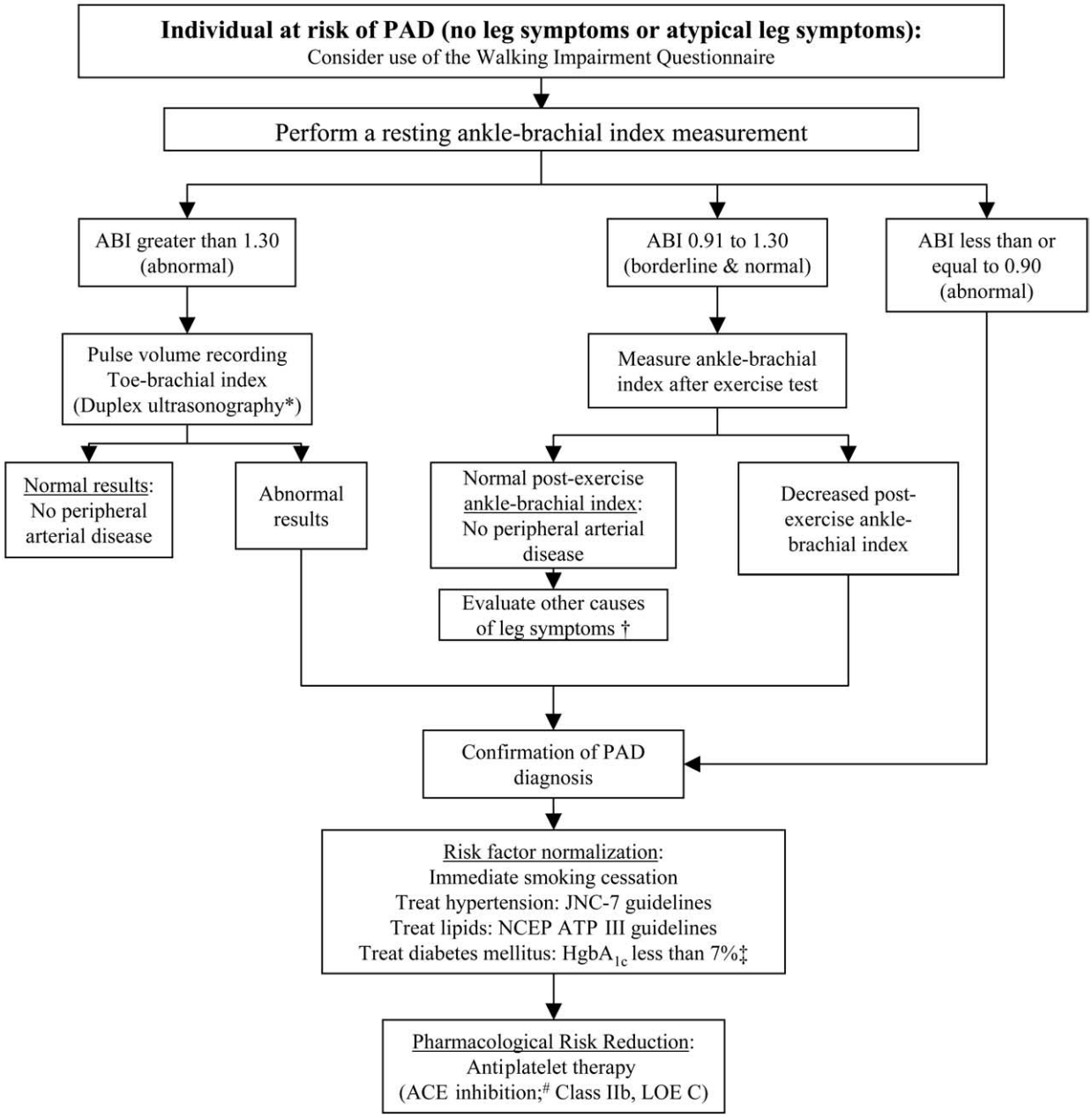

Figure 4. Diagnosis and treatment of asymptomatic peripheral arterial disease (PAD) and atypical leg pain. *Duplex ultrasonography should generally be reserved for use in symptomatic patients in whom anatomic diagnostic data is required for care. †Other causes of leg pain may include: lumbar disk disease, 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 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. Adapted with permission from Hiatt WR. Medical treatment of peripheral arterial disease and claudication. N Engl J Med 2001;344:1608–21 (179a). Copyright © 2001 Massachusetts Medical Society. All rights reserved. ABI = ankle-brachial index; $HgbA_{1c}$ = hemoglobin A; JNC-7 = Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; LOE = level of evidence; NCEP ATP III = National Cholesterol Education Program Adult Treatment Panel III.

Several clinical features raise the suspicion of RAS and provide relative indications for application of more specific diagnostic testing strategies. Such diagnostic testing strategies should be performed when establishment of the RAS diagnosis is likely to offer information that can beneficially be linked to an effective patient-specific treatment strategy. See Figure 10 for a summary.

C. Diagnostic Methods

RECOMMENDATIONS

Class I

1. **Duplex ultrasonography is recommended as a screening test to establish the diagnosis of RAS.** ***(Level of Evidence: B)***

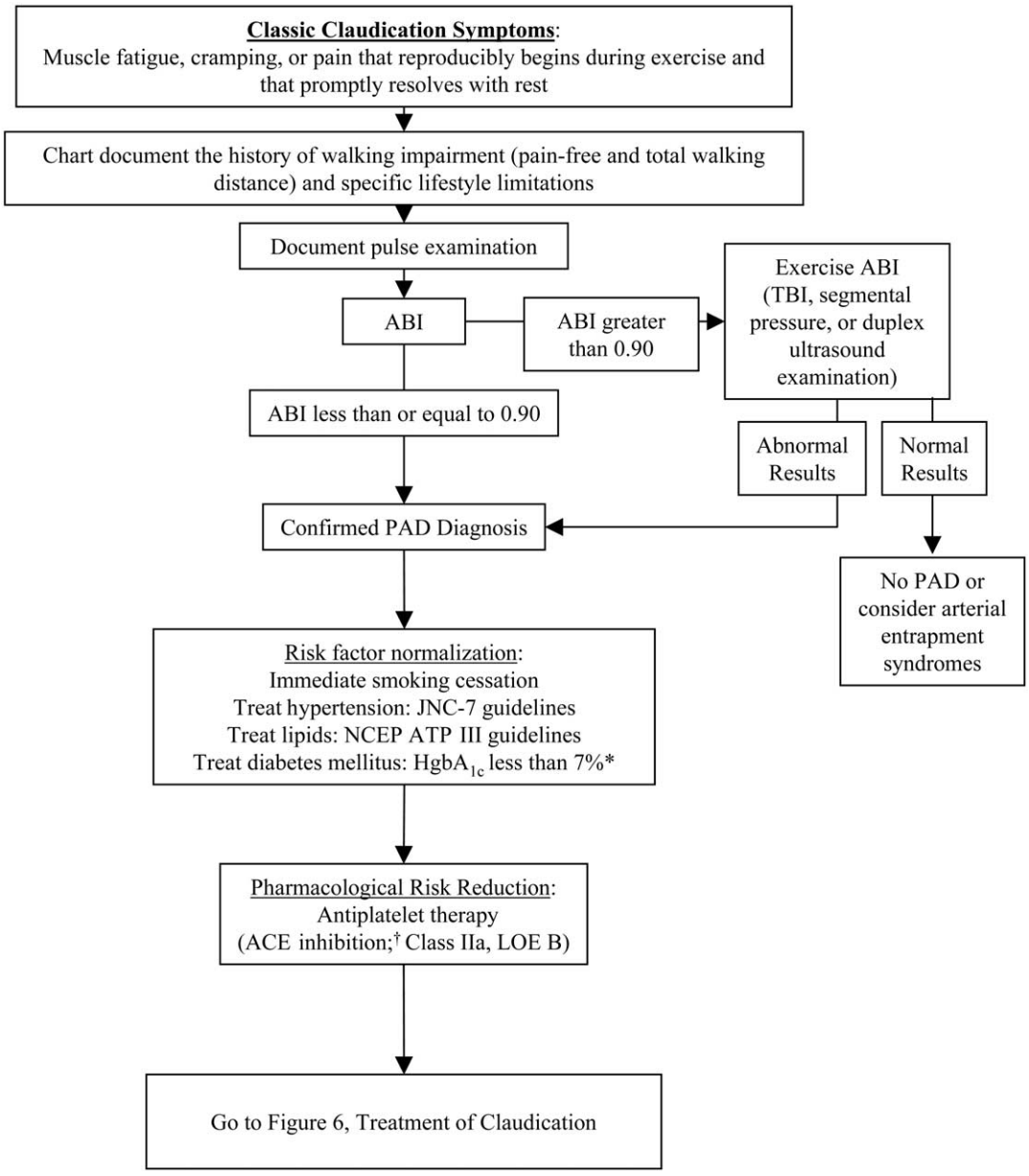

Figure 5. Diagnosis of claudication and systemic risk treatment. *It is not yet proven that treatment of diabetes mellitus will significantly reduce peripheral arterial disease (PAD)-specific (limb ischemic) endpoints. Primary treatment of diabetes mellitus should be continued according to established 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. ABI = ankle-brachial index; $HgbA_{1c}$ = hemoglobin A; JNC-7 = Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; LOE = level of evidence; NCEP ATP III = National Cholesterol Education Program Adult Treatment Panel III.

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)***

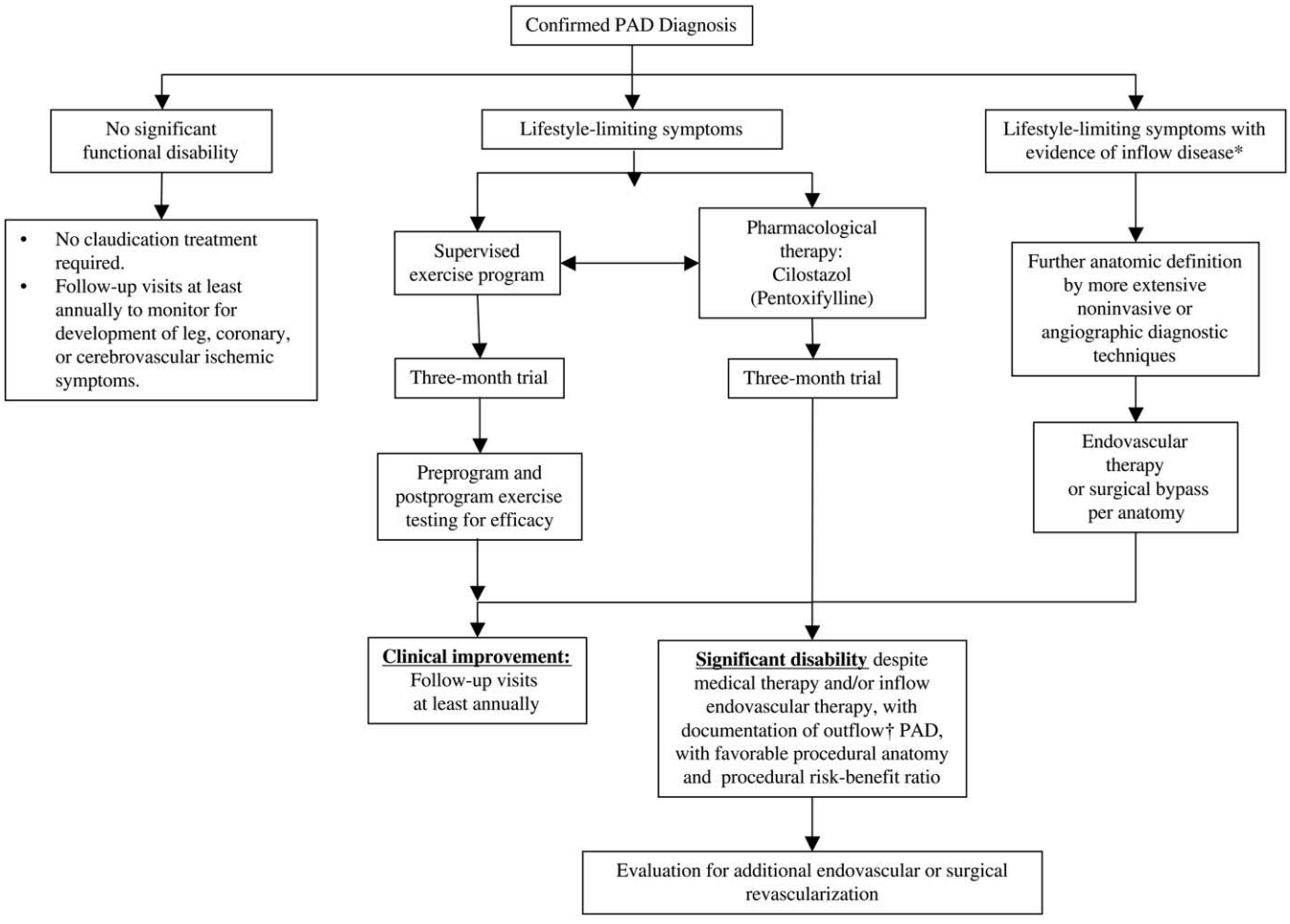

Figure 6. Treatment of claudication. *Inflow disease should be suspected in individuals with gluteal or thigh claudication and femoral pulse diminution or bruit and should be confirmed by noninvasive vascular laboratory diagnostic evidence of aortoiliac stenoses †Outflow disease represents femoropopliteal 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). PAD = peripheral arterial disease.

4. **The captopril test (measurement of plasma renin activity after captopril administration) is not recommended as a useful screening test to establish the diagnosis of RAS.** ***(Level of Evidence: B)***

Renal artery stenosis is best diagnosed with an imaging modality. The ideal tool should evaluate both the main and accessory renal arteries, assess the hemodynamic significance of the demonstrated lesions, identify the site and severity of the stenosis, and identify associated perirenal pathology, including the presence of an abdominal aortic aneurysm or renal or adrenal masses. Direct imaging modalities such as duplex ultrasound, CTA, and MRA are best suited to serve as effective diagnostic screening methods. The choice of imaging procedure will depend on the availability of the diagnostic tool, the experience and local accuracy of the chosen modality, and patient characteristics (e.g., body size, renal function, contrast allergy, and presence of prior stents or metallic objects that may serve as contraindications to MRA or CTA techniques).

Summary of Noninvasive Renal Artery Diagnostic Imaging Strategies. There are relative advantages and disadvantages to each of the aforementioned imaging modalities. Captopril renography has been validated in a large number of patients but is limited in value to a subset of all potential renovascular patients and is of limited value in patients with significant azotemia, bilateral RAS, or RAS to a single functioning kidney. Duplex renal sonography, because of the critical role of the sonographer, is accurate in experienced laboratories and is thus ideally performed in high-volume accredited laboratories. The diagnostic accuracy of these ultrasound-based examinations is further limited in patients with large body habitus or intestinal gas obscuring visualization of the entirety of the renal artery. Computed tomographic angiography currently provides higher spatial resolution than MRA and may be more readily available; however, the requirement to use iodinated contrast makes it an unattractive modality in patients with impaired renal function. Gadolinium-enhanced MRA provides excellent and less-nephrotoxic characterization of the renal arteries, sur-

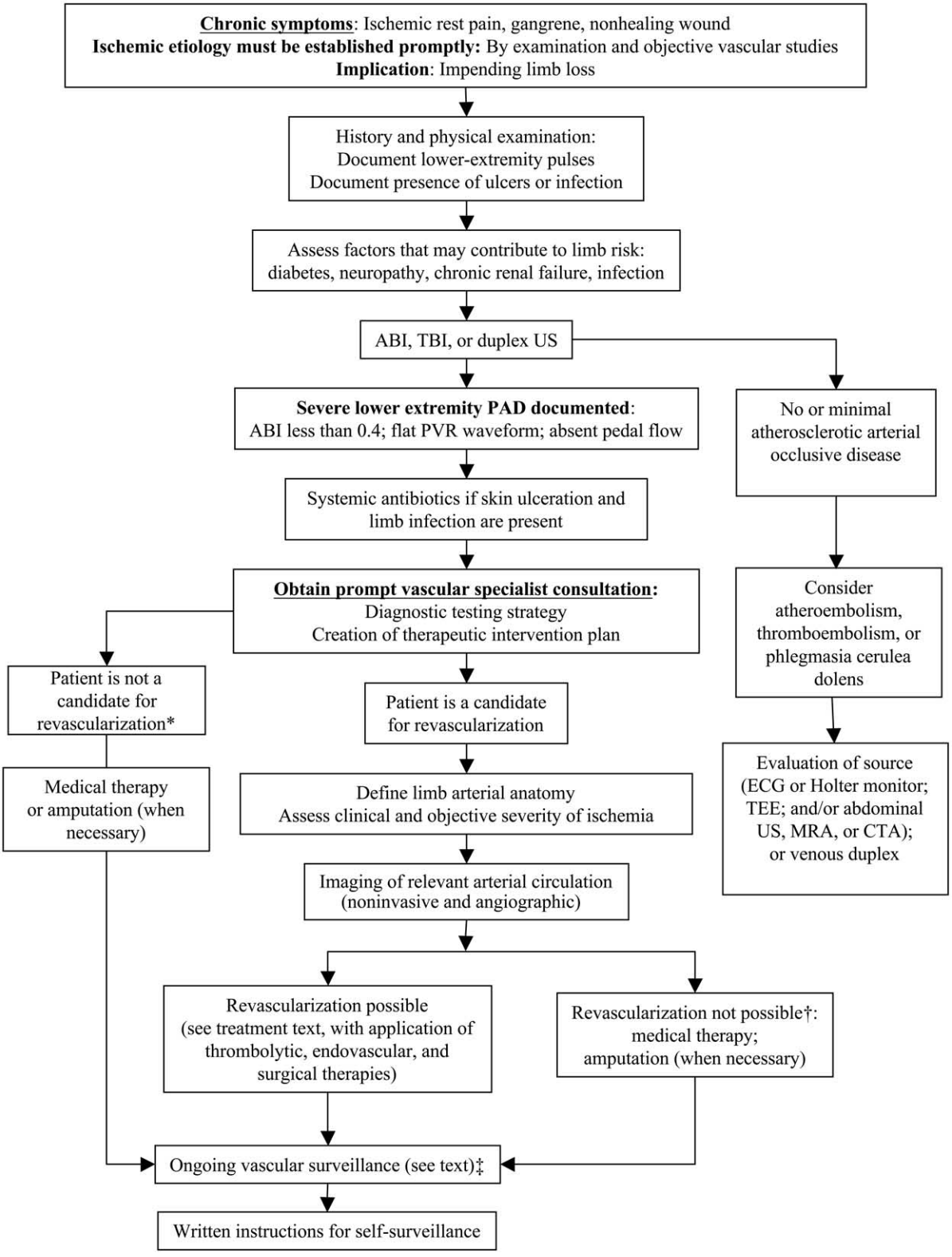

Figure 7. Diagnosis and treatment of critical limb ischemia (CLI). *Based on patient comorbidities. †Based on anatomy or lack of conduit. ‡Risk factor normalization: immediate smoking cessation, treat hypertension per the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, 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] 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. 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 = ultrasound.

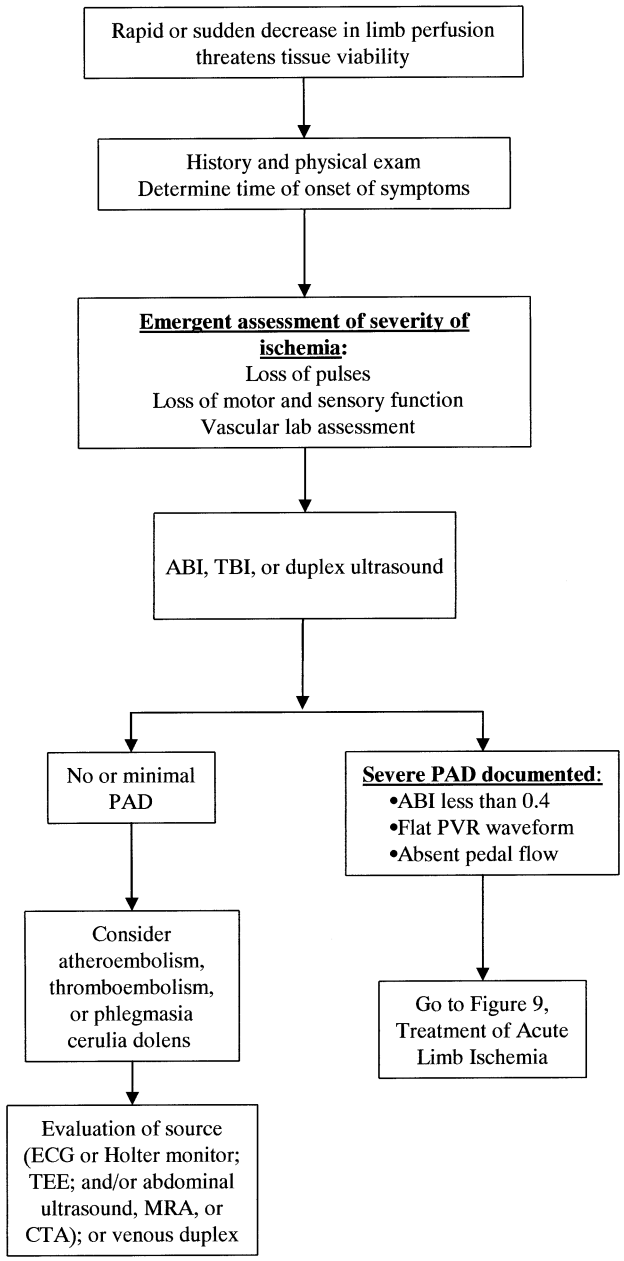

Figure 8. Diagnosis of acute limb ischemia. Adapted from J Vasc Surg 26, Rutherford RB, Baker JD, Ernst C, et al., Recommended standards for reports dealing with lower extremity ischemia: revised version, 517–38, Copyright 1997, with permision from Elsevier (180a). ABI = ankle-brachial index; CTA = computed tomographic angiography; ECG = electrocardiogram; MRA = magnetic resonance angiography; PVR = pulse volume recording; TBI = toe-brachial index; TEE = transesophageal echocardiography.

rounding vessels, renal mass, and perhaps renal function, but it remains the most costly renal artery examination. It is far less useful in patients who have had a metallic renal artery stent placed because of the inability to image inside of the stent to detect restenosis. Comparisons of contrast-enhanced 3-dimensional MRA and multidetector CTA with digital subtraction catheter angiography in a large number of arterial segments have demonstrated equally high sensitivities for detection of hemodynamically significant stenoses for MRA and CTA (greater than 90%), with excellent interobserver and intermodality agreement (kappa equals 0.88 to 0.90) (180).

1. Catheter Angiography. The indications for catheter-based contrast renal angiography include (a) individuals in whom there are prespecified indications to suspect clinically important RAS ("clinical clues") in whom definitive diagnostic noninvasive images cannot be obtained and (b) individuals in whom these prespecified clinical indications and patient consent have been documented and in whom concomitant angiographic access has been obtained for peripheral angiography or coronary angiography. Catheter-based contrast angiography is associated with a low rate of serious adverse outcomes.

2. Renin.

A. SELECTIVE RENAL VEIN RENIN STUDIES. The utility of renal vein renin measurements depends on the ability to differentiate the unilateral elevation of renin concentration from the renal vein that drains the kidney with renal artery disease from the systemic plasma renin levels and/or renal vein renin levels collected from the contralateral (normal) kidney. The test may have more utility in establishing an indication for nephrectomy in patients with renal artery occlusion than in identifying patients with RAS who may derive benefit from revascularization (181); for pediatric patients with questionably severe RAS before revascularization; or for patients with very marked aortoiliac-renal atherosclerosis, in whom revascularization could carry unusually high risk.

B. PLASMA RENIN ACTIVITY: CAPTOPRIL TEST. The overall sensitivity of this test is 61%, with a specificity of 86% for the detection of RAS; however, this test is less accurate in patients who are volume expanded or who have chronic renal failure, bilateral renal artery disease, or disease to a solitary functioning kidney. Plasma renin activity is not recommended as a useful screening test to establish the diagnosis of RAS.

D. Treatment of Renovascular Disease: Renal Artery Stenosis

Treatment of renal arterial disease should serve to aid in the normalization of blood pressure and to preserve renal function. Both medical (pharmacological) and revascularization strategies should be considered for patients with documented renal arterial disease. A treatment algorithm is provided in Figure 11.

1. Medical Treatment.

RECOMMENDATIONS

Class I

1. **Angiotensin-converting enzyme inhibitors are effective medications for treatment of hypertension associated with unilateral RAS.** ***(Level of Evidence: A)***

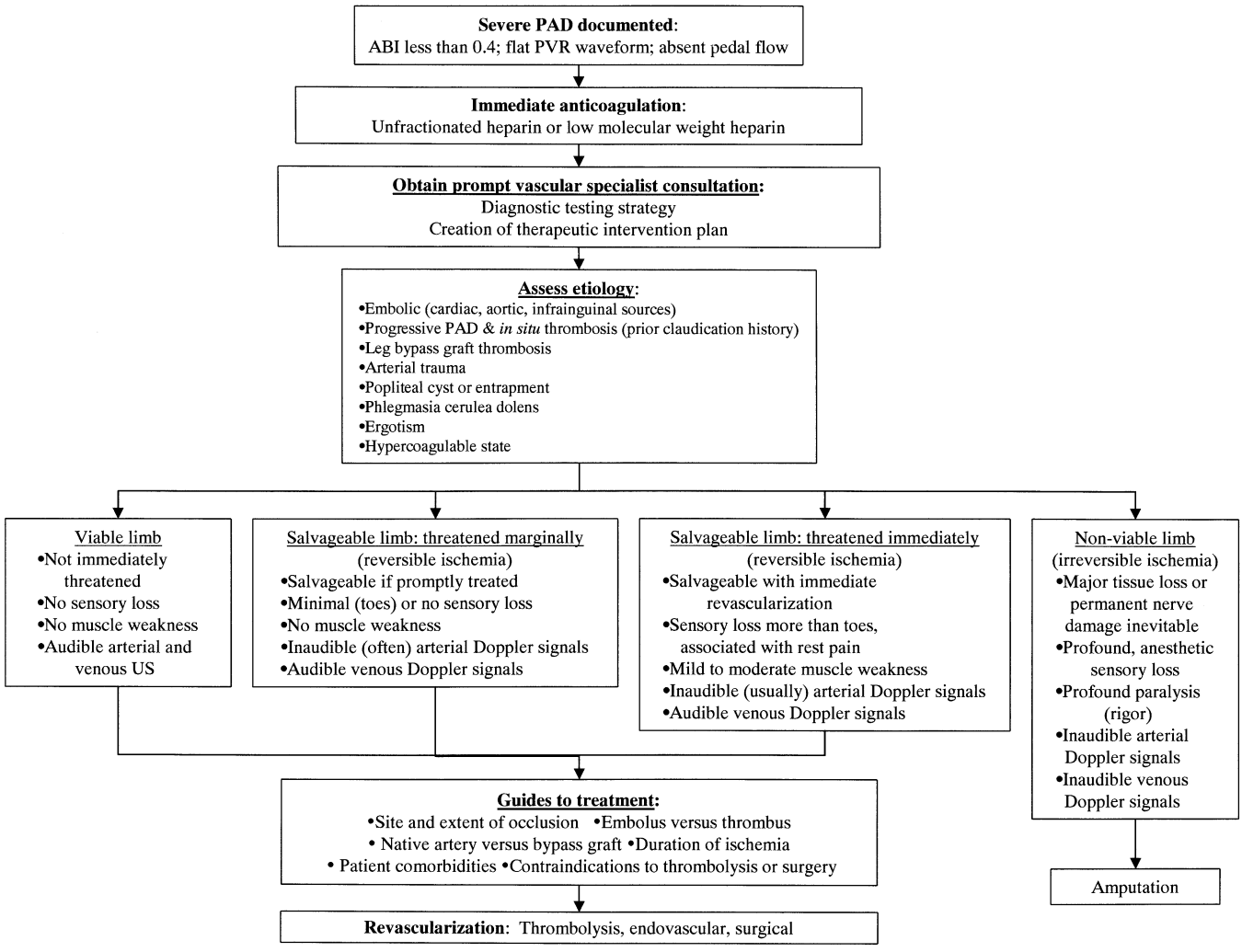

Figure 9. Treatment of acute limb ischemia. Adapted from J Vasc Surg 26, Rutherford RB, Baker JD, Ernst C, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version, 517–38, Copyright 1997, with permission from Elsevier (180a). PAD = peripheral arterial disease; PVR = pulse volume recording; US = ultrasound.

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)***

Multiple studies have now shown that ACE inhibitors and calcium-channel blockers are effective in the treatment of hypertension in the presence of RAS (182–186). These results address primarily the treatment of hypertension, but diminution in the progression of renal disease has also been demonstrated. There is also evidence that alternative therapies, based largely on chlorothiazide, hydralazine, and beta-blockers, also appear effective to achieve target blood pressures in individuals with RAS. Although the angiotensin II receptor blockers also have an evidence base of efficacy for normalization of blood pressure in individuals with RAS, their effects need to be tested further in large randomized trials. There are currently few objective clinical clues that permit selection of specific patient cohorts that would best be treated by medical therapy versus renal arterial revascularization, which remains an area of active clinical investigation. Individuals with atherosclerotic disease and hypertension should be treated according to the goals of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (187).

2. Indications for Revascularization.

A. ASYMPTOMATIC STENOSIS.

RECOMMENDATIONS

Class IIb

1. **Percutaneous revascularization may be considered for treatment of an asymptomatic bilateral or solitary**

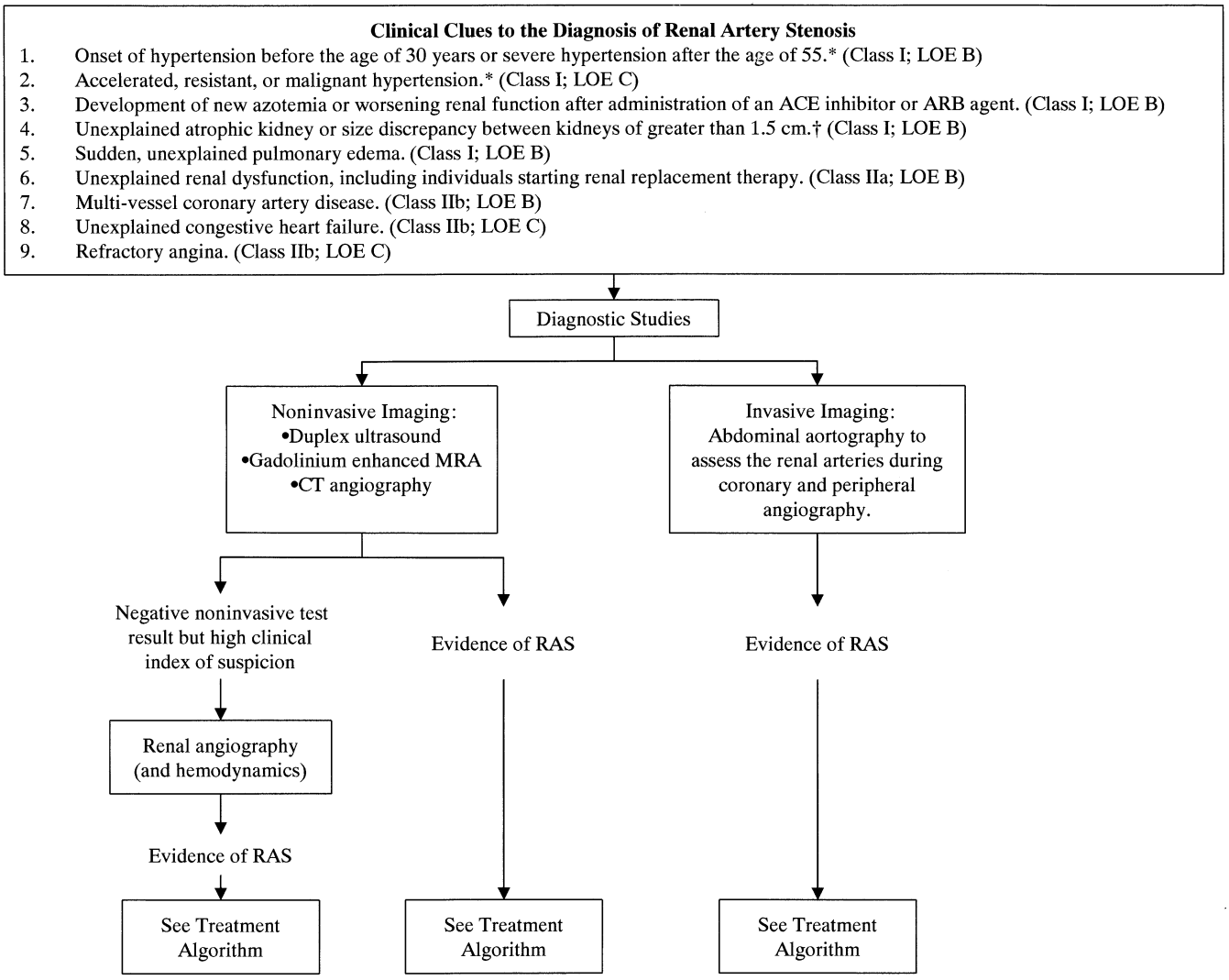

Figure 10. Clinical clues to the diagnosis of renal artery stenosis. *For definition of hypertension, please see Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 2003;289:2560–72 (187). †For example, atrophic kidney due to chronic pyelonephritis is not an indication for RAS evaluation. ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocking agent; CT = computed tomography; LOE = level of evidence; MRA = magnetic resonance angiography; RAS = renal artery stenosis.

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)***

There are no well-controlled prospective randomized investigations to measure the relative risk and benefit of endovascular interventions (or associated medical therapies) in individuals with asymptomatic renal artery disease, and thus, the role of such interventions remains controversial. Recommendations regarding the role of percutaneous revascularization of asymptomatic renal disease are made largely on the basis of expert opinion and are not based on evidence that treatment of asymptomatic RAS improves any renal or systemic outcome, including renal preservation, blood pressure, or cardiovascular morbidity or mortality. Therefore, these recommendations must be individualized for the patient by each treating physician.

B. HYPERTENSION.

RECOMMENDATIONS

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)***

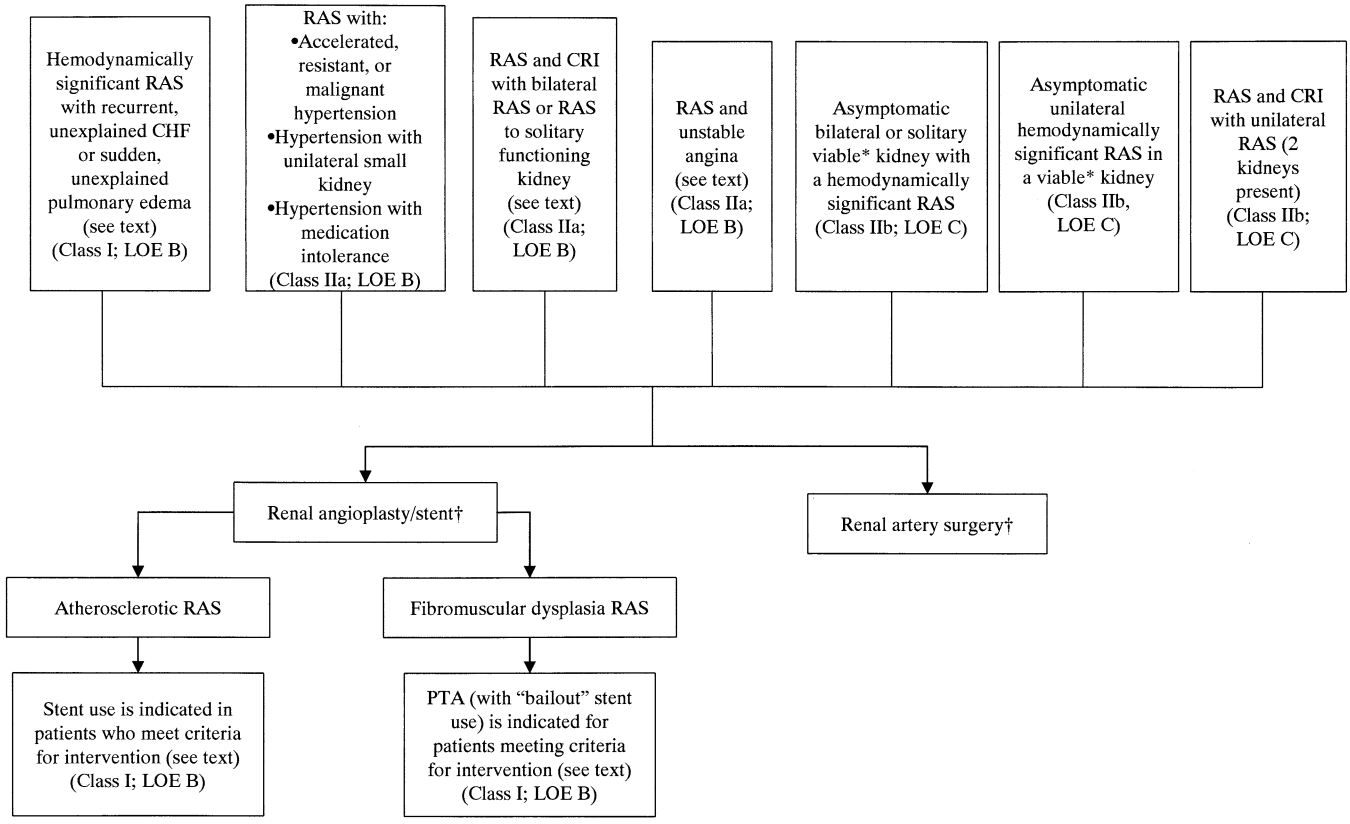

Figure 11. Indications for renal revascularization. *Viable means kidney linear length greater than 7 cm. †It is recognized that renal artery surgery has proven efficacy in alleviating renal arterial stenosis (RAS) due to atherosclerosis and fibromuscular dysplasia. Currently, however, its role is often 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; PTA = percutaneous transluminal angioplasty.

The current evidence base suggests that patients with severe atherosclerotic RAS and accelerated, resistant, and malignant hypertension may expect to receive some clinical benefit, including improved blood pressure control, the need for fewer medications, or both. However, "cure" of hypertension is rare, improvement in blood pressure control is common, and a moderate fraction of individuals do not achieve measurable benefit (see Table 36 of the full-text guidelines).

C. PRESERVATION OF RENAL FUNCTION.

RECOMMENDATIONS

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)***

Revascularization is effective in stabilizing or improving renal function in patients with symptomatic atherosclerotic RAS (188–193). Several factors may argue against renal revascularization or predict poorer outcomes, including the presence of proteinuria greater than 1 g every 24 hours, renal atrophy, severe renal parenchymal disease, and severe diffuse intrarenal arteriolar disease. Moreover, the adverse consequences of renal atheroembolization at the time of surgical revascularization have been documented (194). Similarly, potentially severe atheroembolization may be provoked by renal percutaneous revascularization methods (195).

D. IMPACT OF RAS ON CONGESTIVE HEART FAILURE AND UNSTABLE ANGINA.

RECOMMENDATIONS

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

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

The potential physiological benefits of renal stent placement include reperfusion of the ischemic kidney(s), resulting in a reduction in the stimulus to renin production, which decreases angiotensin and aldosterone production, thereby decreasing peripheral arterial vasoconstriction and the tendency to develop an expanded extracellular fluid volume. Improvement in renal perfusion enhances glomerular filtration and therefore promotes natriuresis. Finally, in patients with a solitary kidney or bilateral RAS, the ability of the patient to tolerate long-term administration of angiotensin antagonist medications may be facilitated by relief of a hemodynamic renal artery obstruction. The recommendations in these guidelines are intended to apply to individuals with refractory heart failure or unstable angina in whom nonrenal exacerbating factors have been evaluated and in whom there are reasonable clinical indications to suggest the presence of RAS (e.g., systemic atherosclerosis), as is more fully described in the full-text version of the guidelines.

3. Catheter-Based Interventions.

RECOMMENDATIONS

Class I

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

Percutaneous transluminal renal balloon angioplasty is the treatment of choice for symptomatic RAS caused by fibromuscular dysplasia (188,196–198). However, in atherosclerotic RAS, balloon angioplasty alone is associated with a lower procedural success rate and a higher restenosis rate (189,199–205). Aorto-ostial stenoses represent the most common atherosclerotic lesions and are prone to vascular recoil due to confluent plaque that extends from the wall of the aorta into the ostium of the renal artery. These atherosclerotic aorto-ostial lesions are generally considered unsuitable for treatment by balloon angioplasty alone (197,198,206).

Stent placement has consistently proven superior to balloon angioplasty in the treatment of renal artery atherosclerotic lesions (207,208). For renal artery atherosclerotic lesions, the larger the poststent minimal lumen diameter, as measured by quantitative vascular angiography, the better the late stent patency (209). Similar to coronary stents, larger-diameter renal arteries have lower restenosis rates than smaller-diameter vessels (210,211).

4. Surgery for RAS.

RECOMMENDATIONS

Class I

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

A. RESULTS OF OPERATIVE THERAPY. Surgical treatment of renovascular hypertension affords good clinical outcomes (212–216). The risk of surgery increases in patients who require concomitant aortic reconstruction, in patients with renal insufficiency, and when aortic grafts are used as a source of the bypass graft. The need for reoperation has been reported in 5% to 15% and survival in 65% to 81% of patients (212–216).

IV. MESENTERIC ARTERIAL DISEASE

Regardless of cause, intestinal ischemia is rare, and there are no randomized or controlled trials of diagnosis or therapy for intestinal ischemia. Important gaps thus exist in our knowledge of the natural history of intestinal ischemia, and yet the primary diagnoses responsible for most cases have been known for decades. Numerous series documenting the results of surgical treatment have been reported, and the clinical course of patient case series treated by percutaneous intervention has also been documented. These largely retrospective clinical reviews form the basis for our knowledge of and recommendations for treatment of intestinal ischemia.

A. Acute Intestinal Ischemia

1. Acute Intestinal Ischemia Caused by Arterial Obstruction.

A. ETIOLOGY. Acute obstructive intestinal ischemia occurs when the intestinal arteries are suddenly blocked to such a degree that all or part of the intestine has insufficient perfusion for viability. The many possible causes include embolism from cardiac or proximal arterial sources and arterial thrombosis (217–222). Regardless of the cause, patients with acute intestinal ischemia present with severe abdominal pain that is initially out of proportion to any physical findings that may be present.

B. DIAGNOSIS.

RECOMMENDATIONS

Class I

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

Class III

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

Clinical Presentation. Approximately two thirds of patients with acute intestinal ischemia are women, with a median age of 70 years. Most patients have a history of pre-existing cardiovascular disease (217–219,222). Abdominal pain is always present; its nature, location, and duration are variable, but most commonly, the pain is anterior, periumbilical, and sufficiently severe that medical attention is sought immediately. Initially, signs of peritoneal irritation are absent, which is classically referred to as "pain out of proportion to physical findings."

Laboratory Findings. Laboratory evaluation most frequently shows leukocytosis and lactic acidosis, and amylase is elevated in approximately 50% of patients; approximately 25% of patients have occult blood in the stool. Abdominal radiographs most frequently show some dilated loops of intestine. There are no specific laboratory or plain radiograph findings for acute intestinal ischemia.

Ultrasound. Although duplex ultrasound scanning is capable of identifying occlusive lesions of the intestinal arteries, in practice, it is not very helpful. The abdominal distention and fluid frequently present with acute ischemia preclude successful scanning in most patients. Because of the need for emergent treatment in acute ischemia and the time required to attempt duplex scanning, this test is contraindicated.

Computed Tomographic (CT) Scanning. Because CT scanning for evaluation of abdominal pain requires administration of intravenous iodinated contrast material, which may affect later arteriography, this test is not the best initial examination for suspected acute intestinal ischemia, although it is frequently performed before consideration of the mesenteric ischemia diagnosis.

Arteriography. Arteriography is the most helpful diagnostic test in patients suspected of having acute intestinal ischemia; however, its use is controversial because of the time required for its performance in the emergency setting. In patients suspected of having intestinal ischemia, arteriography can be diagnostic and can differentiate occlusive from nonocclusive ischemia. The decision for arteriography is probably best individualized in patients suspected of having acute intestinal ischemia. For those with a very acute presentation, a high likelihood of arterial obstruction, and suspected bowel infarction, immediate laparotomy by a surgeon capable of intestinal revascularization is the best approach. In patients with acute onset in whom angiography can be performed rapidly and without delay, this is a reasonable approach. For those with a more delayed presentation or a high likelihood of nonocclusive ischemia, initial arteriography is indicated. In these cases, the advantages of the additional information provided by arteriography outweigh the time required for its performance.

C. NATURAL HISTORY. All series of acute intestinal ischemia patients include some who had a history of chronic abdominal pain and weight loss. The frequency with which chronic intestinal ischemia caused by arterial obstruction becomes acute intestinal ischemia (presumably by thrombosis) is unknown.

D. SURGICAL TREATMENT.

RECOMMENDATION

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)***

Surgical treatment consists of laparotomy, revascularization of the ischemic intestine either by embolectomy or by bypass grafting, assessment of the viability of the intestine after revascularization, resection of nonviable intestine, and intensive care. Scheduled "second look" operations, 24 to 48 hours after the initial procedure, are the best way to avoid both excessive resection of potentially viable bowel and failure to resect nonviable intestine.

E. ENDOVASCULAR TREATMENT.

RECOMMENDATION

Class IIb

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

Despite limited data, percutaneous treatment (lytic therapy; balloon angioplasty or stenting or both) of the arterial

obstruction is reasonable given the high mortality associated with the standard operative approach (223–225). However, because most patients with acute intestinal ischemia have at least some nonviable intestine at the time of presentation, most will still require laparotomy, and surgical assessment of intestinal viability may be required even if percutaneous therapy is successful in relieving the obstruction. Reestablishment of flow to infarcted bowel may cause a sudden systemic release of endotoxins, which may be associated with the sudden onset of disseminated intravascular coagulation, adult respiratory distress syndrome, and sudden cardiovascular collapse. Therefore, in the presence of infarcted bowel or markedly elevated lactic acid levels, initial percutaneous treatment should be weighed against surgical options in which control of the venous outflow (and the endotoxins) from the infarcted bowel segment can be achieved.

2. Acute Nonocclusive Intestinal Ischemia.

A. ETIOLOGY.

RECOMMENDATIONS

Class I

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

Acute intestinal ischemia sufficient to produce infarction also occurs in the absence of fixed arterial obstruction. The most frequent setting is severe systemic illness with systemic shock, usually as a result of reduced cardiac output (217,226–230). In this situation, the intestinal ischemia has been shown to be the result of severe and prolonged intestinal arterial vasospasm. Intestinal vasospasm sufficient to produce ischemia/infarction also occurs as a result of cocaine ingestion and ergot poisoning (231,232). Therapeutic drugs may produce intestinal ischemia from vasospasm, especially when vasopressors are used in high doses to treat circulatory shock.

Intestinal ischemia can also occur as a result of mesenteric arterial spasm after repair of aortic coarctation (233) and occasionally occurs after revascularization procedures for chronic mesenteric ischemia (228). The mechanism of this apparently paradoxical spasm is unknown.

B. DIAGNOSIS.

RECOMMENDATIONS

Class I

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

There are no physical findings or laboratory tests specific for nonocclusive intestinal ischemia, although it should be suspected whenever patients with circulatory shock, especially cardiogenic shock, develop abdominal pain or distention and in patients treated with ergot, as well as in persons using cocaine or amphetamines who have abdominal pain. Arteriography is the "gold standard" for diagnosis. It can demonstrate the characteristic mesenteric arterial vasospasm and allow direct intra-arterial instillation of vasodilator medications (227,229,232).

C. TREATMENT.

RECOMMENDATIONS

Class I

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

Class IIa

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

Initial treatment of nonocclusive intestinal ischemia should be directed at treatment of the underlying shock state. The most intensive hemodynamic monitoring possible, including appropriate fluid/pharmacological therapy to improve cardiac output/peripheral perfusion, is the most reliable way to relieve the inappropriate vasospasm. Administration of vasodilators by percutaneously placed catheters at the site of inappropriate vasospasm has been associated with relief of vasospasm/ischemic symptoms in multiple patients (226). Transcatheter administration of vasodilators is especially appropriate in nonocclusive mesenteric ischemia caused by drugs such as ergot or cocaine, in which systemic shock may not coexist (234). Abdominal symptoms/findings that persist after relief of intestinal arterial vasospasm are an indication for laparotomy/resection of necrotic intestine.

B. Chronic Intestinal Ischemia

1. Etiology. Although atherosclerotic disease of the celiac and mesenteric vessels is common, the clinical presentation of chronic intestinal ischemia is rare. It is nearly uniformly caused by atherosclerosis (235). Classic clinical approaches to the diagnosis of intestinal ischemia have often suggested that this syndrome requires occlusion or stenosis of at least 2 of the 3 intestinal arteries; however, this is not entirely true (236,237). Well-documented cases of intestinal ischemia occur as a result of single-vessel disease, virtually always of the superior mesenteric artery. Patients in whom some of the normal collateral intestinal arterial connections have been interrupted by previous surgery are especially vulnerable to single-vessel occlusions.

Patients with chronic intestinal ischemia are most often female (70%) and classically complain of severe abdominal pain induced by eating. The pattern of pain is quite variable, however, and the relationship to food is not always clear, at least by history. What is clear is that patients voluntarily vastly reduce their food intake, so that weight loss occurs, and this may be profound. Vomiting, diarrhea, and constipation are present in a minority of patients. A majority have a history of cardiovascular disease, and 30% to 50% have had previous operations for atherosclerotic disease, most frequently coronary and lower extremity bypass (238,239).

2. Diagnosis.

RECOMMENDATIONS

Class I

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

Because there are many common causes of abdominal pain and weight loss, and because chronic intestinal ischemia is rare, diagnosis is delayed in most patients. At present, there are no diagnostic tests that establish the diagnosis definitively. Rather, it is the combination of the typical clinical presentation of abdominal pain and weight loss, with other evidence of cardiovascular disease, combined with the finding of intestinal arterial obstruction in the absence of any other obvious cause of the symptoms that should lead to consideration of the diagnosis. Duplex scanning of visceral vessels is technically difficult but can be accomplished in more than 85% of subjects in the elective setting. The test has an overall accuracy of approximately 90% for detection of greater than 70% diameter stenoses or occlusions of the celiac and superior mesenteric arteries when performed in highly experienced laboratories (240–242). Both contrast-enhanced CTA and gadolinium-enhanced MRA are well suited for visualizing the typical atherosclerotic lesions at the origins of the intestinal arteries, although they are less suited for visualizing the more distal intestinal arteries and for diagnosis of some of the more unusual causes of intestinal ischemia. Arteriograms provide definitive diagnosis of intestinal arterial lesions, although not chronic intestinal ischemia. Lateral aortography is best suited for display of the typical origin lesions, which may not be apparent on frontal projections. The presence of an enlarged "Arc of Riolan" (an enlarged collateral vessel connecting the left colic branch of the inferior mesenteric artery with the superior mesenteric artery) is an arteriographic sign of proximal mesenteric arterial obstruction that is visible on anteroposterior aortograms.

3. Natural History. Significant atherosclerotic obstruction of the intestinal arteries is present in 6% to 10% of unselected autopsies and in 14% to 24% of patients undergoing abdominal arteriography. Development of symptomatic intestinal ischemia in patients with asymptomatic intestinal arterial obstruction after abdominal surgery for other reasons has been described (243). The natural history of symptomatic chronic intestinal ischemia is known in part. An unknown percentage of patients progress to acute intestinal ischemia. The remainder have progressive weight loss with ultimate death from inanition.

4. Interventional Treatment.

RECOMMENDATION

Class I

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

A large number of reports in the literature have documented that percutaneous interventional treatment of intestinal arterial obstructions is possible with a high technical success rate and few complications in properly selected cases (244–249). Most procedures have been performed to treat intestinal arterial stenoses, with few attempting to treat occlusions. To date, there have been no prospective therapeutic trials, and follow-up information is limited. Several reports of concurrent series treated by angioplasty/stenting or surgery indicate that recurrences after percutaneous procedures have been more frequent than after open surgery, but many of the recurrences can be managed by percutaneous interventions (250). The results of several series are listed in Table 42 of the full-text guidelines. The reported recurrence rates mandate that patients treated with angio-

plasty and stents undergo careful follow-up. As with open surgery, recurrent symptoms have nearly always indicated recurrent arterial obstruction.

5. Surgical Treatment.

RECOMMENDATIONS

Class I

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

Class IIb

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

Class III

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

Surgical treatment of chronic intestinal ischemia is accomplished by endarterectomy or bypass grafting, with the majority of surgeons preferring the latter approach (239,251–258). In chronic cases, the overall operative mortality and durability of revascularization described by multiple contemporary reports are listed in Table 42 in the full-text guidelines. Long-term patency and relief of symptoms are the rule, with few recurrences; however, long-term follow-up is mandatory. Essentially all symptomatic recurrences are the result of recurrent stenosis or occlusion of visceral arteries or the reconstructions.

V. ANEURYSMS OF THE ABDOMINAL AORTA, ITS BRANCH VESSELS, AND THE LOWER EXTREMITIES

Although their etiology may be substantially different in some cases, 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. Like occlusive 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. Just as important, there are now a variety of therapeutic options that include both traditional open surgery and endovascular techniques, such that relatively few large aneurysms should merely be observed until morbid events occur.

A. Definition

There is abundant information concerning normal diameters of the abdominal aorta and its branches in healthy adults that indicates enlargement with age and body size and larger diameters in men than in women (Table 23) (259–261). Generally, an abdominal aortic aneurysm (AAA) is considered to be present when the minimum anteroposterior diameter of the aorta reaches 3.0 cm.

B. Abdominal Aortic and Iliac Aneurysms

1. Prevalence. The prevalence of AAAs varies with a number of demographic factors, including advancing age, family history, male gender, and tobacco use. In general, the prevalence of AAAs 2.9 to 4.9 cm in diameter ranges from 1.3% for men aged 45 to 54 years up to 12.5% for men 75 to 84 years of age. Comparable prevalence figures for women are 0% and 5.2%, respectively (Table 24).

Table 23. Dimensions of Normal Arteries

	Female		Male		
Artery	Mean Diameter, cm, Range	SD, cm, Range	Mean Diameter, cm, Range	SD, cm, Range	Assessment Method
Abdominal aorta, supraceliac	2.10–2.31	0.27	2.50–2.72	0.24–0.35	CT
Abdominal aorta, suprarenal	1.86–1.88	0.09–0.21	1.98–2.27	0.19–0.23	CT
Abdominal aorta, infrarenal	1.66–2.16	0.22–0.32	1.99–2.39	0.30–0.39	CT, intravenous arteriography
Abdominal aorta, infrarenal	1.19–1.87	0.09–0.34	1.41–2.05	0.04–0.37	B-mode ultrasound, CT, intravenous arteriography
Celiac	0.53	0.03	0.53	0.03	B-mode ultrasound
Superior mesenteric	0.63	0.04	0.63	0.04	B-mode ultrasound
Common iliac	0.97–1.02	0.15–0.19	1.17–1.23	0.20	CT
Internal iliac	0.54	0.15	0.54	0.15	Arteriography
Common femoral	0.78–0.85	0.07–0.11	0.78–1.12	0.09–0.30	CT, B- or M-mode ultrasound
Popliteal	NA	NA	0.9	0.2	B-mode ultrasound
Posterior tibial	NA	NA	0.3	0.01	M-mode ultrasound

Adapted from J Vasc Surg, 13, Johnston K, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. Subcommittee on Reporting Standards for Arterial Aneurysms, Ad Hoc Committee on Reporting Standards, Society for Vascular Surgery and North American Chapter, International Society for Cardiovascular Surgery, 452–8, Copyright 1991, with permission from the Society for Vascular Surgery and the American Association for Vascular Surgery (261a).

CT = computed tomography; NA = not available; SD = standard deviation.

Table 24. Prevalence of Abdominal Aortic Aneurysms in Population-Based Screening Studies

Country/Study	First Author	Reference	Number Screened	Age, yrs	Criteria	Prevalence/ Gender, %	Relative Risk
Western Australia	Jamrozik	(262)	12 203	65–69	Larger than 3.0 cm	4.8/Male	Higher risk: Current or ex-smokers
				80–83	Larger than 3.0 cm	10.8/Male	Established PAD, CAD
				65–83	Larger than 5.0 cm	0.69/Male	Waist-hip ratio larger than 0.9
							Lower risk: Mediterranean born vs. Australian born (OR 0.6) Regular vigorous exercise
Veterans Affairs Cooperative Study	Lederic	(263)	126 196*	50–79	Larger than 4.0 cm	1.3/Male and female	Higher risk: Increased age per 7 yrs (OR 1.7)
				50–79	Larger than 4.9 cm	0.45/Male and female	Smoking history (OR 5.17) Family history (OR 1.9)
				50–79	Larger than 5.4 cm	0.27/Male and female	Established atherosclerosis (OR 1.6)
							Lower risk: Female (OR 0.18; 2.7% of total) Black race (OR 0.59) Diabetes mellitus (OR 0.50)
Norway	Singh	(264)	6386	25–84	Larger than 2.9 cm	8.9/Male 2.2/Female	Higher risk: Increased age
				45–54	Larger than 2.9 cm	1.9/Male	Smoker older than 40 yrs versus never-smoker (OR 8.0)
				55–64		0/Female 6.0/Male 1.1/Female	
				65–74		12.8/Male 2.8/Female	
				75–84		18.5/Male 4.8/Female	
				55–64	Larger than 3.9 cm	1.1/Male 0.1/Female	
				65–74		4.1/Male 0.7/Female	
				75–84		8.6/Male 1.0/Female	
The Netherlands	Pleumeekers	(265)	5283†	Older than 54	3.4 to 3.6 cm or distal dilation greater than 49%	2.8/Male, 0.5/female	Higher risk: Smoker High serum cholesterol Established cardiovascular disease
				Older than 54	Larger than 4.0 cm	1.6/Male, 0.3/female	
The Netherlands	Boll	(266)	2419‡	60–80	Larger than 2.9 cm	8.1/Male	
					Larger than 4.9 cm	1.7/Male	
Japan	Adachi	(267)	1591	—	—	0.3/Male	

*52 745 plus prior report of 73 451. †Of 10 215 eligible. ‡Of 2914 eligible.
CAD = coronary artery disease; PAD = peripheral arterial disease; OR = odds ratio.

A. GENERALIZED ARTERIOMEGALY. Generalized arteriomegaly reflects a systemic alteration of the elastic component of the arterial wall that results in dilation and elongation of many arteries. Patients with localized AAAs are relatively unlikely to have generalized arteriomegaly (268), but the familial pattern of generalized arteriomegaly is similar. In one series, there was a family history of aneurysms in 10% (4/40) of patients with peripheral aneurysms,

in 22% (19/86) of patients with AAAs, and in 36% (5/14) of patients with generalized arteriomegaly (269).

2. Etiology. Most aortic and peripheral aneurysms represent a manifestation of aortic medial degeneration, which has complex biological mechanisms. Data (see full-text guidelines) suggest that many aneurysms form in response to altered tissue metalloproteinases that diminish the integrity of the arterial wall.

A. HEREDITARY RISK FACTORS. A family history of AAAs is particularly relevant for male siblings of male probands, in whom the relative risk for AAA is as high as 18% (270), which suggests a single dominant gene effect. (See Table 45 of the full-text guidelines.) First-degree male relatives of patients with AAA have 2 to 4 times the normal risk for AAA. Female first-degree relatives appear to be at similar risk, but the data are less certain.

B. ATHEROSCLEROTIC RISK FACTORS.

RECOMMENDATIONS

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)***

Patients with AAAs have a significantly higher prevalence of hypertension, smoking, MI, heart failure, and carotid artery disease or lower extremity PAD than do age- and gender-matched controls. The lipoprotein(a) serum level, an indicator of atherosclerosis, is also elevated in patients with AAA, independent of other cardiovascular risk factors and the extent of atherosclerosis (271).

C. COLLAGENASE, ELASTASE, AND METALLOPROTEASES. The striking histological feature of aortic aneurysms is destruction of the media and elastic tissue. Excessive proteolytic enzyme activity in the aortic wall may promote deterioration of structural matrix proteins, such as elastin and collagen (272). Smooth muscle cells derived from patients with AAAs display increased migration, perhaps related to overproduction of the matrix metalloproteinase MMP-2, which may lead to extracellular matrix remodeling and medial disruption (273). Abnormal biochemical elastolytic and active proteolytic activity has also been identified in aneurysmal aortas (274).

The association between AAAs and chronic obstructive pulmonary disease has been attributed to elastin degradation caused by tobacco smoking. Among 4404 men 65 to 73 years of age with a 4.2% prevalence of AAA, 7.7% of those with chronic obstructive pulmonary disease had aortic aneurysms (275). The overall mean annual expansion rate was 2.7 mm per year irrespective of chronic obstructive pulmonary disease but was 4.7 mm per year among patients treated with corticosteroid agents compared with 2.6 mm per year among those who were not treated (p less than 0.05).

D. INFLAMMATORY ANEURYSMS. Inflammatory AAAs represent a unique clinical entity, typically consisting of an AAA that is associated with an unusually thickened aneurysm wall, shiny white perianeurysmal fibrosis, and intense adherence of adjacent intra-abdominal structures. Abnormal accumulation of macrophages and cytokines in aneurysmal aortic tissue supports an association with inflammation (276,277). In a case-control study, there were no distinctions between patients with inflammatory aneurysms and those with noninflammatory aneurysms with respect to risk factors, treatment requirements, or prognosis, but patients with inflammatory aneurysms were more often symptomatic and had a higher erythrocyte sedimentation rate, larger aneurysm diameter, and more retroperitoneal inflammatory reaction (278).

The triad of chronic abdominal pain, weight loss, and elevated erythrocyte sedimentation rate in a patient with AAA is highly suggestive of an inflammatory aneurysm. Inflammatory aortic or iliac aneurysms were present in 4.5% of the 2816 patients who underwent elective AAA repair at the Mayo Clinic from 1955 to 1985 (279). More than 90% of the patients with inflammatory aneurysms were smokers, and clinical evidence of peripheral arterial occlusive disease and coronary artery disease was found in 27% and 39%, respectively. Compared with patients with noninflammatory atherosclerotic aneurysms, those with inflammatory aneurysms were more likely to have symptoms (66% vs. 20%, p less than 0.0001), weight loss (20.5% vs. 10%, p less than 0.05), a higher erythrocyte sedimentation rate (73% vs. 33%, p less than 0.0001), and a higher operative mortality rate (7.9% vs. 2.4%, p less than 0.002).

3. Natural History. The natural history of arterial aneurysms is distinguished by gradual and/or sporadic expansion in their diameter and by the accumulation of mural thrombus caused by turbulent blood flow at their periphery. These features contribute to the 3 most common complications of aneurysms, that is, rupture, thromboembolic ischemic events, and the compression or erosion of adjacent structures, which often are quite specific to their location.

A. AORTIC ANEURYSM RUPTURE.

RECOMMENDATIONS

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)***

Table 25. Rupture and Survival Rates for Patients With Abdominal Aortic Aneurysms, Since 1990

First Author	Reference	Year	No. of Patients	Baseline Aneurysm Diameter	Follow-Up Interval	Aneurysm Rupture Rate	Survival Rate
Case series							
Bengtsson	(282)	1993	155	Median, 4 cm	Median, 3.4 yrs	14%	30%
Perko	(283)	1993	63	Smaller than 6 cm		Less than 5%	NA
				Larger than or equal to 6 cm		10% to 15%	NA
Galland	(284)	1998	267	Smaller than 4 cm	5 yrs	4%	NA
				4 to 5.5 cm	5 yrs	21%	NA
Jones	(285)	1998	25	5 to 5.9 cm	3 yrs	28%	NA
			32	6 cm or larger	3 yrs	41%	NA
Scott	(286)	1998	218	3 to 4.4 cm	7 yrs	2.1% per year and/or operation	NA
				4.5 to 5.9 cm	7 yrs	10% per year and/or operation	NA
Conway	(281)	2001	23	5.5 to 5.9 cm	10 yrs	22%	39%
			62	6 to 7 cm	10 yrs	34%	32%
			21	Larger than 7 cm	10 yrs	52%	5%
Biancari	(287)	2002	41	2.5 to 4 cm	Median, 7.3 yrs	7.3%	59%
Collective reviews							
Hollier	(288)	1992	349	Smaller than 5 cm	5 yrs	4.6%	NA
			90	Larger than 5 cm	5 yrs	30%	NA
Hallin	(289)	2001	54 048	Smaller than 4 cm	4 yrs	2%	NA
				4 to 5 cm	4 yrs	10%	NA
				Larger than 5 cm	4 yrs	22%	NA
Randomized trials							
UK Small Aneurysm Trial (nonoperated cohort)	(290)	1998	213	4 to 4.4 cm	Mean, 4.6 yrs	NA	75%
			169	4.5 to 4.8 cm	Mean, 4.6 yrs	NA	72%
			145	4.9 to 5.5 cm	Mean, 4.6 yrs	NA	64%
UK Small Aneurysm Trial (nonoperated cohort)	(291)	1999	NA	3 to 3.9 cm	7 yrs	2.1%	NA
			NA	4 to 5.5 cm	7 yrs	4.6%	NA
			NA	5.6 cm or larger	7 yrs	20%	NA

NA = not available; UK = United Kingdom.

2. **Patients with infrarenal or juxtarenal AAAs measuring 4.0 to 5.4 cm in diameter should be monitored by ultrasound or 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 2 to 3 years is reasonable.** ***(Level of Evidence: B)***

Class III

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

Aneurysm size remains the single most important predictor not only for aneurysm rupture but also for unrelated death from other cardiopulmonary events (280,281). Data suggest that the eventual risk for rupture is approximately 20% for aneurysms larger than 5.0 cm in diameter, 40% for those at least 6.0 cm in diameter, and greater than 50% for aneurysms that exceed 7.0 cm in diameter (Table 25).

Conversely, the rupture rate for truly small aneurysms that are less than 4.0 cm in diameter is quite low, perhaps because aged patients with such small aneurysms ordinarily do not survive long enough for this complication to occur. Bengtsson et al. have recommended only 1 annual follow-up scan for aneurysms less than 3.5 cm in diameter, because the unrelated mortality rate in such patients is so high that relatively few live long enough to incur sufficient aneurysm growth to warrant elective surgical treatment (292). Prospective nonrandomized studies have indicated that small aneurysms may be safely monitored by annual or semiannual imaging scans with a low risk for rupture, provided elective repair is advised once a diameter of at least 5.0 cm has been documented (286, 293).

RANDOMIZED TRIALS. Prospective randomized trials comparing early intervention versus expectant observation for infrarenal abdominal aortic aneurysms measuring 4.0 to 5.4 cm in diameter have been conducted in the United Kingdom (UK) and by the U.S. Department of Veterans Affairs (VA) during the past decade (291,294–296). By protocol, elective surgical treatment was not offered to patients who were allocated to the nonoperative cohort in each trial until their aneurysms exceeded 5.4 cm on serial imaging studies. Selected data from both investigations are summarized in Table 26, using updated information from the UK trial at a mean follow-up interval of 8 years (296) compared with 4.6 years when its findings first were disclosed in 1998. Not surprisingly, the principal demographic difference between the 2 trials is the fact that whereas women accounted for 17% of the patients in the UK study, they represented only 0.8% of the VA population. Thirty-day operative mortality rates (UK, 5.4%; VA, 2.1%) were competitive with those from other multicenter studies. Endografts were used in 27 patients in the surgical limb of the UK trial (4.8%) but in just 2 patients in the VA trial.

At a mean of 4.9 years of follow-up, early aneurysm repair has produced no significant benefits with respect to the incidence of either aneurysm-related deaths or deaths due to all causes in the VA trial. These are the same conclusions that originally were reached at a mean follow-up of 4.6 years in the UK trial (290). Although the UK surgical cohort now has a lower overall mortality rate than the nonoperative cohort (p equals 0.03) at a mean follow-up of 8 years, this finding has been attributed in part to a higher rate of smoking cessation in the early-surgery group (296). The annual rupture rate was negligible (0.6%) for observed aneurysms in the VA trial and was 3.2% in the UK trial. Rupture was more likely to occur in women in the UK trial (odds ratio 4.0; 95% confidence interval [CI] 2.0 to 7.9; p less than 0.001), accounting for 14% of all deaths in women compared with 4.6% of all deaths in men (p less than 0.001). Aneurysm size at the time of randomization did not influence the risk for rupture in the UK trial or the long-term mortality rate in either trial, but this may reflect the promptness with which intervention was performed whenever aneurysms reached a diameter of at least 5.5 cm. More than 60% of the patients in the nonoperative limb of each of these trials currently have undergone aneurysm repair because of documented enlargement, including 81% of the patients whose aneurysms were 5.0 to 5.4 cm in diameter when they were recruited into the VA trial.

No randomized trial has yet addressed the size at which suprarenal, pararenal, or type IV thoracoabdominal aortic aneurysms should be repaired to prevent rupture. Because of their higher risk for postoperative death, renal insufficiency, and other surgical complications, however, there has been a consensus that elective intervention should be considered for these aneurysms at a slightly larger diameter than for infrarenal aortic aneurysms.

Table 26. Outcomes of Early Elective Repair Versus Nonoperative Surveillance of Asymptomatic Abdominal Aortic Aneurysms*

	UK Trial (2002)	VA Trial (2002)
Total patients	1090	1136
Early elective repair	563	569
Open	536	567
Endovascular	27	2
Nonoperative surveillance	527	567
Men	902	1127
Women	188	9
Age	69 plus or minus 4 years	68 plus or minus 6 years
Operative mortality rate (surgical cohorts)	5.4% (30 days)	2.1% (30 days); 2.7% (in-hospital)
Follow-up period	Range 6 to 10 years; mean 8 years	Range 3.5 to 8.0 years; mean 4.9 years
Survival rate		
Surgical cohort	57%	75%
Nonoperative cohort	52% (p equals 0.03)	78%
Aneurysm rupture rate (nonoperative cohorts)	3.2% Annually	0.6% Annually
Men	Odds ratio 1.0 (reference set)	NA
Women	Odds ratio 4.0; 95% CI 2.0 to 7.9 (p less than 0.001)	NA
Eventual aneurysm repair		
Surgical cohort	520 (92%)	527 (93%)
Nonoperative cohort	327 (62%)	349 (62%)
Influence of aneurysm diameter (nonoperative cohorts)		
Survival rate	4.0 to 4.4 cm: 57% 4.5 to 4.8 cm: 54% 4.9 to 5.5 cm: 43%	4.0 to 4.4 cm: 79% 4.5 to 4.9 cm: 78% 5.0 to 5.4 cm: 68%
Eventual repair rate	NA	4.0 to 4.4 cm: 27% 4.5 to 4.9 cm: 53% 5.0 to 5.4 cm: 81%

*Results of 2 prospective randomized trials conducted in the United Kingdom (UK) (290,296) and by the United States Department of Veterans Affairs (VA) (297).
CI = confidence interval; NA = not available.

B. COMMON ILIAC ANEURYSMS. Isolated common iliac aneurysms are unusual in the absence of a proximal aortic aneurysm, and comparatively little information is available with respect to their natural history. Approximately one third to one half of common iliac aneurysms are bilateral, and 50% to 85% are asymptomatic at the time of their discovery (298,299). According to a collective review of 3 clinical series, aneurysm rupture usually occurs at a diameter of 5.0 cm or larger, whereas common iliac aneurysms that are less than 3.0 cm in diameter almost never rupture (299). Therefore, isolated common iliac aneurysms that are smaller than 3.0 cm probably can be safely followed up with serial

noninvasive imaging. Contrast-enhanced CT scans or magnetic resonance imaging studies appear to be better suited for this purpose than ultrasonography because many common iliac aneurysms are situated deep in the pelvis.

4. Diagnosis.

A. SYMPTOMATIC AORTIC OR ILIAC ANEURYSMS.

RECOMMENDATIONS

Class I

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

Pain is the most frequent complaint in patients with symptomatic AAAs and usually is located in the hypogastrium or the lower part of the back. Pain is typically steady, lasting for hours to days at a time, and has a gnawing quality. Aneurysm pain is not affected by movement, although patients may be more comfortable in certain positions, such as with the knees flexed. Expansion and impending rupture are heralded by the development of new or worsening pain, characteristically constant, severe, and located in the back or lower part of the abdomen, sometimes with radiation into the groin, buttocks, or legs. Rupture is associated with abrupt onset of back pain, abdominal pain, and tenderness. Unless they are hypotensive because of blood loss, many patients with ruptured aneurysms have a palpable, pulsatile abdominal mass. It must be remembered, however, that the pathognomonic triad of abdominal/back pain, pulsatile abdominal mass, and hypotension occurs in only about one third of cases (300). The symptoms of a ruptured aneurysm may mimic those of renal colic, diverticulitis, or a gastrointestinal hemorrhage, thus leading to a misdiagnosis that can cost valuable time.

B. ASYMPTOMATIC AORTIC OR ILIAC ANEURYSMS. Patients with even small AAAs have a high prevalence of risk factors for and clinical manifestations of atherosclerotic cardiovascular disease. Up to 13% of patients with aortic aneurysms have multiple aneurysms elsewhere (301), and 25% to 28% of those with thoracic aortic aneurysms have concomitant AAAs (302,303). Accordingly, patients in whom an aortic aneurysm is discovered at either level should undergo an appropriate examination of the entire aorta to detect aneurysms in other locations.

C. PHYSICAL EXAMINATION. A comprehensive physical examination should include palpation of the abdomen and the lower extremity arteries in an attempt to detect widened pulses that suggest the presence of aneurysms. Palpation of AAAs is safe and has not been reported to precipitate rupture. Perhaps the best evidence regarding the accuracy of abdominal palpation comes from 15 studies of patients who were not previously known to have AAAs but were screened with both an abdominal examination and ultrasound scans (304). The pooled sensitivity of abdominal palpation increased significantly with aortic diameter (p less than 0.001), ranging from 29% for AAAs of 3.0 to 3.9 cm to 50% for AAAs of 4.0 to 4.9 cm and 76% for AAAs measuring 5.0 cm or more by ultrasonography. In a 3-year retrospective study of 198 patients with AAAs that was conducted by Alcorn et al. (305) in a general hospital setting, 48% of the aneurysms had been discovered clinically, 37% represented incidental findings during the radiographic investigation of another condition, and 15% were encountered during unrelated abdominal operations. Not surprisingly, the average size of palpable AAAs was larger than that of nonpalpable AAAs (6.4 plus or minus 1.2 cm vs. 4.9 plus or minus 1.4 cm, p less than 0.001).

D. SCREENING HIGH-RISK POPULATIONS.

RECOMMENDATIONS

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

2. **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)***

Aortic diameter can be measured accurately by ultrasound imaging in more than 97% of subjects (306,307). Screening by this method has the potential to reduce the incidence of aortic rupture. In a cohort of 52 745 military veterans aged 50 to 79 years who had no history of aneurysms, AAAs measuring 4.0 cm or more in diameter were detected by ultrasound screening in 613 participants (1.2%). When this cohort was combined with a similar cohort of 73 451 veterans in the same age range, the odds ratios for major risk factors were as follows: 1.71 per 7 years of age, 0.18 for female gender, 0.53 for black race, 1.94 for a family history of AAA, 5.07 for smoking, 0.52 for diabetes, and 1.66 for atherosclerotic diseases. The excess prevalence associated with smoking accounted for 75% of all AAAs 4.0 cm or larger in the combined population of 126 196 veterans.

In another population-based study, 67 800 men aged 65 to 74 years were randomly allocated to receive an invitation for an abdominal ultrasound scan (308). Men in whom aortic aneurysms at least 3.0 cm in diameter were detected were followed up with repeat scans for a mean of 4.1 years. Surgical treatment was considered when the diameter reached 5.5 cm, if expansion occurred at a rate of more than

1 cm per year, or if symptoms occurred. More than 27 000 (80%) of the 33 839 men in the invited group agreed to screening, and 1333 aneurysms were detected. There were 65 aneurysm-related deaths (absolute risk 0.19%) in the invited group and 113 (0.33%) in the control group (risk reduction 42%, 95% CI 22% to 58%, p equals 0.0002), including a 53% reduction of risk (95% CI 30% to 64%) among those who actually underwent screening. During the 4 years in which this trial was conducted, there were 47 fewer deaths related to AAAs in the screening group than in the control group, but the additional costs incurred were 2.2 million British pounds (approximately $3.5 million U.S. dollars). The hazard ratio for AAA was 0.58 (95% CI 0.42 to 0.78). Over 4 years, the mean incremental cost-effectiveness ratio for screening was 28 400£/$45 000 per life-year that was gained, a figure that is equivalent to about 36 000£/$57 000 per quality-adjusted life-year. After 10 years, this figure was estimated to decline to approximately 8000£/$12 500 per life-year gained (309).

Selected screening of populations with a high prevalence of AAA (e.g., males 60 years or older who have a family history of AAA, in whom the prevalence is approximately 18%) and the use of a limited ultrasound scan are more cost-effective than conventional abdominal imaging of unselected populations. The United States Preventive Services Task Force meta-analysis supports the concept that screening for AAA and surgical repair of large AAAs (5.5 cm or more) in men aged 65 to 75 years who have ever smoked (inclusive of both current and former smokers) leads to decreased AAA-specific mortality when abdominal ultrasonography is performed in a setting with adequate quality assurance (i.e., in an accredited facility with credentialed technologists). The data do not support the application of AAA screening for men who have never smoked or for women.

5. Observational Management.

A. BLOOD PRESSURE CONTROL AND BETA-BLOCKADE.

RECOMMENDATIONS

Class I

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

Class IIb

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

Perioperative administration of beta-adrenergic blockers reduces the risk of adverse cardiac events and death in patients who undergo AAA surgery (310,311). Long-term beta-adrenergic blockade has slowed the rate of thoracic aortic dilation and decreased the incidence of aortic complications in patients with Marfan syndrome. Retrospective studies have suggested that beta-adrenergic antagonist agents might reduce the risk of AAA expansion and rupture. One prospective randomized trial found that the expansion rate of AAAs was not attenuated by beta-adrenergic blockers.

B. FOLLOW-UP SURVEILLANCE. A number of prospective nonrandomized studies that were reported before the disclosures from the UK Small Aneurysm Trial and the VA Aneurysm Detection and Management (ADAM) Trial were made suggested annual ultrasound surveillance for aneurysms measuring less than 4.0 cm in diameter and ultrasound scans every 6 months for those 4.0 to 4.9 cm in diameter, with a recommendation for elective aneurysm repair in appropriate surgical candidates whenever an AAA reached a size of at least 5.0 cm. One such study of 99 patients documented a mean expansion rate of 2.2 mm in the first year of observation, 2.8 mm in the second year, and 1.8 mm in the third year for aneurysms that initially were smaller than 4.0 cm. The corresponding growth rates for aneurysms measuring 4.0 to 4.9 cm were 2.7, 4.2, and 2.2 mm (312). Given the usual slow rate of expansion for truly small aneurysms, however, some studies have recommended that those measuring less than 4.0 cm in diameter can safely be followed up with ultrasound scans every 2 to 3 years.

6. Open Aortic Aneurysm Repair. The management of patients who have AAAs that are sufficiently large to represent a predictable risk for fatal rupture is guided by several considerations. First, the survival rate of this patient population generally is acknowledged to be significantly lower than that of a normal population of the same age (313–316). Second, it has long been recognized that coronary artery disease and its consequences represent the leading causes of late death in these patients, superseding even the mortality rate that can be attributed directly to unoperated aneurysms (317,318). Therefore, in addition to their importance regarding early surgical risk, these observations have long-term implications with respect to the identification and treatment of underlying coronary disease before the elective repair of aortic aneurysms. Finally, the emergence of new technology for transfemoral endovascular repair of AAAs with a variety of commercially available U.S. Food and Drug Administration–approved stent grafts now provides an alternative to open surgical treatment in patients with aneurysms that warrant repair on the basis of their size or expansion rate. Thus, management of aortic aneurysms must be tailored to the individual patient.

A. INFRARENAL AAAS.

PREOPERATIVE CARDIAC EVALUATION. A number of studies have demonstrated that the perioperative and long-term mortality rates in conjunction with open aortic aneurysm repair are highest among patients who have symptomatic

coronary disease (i.e., Class III to IV angina pectoris or congestive heart failure), intermediate in those who have chronic stable angina and/or a history of remote MI, and lowest among those who have no indication of coronary disease whatsoever (319–323). Several large clinical series have reported that the mortality rate for open aortic aneurysm repair can be reduced to less than 2% in a setting in which approximately 5% to 15% of patients undergo preliminary coronary artery intervention (324–327). However, the role of revascularization in the context of contemporary medical management appears to be less than has been assumed traditionally. Intensive medical therapy and coronary revascularization (including percutaneous coronary intervention and coronary artery bypass grafting), when offered to individuals anticipated to undergo AAA repair or lower extremity revascularization surgery, had equal postoperative rates of cardiovascular ischemic events in a recent prospective investigation (328). A comprehensive discussion of this topic may be found in a previous guidelines document sponsored by the ACC/AHA (89).

Open Surgical Approaches. Open aortic aneurysm repair can be performed with a midline transabdominal approach or an extraperitoneal incision in the left flank. There is no clear consensus, however, regarding the superiority of either of these incisions on the basis of prospectively randomized studies.

Early Mortality and Complication Rates. In a collective review of nearly 40 000 reported cases, Blankensteijn et al. concluded that the operative mortality rate for elective open aortic aneurysm repair varied according to whether the individual case series were prospective or retrospective in design and whether they were population-based or hospital-based (329). Such factors undoubtedly account for some of the variability in the representative early outcomes that are summarized in Table 27. Mortality rates from single centers generally were in the range of 4% to 5% during the 1980s, whereas information that has been published during the 1990s contains several series in which the mortality rate has declined to less than 2%. In comparison, regional or multicenter studies in the U.S. and elsewhere generally have been associated with slightly higher mortality rates, ranging from 5% to 7%. The operative mortality rate for open repair of ruptured AAAs is uniformly grim, however, ranging from 40% to 70% regardless of whether it has been reported from single-center case series, collective reviews, regional or multicenter studies, or large national databases.

During the past 15 years, a growing number of studies have demonstrated an inverse relationship between the mortality rate for aortic aneurysm repair and both the annual hospital volume and the experience of individual surgeons with these procedures. Representative data showing these relationships for intact and ruptured aneurysms are summarized in Table 28. Other studies have reconfirmed these observations with respect to hospital volume (341,348), surgeon experience (336), or both (349). Manheim et al. (338) and Dimick et al. (347) have estimated that the operative mortality rate for elective aneurysm repair is reduced by approximately 50% in high-volume hospitals in the U.S., and Wen et al. (335) have calculated that there is a 6% reduction in the relative odds for death with every 10 additional elective cases that are added to the annual hospital volume in Ontario. Pearce et al. (340) discovered that a doubling of the annual surgeon volume was associated with an 11% reduction in the relative risk for death after aortic aneurysm repair in Florida, and Dardik et al. (339) have determined that hospital charges are significantly lower in conjunction with the repair of either intact or ruptured aortic aneurysms by high-volume surgeons in Maryland.

B. JUXTARENAL, PARARENAL, AND SUPRARENAL AORTIC ANEURYSMS. Aneurysms involving the upper abdominal aorta generally are classified according to their relationship to the renal arteries. Juxtarenal aneurysms arise distal to the renal arteries but in very close proximity to them; pararenal aneurysms involve the origin of 1 or both renal arteries; suprarenal aneurysms encompass the visceral aortic segment containing the superior mesenteric and celiac arteries, and specifically are termed type IV thoracoabdominal aneurysms if they extend upward to the crus of the diaphragm (352). Irrespective of the incision that is used for their exposure, the principal technical consideration that is common to most of these aneurysms is that they require a period of aortic cross-clamping above the renal arteries.

Juxtarenal aneurysms represent the only exception to the requirement for suprarenal aortic cross-clamping, because some of these aneurysms are associated with an adequate cuff of relatively normal aorta for proximal control just below the renal arteries. Even when suprarenal cross-clamping is required, it is only for the period of time that is necessary to construct the proximal anastomosis of the replacement graft near the uninvolved renal arteries. This feature undoubtedly accounts for the observation that operative mortality and morbidity rates for juxtarenal aortic aneurysms are higher than those for standard infrarenal aneurysms but lower than those for aneurysms that extend above the renal arteries.

Selected but representative data regarding the operative mortality and complication rates for all upper abdominal aortic aneurysms involving the renal arteries are presented in Table 29.

7. Endovascular Aortic Aneurysm Repair.

A. INTRODUCTION. Endovascular AAA repair can eliminate the need for a major transabdominal procedure, can be performed under regional or even local anesthesia, and clearly represents a major advance in the management of patients with AAAs who have severe cardiopulmonary disease or other risk factors, such as advanced age, morbid obesity, or a hostile abdomen from multiple previous operations. Since its feasibility had been demonstrated in such patients, endovascular repair also has been offered at many centers to low- or average-risk patients who have no

Table 27. Operative Mortality Rates for Open Repair of Intact Abdominal Aortic Aneurysms, Since 1995

First Author	Reference	Year (Study Period)	No. of Patients	Mortality Rate (%)
Case series				
Sicard	(330)	1995	145	1.4
Lloyd	(331)	1996 (1980–1995)	1000	2.4
Starr	(318)	1996 (1983–1989)	Men: 490	5.1
			Women: 92	4.3
			Total: 582	5.0
Aune	(315)	2001 (1985–1999)	Age less than 66 yrs: 118	1.7
			Age 66 yrs and older: 333	6.0
			Total: 451	4.9
Hertzer	(327)	2002 (1989–1998)	1135	1.2
Menard	(332)	2003 (1990–2000)	Low risk: 444	0.0
			High risk: 128	4.7
			Total: 572	1.0
Randomized trials				
UK Small Aneurysm Trial (surgical cohort)	(290)	1998	563	5.8
U.S. Veterns Affairs Small Aneurysm Trial (surgical cohort)	(297)	2002	569	2.7
Collective reviews				
Zarins	(333)	1997 (1987–1992)	2162	2.1
Blankensteijn	(329)	1998 (1985–1997)	Prospective population: 692	8.2
			Prospective hospital: 1677	7.4
			Retrospective population: 21 409	3.8
			Retrospective hospital: 12 019	3.8
			Subset analyses: 1857	3.5
Regional or multicenter studies				
Kazmers (Veterans Affairs)	(334)	1996 (1991–1993)	3419	4.9
Wen (Ontario Aneurysm Study)	(335)	1996 (1988–1992)	5492	3.8
Kantonen (Finland Vascular Registry)	(336)	1997	929	5.1
Koskas (French AURC)	(314)	1997 (1989)	1107	4.8
Bradbury (Edinburgh Vascular Registry)	(337)	1998 (1976–1996)	492	6.1
Manheim (California statewide)	(338)	1998 (1982–1994)	35 130	7.6
Dardik (Maryland statewide)	(339)	1999 (1990–1995)	2335	3.5
Pearce (Florida statewide)	(340)	1999 (1992–1996)	13 415	5.7
Sollano (New York statewide)	(341)	1999 (1990–1995)	9847	5.5
Kazmers (Veterans Affairs)	(342)	2001 (1991–1995)	5833	4.5
Axelrod (Veterans Affairs)	(343)	2001 (1997–1998)	1001	3.7
U.S. hospital databases				
Lawrence (National Hospital Discharge Survey)	(344)	1999 (1994)	32 387	8.4
Heller (National Hospital Discharge Survey)	(345)	2000 (1979–1997)	358 521	5.6
Huber (Nationwide Inpatient Sample)	(346)	2001 (1994–1996)	16 450	4.2
Dimick (Nationwide Inpatient Sample)	(347)	2002 (1996–1997)	13 887	3.8

AURC = Association for Academic Research in Vascular Surgery; UK = United Kingdom.

particular contraindications to conventional surgical treatment. This has resulted in a distinct shift in the paradigm for management of infrarenal aortic aneurysms in some geographic areas during a relatively short period of time. According to statewide data from New York, for example, 53% of patients who underwent AAA repair received endografts in 2002 compared with 40% in 2001 (363).

Most contemporary stent grafts are supported by a metallic skeleton that is secured to the fabric of the graft during the manufacturing process to maintain linear stability once the device has been implanted and to avoid kinking that can result in graft limb occlusion with unsupported grafts. To better accommodate the aortoiliac anatomy and facilitate graft deployment, the majority of modern endografts also are modular in construction. The absence of an adequate length of relatively normal aorta below the renal arteries historically has excluded patients from consideration for endovascular repair because of the high risk for proximal attachment failure, graft migration, and endoleak.

In an attempt to overcome the risk of distal migration and proximal attachment failure, a growing number of new devices now incorporate barbed hooks that are sufficiently long to secure the metallic frame of the stent graft to the visceral segment of the aorta above the renal arteries. In aggregate, modular externally supported bifurcation endografts are more widely applicable, less prone to migrate

Table 28. Volume/Outcome Relationships for Open Abdominal Aortic Aneurysm Repair in the United States Since 1990

First Author	Reference	Year (Study Period)	No. of Patients	Overall Mortality Rate (%)	Annual Volume	
					Hospital	Surgeon
Intact aneurysms						
Hannan (New York statewide)	(350)	1992 (1982–1987)	6042	7.6	Low: 12%; medium: 6.8%; high: 5.6%	Low: 11%; medium: 7.3%; high: 5.6%
Katz (Michigan statewide)	(351)	1994 (1980–1990)	8185	7.5	Low: 8.9%; high: 6.2% (p less than 0.001)	NA
Kazmers (Veterans Affairs)	(334)	1996 (1991–1993)	3419	4.9	Low: 6.7%; high: 4.2% (p less than 0.05)	NA
Dardik (Maryland statewide)	(339)	1999 (1990–1995)	2335	3.5	Low: 4.3%; medium: 4.2%; high: 2.5% (p equals 0.08)	Very low: 9.9%; low: 4.9%; medium: 2.8%; high: 2.9%
Ruptured aneurysms						
Katz (Michigan statewide)	(351)	1994 (1980–1990)	1829	50	Low: 54%; high: 46% (p equals 0.0026)	NA
Dardik (Maryland statewide)	(339)	1998 (1990–1995)	527	47	Low: 46%; medium: 49%; high: 47% (p equals NS)	Low: 51%; medium: 47%; high: 36% (p equals 0.05)

NA = not available; NS = nonsignificant.

from their sites of attachment, and more likely to remain patent than was the case with the first generation of unsupported endografts only a few years ago. Some aspects of endovascular aneurysm repair remain problematic, however, and will require further refinements in the future. In addition to the vexing problem of metal fatigue (364,365), these include anatomic limitations and intrasac endoleaks.

Anatomic Limitations. Because of the inflexibility of externally supported grafts, this segment of the aorta must not be severely angulated. This requirement may impose a gender bias in patient selection because, in addition to the fact that their small external iliac arteries often present problems with respect to vascular access, women also appear to have a higher prevalence of short, angulated aneurysm necks than

Table 29. Operative Mortality and Postoperative Complication Rates for Open Repair of Pararenal, Suprarenal, and/or Type IV Thoracoabdominal Aortic Aneurysms, Since 1990

First Author	Reference	Year (Study Period)	No. of Patients	Mortality Rate (%)	Postoperative Complication Rate (%)		
					Renal	Paraplegia	Other
Pararenal or suprarenal							
Nypaver	(353)	1993 (1985–1992)	53	3.8	Transient: 23; dialysis: 5.7	NA	NA
Faggioli	(354)	1998	50	12	NA	NA	NA
Jean-Claude	(355)	1999 (1977–1997)	257	5.8	Transient: 30; sustained: 9.3; dialysis: 7.0	0.4	31
Anagnostopoulos	(356)	2001 (1986–1999)	65	0	Total: 42; dialysis: 9.2; permanent: 1.5	0	NA
Type IV thoracoabdominal							
Cox	(357)	1992 (1966–1991)	42	Total: 31; elective: 12; urgent: 55	NA	Total: 11; elective: 4.3; urgent: 20	NA
Svensson	(358)	1993 (1960–1991)	346	5.8	Total: 22	4.3	NA
Coselli	(359)	1995 (1984–1993)	35	14 (Reoperations)	None permanent	2.9	NA
Schwartz	(360)	1996 (1977–1994)	58	5.3	Transient: 31; sustained: 28; dialysis: 8.8; permanent: 1.9	1.8	42
Dunning	(361)	1999 (1995–1998)	26	12	Dialysis: 3.8	3.8	42
Martin	(362)	2000 (1989–1998)	165	Total: 11; elective: 7.2; urgent: 22	Transient: 19; dialysis: 14; permanent: 3.0	3.6	56

NA = not available.

men (366,367). Considering all of these criteria, Carpenter et al. reported that a disproportionate number of women were excluded from endograft repair because of anatomic limitations (60% of women vs. 30% of men; p equals 0.0009) (368). Becker et al. (369) also found that significantly fewer women qualified for endovascular aneurysm repair (26% of women vs. 41% of men), and Mathison et al. (370) were forced to abandon more attempted endograft procedures in women (17%) than in men (2.1%, p less than 0.01). Wolf et al. described comparable eligibility rates for endograft repair in women (49%) and men (57%), but the women in this series had a higher incidence of intraoperative complications than men (31% vs. 13%, p less than 0.05) and required more adjunctive arterial reconstructions (42% vs. 21%, p less than 0.05) to correct those complications (371).

Intrasac Endoleaks. Type I endoleaks are caused by incompetent proximal or distal attachment sites, produce high intrasac pressure that can lead to rupture, and should be repaired with intraluminal extender cuffs or conversion to an open procedure as soon as they are discovered. Type II endoleaks are the result of retrograde flow from branch vessels (e.g., lumbar arteries and the inferior mesenteric artery), occur in as many as 40% of patients at some point in time after endograft implantation, and often may be corrected by selective arterial catheterization and therapeutic embolization. More than half of type II endoleaks will seal spontaneously, however, and although isolated examples of aneurysm rupture on the basis of persistent type II endoleaks have been reported (372,373), they do not yet appear to influence the risk for rupture during 18 to 36 months of surveillance in large series of patients (374,375). Type III endoleaks are caused by midgraft defects from fabric tears or the junctional disruption of modular graft components, especially if these components are buckled as the excluded aneurysm sac shrinks and foreshortens. Type III endoleaks are considered to have the same potential for delayed aneurysm rupture as type I endoleaks and therefore should be repaired promptly at the time of their discovery. Type IV endoleaks are the result of high graft porosity and diffuse leakage through its interstices, usually occur within 30 days of implantation, and are rare compared with the frequency of other endoleaks. Finally, the term "endotension" has been applied to those circumstances in which the excluded sac continues to enlarge and appears to remain pressurized despite the absence of any visible endoleaks on contrast-enhanced CT scans.

In summary, it is largely because of the uncertainties related to intrasac endoleaks that clinical investigators and the Food and Drug Administration consider follow-up imaging to be mandatory every 6 to 12 months for any patient whose aortic aneurysm is treated with an endovascular stent graft (376,377).

B. PREOPERATIVE CARDIAC EVALUATION. The preoperative cardiac evaluation before endovascular aneurysm repair may be dictated by patient selection, because severe cardiac disease already will have been documented in many patients who are treated at centers where endografting is restricted to high-risk cases. Perhaps for this reason, relatively little published information is available on this topic. On the basis of admittedly incomplete data, elective endovascular aortic aneurysm repair in unselected patients probably should be considered as an "intermediate or low surgical risk procedure" according to the previous ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery (89).

C. EARLY MORTALITY AND COMPLICATION RATES. Table 30 contains representative data regarding the procedural mortality rate for endovascular aneurysm repair, the incidence of early endoleaks, and the risk for immediate conversion to an open operation. This information has been collected from case series, from Food and Drug Administration- and industry-sponsored device trials in the U.S., and from the European collaborators registry on stent-graft techniques for abdominal aortic aneurysm repair (EUROSTAR), a cooperative archive for endograft data that are submitted voluntarily by nearly 60 participating centers. The study periods for the references that are cited in Table 30 help to identify the generation of devices that were under investigation, and they also provide points of reference during an era in which rapid advances in technology tend to make the preceding iteration of stent grafts and delivery systems obsolete as soon as new devices are introduced.

The early mortality rate for endograft repair generally has been less than 3%, but May et al. (385) have shown this to be substantially lower than the mortality rate for a concurrent series of open procedures. The comparative safety of endograft repair is difficult to assess because it often is difficult to determine from published reports whether aortic stent grafts were offered only to high-risk surgical patients or to a mix of high-risk, average-risk, and low-risk patients. Several EUROSTAR studies have demonstrated that both early mortality rates and nonfatal complication rates were significantly higher among patients who were deemed to be unfit for open repair or general anesthesia (396,399,401), as well as among those who needed adjunctive procedures in addition to the placement of an aortic stent graft (396). Consequently, the perceived margin of safety for endovascular aneurysm repair in truly high-risk candidates may be slightly overestimated by results from nonuniform patient populations. Irrespective of case mix, however, the comparatively low early mortality rate for endograft repair of aortic aneurysms in New York State deserves close attention. According to data reported by Anderson et al., the mortality rate for endograft procedures was significantly lower than for open procedures in New York during both 2001 (1.1% vs. 3.6%, p equals 0.0018) and 2002 (0.8% vs. 4.2%, p less than 0.0001) (363).

Table 30. Representative Early Results for Endovascular Repair of Infrarenal Abdominal Aortic Aneurysms Since 2000

First Author (Study/Sponsor)	Reference	Year (Study Period)	No. of Patients	Immediate Open Conversion (%)	Endoleaks (%)		Procedural Mortality Rate (%)
					Total	Persistent	
Case series							
Becquemin	(378)	2000 (1995–1999)	Endo: 73; Open: 195	None	23	9.6	2.7
Chuter	(379)	2000 (1996–1999)	High risk: 116	None	NA	10	1.7
Zarins	(380)	2000 (1996–2000)	149	1.30	36	18	1.3
Blum	(381)	2001 (1994–2001)	1994–1996; 111 1996–1997: 159 1998–2001: 28	3.6 (1994–1996) 0.6 (1996–1997) None (1998–2001)	14 (1994–1996) 3.1 (1996–1997) 11 (1998–2001)	NA	Total: 8.1
Becker	(369)	2001 (1994–2001)	305	1.30	23	17	2.6
Fairman	(382)	2001 (1998–1999)	75	None	44	20	0
Holzenbein	(383)	2001	173	1.2	4.6 (Type I)	NA	2.8
Howell	(384)	2001	215	None	42	11	0
Mathison	(370)	2001 (1994–2000)	305	1.3	23	NA	2.6
May	(385)	2001 (1995–1998)	Endo: 148; Open: 135	0.7	6.8	5.4	2.7 (Endo); 5.9 (Open)
Sicard	(386)	2001 (1997–2000)	Endo: 260; Open: 210	0.8	13	3	1.9 (Endo); 2.9 (Open)
Abraham	(373)	2002 (1998–2001)	116	None	15	11	0.9
Dattilo	(387)	2002 (1994–2000)	362	1.40	NA	NA	1.5
Sampram	(388)	2003 (1996–2002)	703	NA	NA	NA	1.7
Ouriel	(389)	2003 (1996–2002)	606 Men; 98 Women	NA	NA	NA	1.3 (Men); 3.1 (Women)
Shames	(390)	2003 (1999–2001)	302 Men; 42 Women	0.5 (Men); 14 (Women)	NA	NA	1.5 (Men); 2.3 (Women)
Anderson (New York State)	(363)	2004 (2000–2002)	Endo: 1,706; Open: 3,063	NA	NA	NA	1.1 (Endo); 4.0 (Open)
Device trials							
Zarins (AneuRx, Medtronic)	(391)	2000 (1997–1998)	425	1.20	Centers 38; core lab 50	13	1.4
Beebe (Vanguard, Boston Scientific)	(392)	2001 (1997–1998)	Endo: 268; Open: 98	1.90	5.70	2.7	1.5 (Endo); 3.1 (Open)
Greenberg (Zenith, Cook)	(393)	2001 (1995–2000)	528	0.80	16	5.5	0.2
Faries (Talent, Medtronic/ AVE-WorldMedical)	(394)	2002 (1999–2001)	368	1.10	12	4.8	1.9
Matsumura (Excluder; WL Gore & Associates)	(395)	2003 (2000–2002)	Endo: 235; Open: 99	None	22	17	0.9 (Endo); 0 (Open)
EUROSTAR registry							
Buth	(396)	2000 (1994–1999)	1554	1.70	16	0.9	2.6
Harris	(397)	2000 (1996–2000)	2464	1.30	17	8.3	3.2
Vallabhaneni	(398)	2001 (1994–2000)	2812	NA	NA	NA	2.9
Buth	(399)	2002 (1996–2001)	3075	1.70	17	NA	2.5
Peppelenbosch	(400)	2004 (1996–2002)	1962 (4.0 to 5.4 cm); 1528 (5.5 to 6.4 cm); 902 (over 6.4 cm)	1.1 (4.0 to 5.4 cm); 1.4 (5.5 to 6.4 cm); 2.3 (over 6.4 cm)	3.7 (4.0 to 5.4 cm); 6.8 (5.5 to 6.4 cm); 9.9 (over 6.4 cm), all Type I	NA	1.6 (4.0 to 5.4 cm) 2.6 (5.5 to 6.4 cm); 4.1 (over 6.4 cm)

Endo = endovascular repair; EUROSTAR = European collaborators registry on stent-graft techniques for abdominal aortic aneursym repair; NA = not available.

Immediate conversion to an open operation presently is necessary in only 1% of patients, and approximately half of all early endoleaks appear to resolve spontaneously within a period of 30 days. Several reports have indicated that endovascular procedures have fewer early complications than open operations, require less intensive care, and are associated with correspondingly shorter lengths of stay in the hospital (402–404). Nevertheless, these and other studies (405–407) also have suggested that the total costs of endovascular repair probably exceed those for open repair, especially when the expense of subsequent follow-up imaging, further intervention, and secondary hospital admissions is added to the base cost ($6000 to $12 000 U.S. dollars) of most endografts. Despite the shorter length of stay and earlier return to normal activity associated with aortic endografting, this procedure does not appear to be associated with superior late functional outcome or longer quality-adjusted life expectancy compared with open surgical treatment (408,409).

Technical Success Rates. The technical success rate is a useful way to express endograft results because it condenses a number of events into a single outcome value that ordinarily is calculated with the life-table method. Table 31 summarizes the early and intermediate-term technical suc-

Table 31. Technical Success Rates For Endograft Repair of Infrarenal Abdominal Aortic Aneurysms, Since 2000

First Author (Device/Vendor)	Reference	Year (Study Period)	No. of Patients	Criteria for Technical Success	Technical Success Rate	
					Early	Late
Case series						
Becquemin	(378)	2000 (1995–1999)	Endo: 73; Open: 107	No endoleaks; no reintervention		74% (p equals 0.001); 94% (1 yr)
Chuter	(379)	2000 (1996–1999)	High risk: 116	Successful deployment; no endoleaks	86% (2 weeks)	
Howell	(411)	2000	56	NA		83% Primary; 85% secondary (6 months)
Blum	(381)	2001 (1994–2001)	111 (1994–1996); 159 (1996–1997); 28 (1998–2001)	Successful deployment; no endoleaks	82% (1994–1996); 96% (1996–1997); 89% (1998–2001)	
Ohki	(412)	2001 (1992–2000)	239	Successful deployment; no endoleaks	89%	
Device trials						
Zarins (AneuRx/Medtronic)	(380)	2000 (1997–1998)	398	Survival free of aneurysm rupture, open conversion, or reintervention for endoleaks or graft thrombosis		88% (18 months)
Beebe (Vanguard/Boston Scientific)	(392)	2001 (1997–1998)	240	Successful deployment; no endoleaks; graft patent; no deaths	89% (30 days)	
Criado (Talent/Medtronic WorldMedical)	(413)	2001 (1997–2001)	High risk: 127; Low risk: 151	Successful deployment; no endoleaks	High risk: 86% (96% at 30 days); low risk: 88% (97% at 30 days)	
EUROSTAR						
Buth	(396)	2000 (1994–1999)	1554	Successful deployment; no endoleaks; no deaths	72% (30 days)	
Laheij	(414)	2000 (1996–1999)	1023	Freedom from any secondary intervention		1 yr; 89%; 3 yrs; 67%; 4 yrs; 62%

Endo = endovascular repair; EUROSTAR = European collaborators registry on stent-graft techniques for abdominal aortic aneurysm repair; NA = not available.

cess rates from 10 recent reports. These data suggest that longer follow-up will be necessary to determine the relative merit of endovascular repair compared with open operations for AAAs. In comparison, the technical success rate for endograft repair of isolated iliac aneurysms appears to be quite favorable according to the scant follow-up information that is available. Scheinert et al. (410) described a series of 53 such aneurysms in 48 patients with successful endograft deployment in 98%, no persistent or secondary endoleaks, and patency rates of 95% and 88% at 3 and 4 years of follow-up, respectively.

8. Prevention of Aortic Aneurysm Rupture. Aside from their infrequent other complications (e.g., peripheral or visceral embolism, aortocaval or primary aortoenteric fistula), the single most compelling reason to repair AAAs is to prevent fatal rupture. The first step in this process is to identify the presence of these aneurysms, beginning with a thorough physical examination or their recognition as an incidental finding on unrelated abdominal imaging studies. This is especially important in certain high-prevalence populations, such as those with known popliteal aneurysms or a family history of aortic aneurysms. The next step is to establish, on the basis of ultrasonography or CT/magnetic resonance scanning, whether a particular aortic aneurysm already is large enough to warrant intervention or instead should be placed under periodic surveillance to determine its rate of expansion. Ultimately, once an infrarenal aortic aneurysm reaches an appropriate size for graft replacement, a choice must be made between a traditional open operation or endovascular repair. Like all other aspects of aneurysm management, this decision requires a balanced judgment of relative risks.

A. MANAGEMENT OVERVIEW.

RECOMMENDATIONS

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)***

An overview of the management of AAAs is depicted in Figure 12. This algorithm incorporates the results of the randomized UK and VA trials and takes into account the still relatively limited information that is available regarding the long-term outcome of endograft repair for infrarenal aneurysms. It must be conceded from the outset that there could be honest scientific disagreement regarding a few of the recommended pathways that are illustrated in this algorithm. Some clinicians may think that infrarenal aneurysms should continue to be repaired at a size of only 5.0 cm, whereas others could believe that the conclusions of the UK and VA trials are not directly applicable to aortic aneurysms that involve the renal arteries and that these aneurysms should be even larger than 5.5 cm in diameter before elective surgical treatment is advised, to warrant its additional risks. In addition, there undoubtedly are many who consider the present technology of endovascular repair to be at a state of development that justifies its general use in low-risk and average-risk patients and in those who appear to be at high risk for conventional open operations. There is nothing unfavorable about its early safety to discourage this opinion. As an example from northern California and Nevada, proctored endovascular aneurysm repair was undertaken at 22 community hospitals in a series of 257 patients, only 29% of whom had medical contraindications to conventional operations, with 2 immediate open conversions and a 30-day mortality rate of 1.2% (415). However, this report shares the current liability of many studies concerning aortic stent grafts; the mean follow-up period for these patients is only 9.6 months, during which another 8% of them have required reintervention.

C. Visceral Artery Aneurysms

RECOMMENDATIONS

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)***

Visceral aneurysms are insidious because they usually cannot be detected by physical examination, are easily overlooked on plain roentgenograms unless mural calcification is present, and occur so infrequently that they may not be fully appreciated during incidental CT/magnetic resonance imaging scanning. Not surprisingly, therefore, several studies have indicated that approximately half of all visceral artery aneurysms present with rupture (Table 32). In comparison, spontaneous rupture appears to be an unusual event for renal artery aneurysms, possibly because exceptionally large renal artery aneurysms may be discovered on the basis of nonacute symptoms, such as hypertension or hematuria. Although rare under any circumstances, both visceral and renal artery aneurysms most commonly occur in multiparous women (416,417). Furthermore, some studies have suggested that the incidence of splenic artery aneurysms is particularly high among patients who have portal hypertension or a history of previous liver transplantation (418–420). The mortality rate for surgical repair of ruptured visceral aneurysms is sufficiently ominous (25% or higher) that patients who have these risk factors probably should be investigated for visceral artery aneurysms in the presence of unexplained abdominal symptoms.

1. Management Options. An array of open surgical and laparoscopic approaches has been reported for visceral artery aneurysms, with varying mortality rates depending on the clinical setting. Percutaneous catheter-based therapy with coil embolization leading to thrombosis of visceral aneurysms has been described for elective patients and for those who present with acute rupture. The technical success rate for these nonsurgical options ranges from 67% to 100%, with few fatalities or complications (250,426,427). One concern that should be recognized related to the catheter-based management of visceral artery aneurysms is the limited ability to assess the end organ after aneurysm treatment. This is in contrast to open surgical visceral artery aneurysm repair, in which the end organ may be visualized and assessed.

D. Lower Extremity Aneurysms

1. Etiology. As illustrated in Figures 20 and 21 of the full-text guideline, the diameters of peripheral arteries increase approximately 20% to 25% between the ages of 20 and 70 years (260,428). Coexistent AAAs have been reported in 85% of patients with femoral aneurysms (429) and

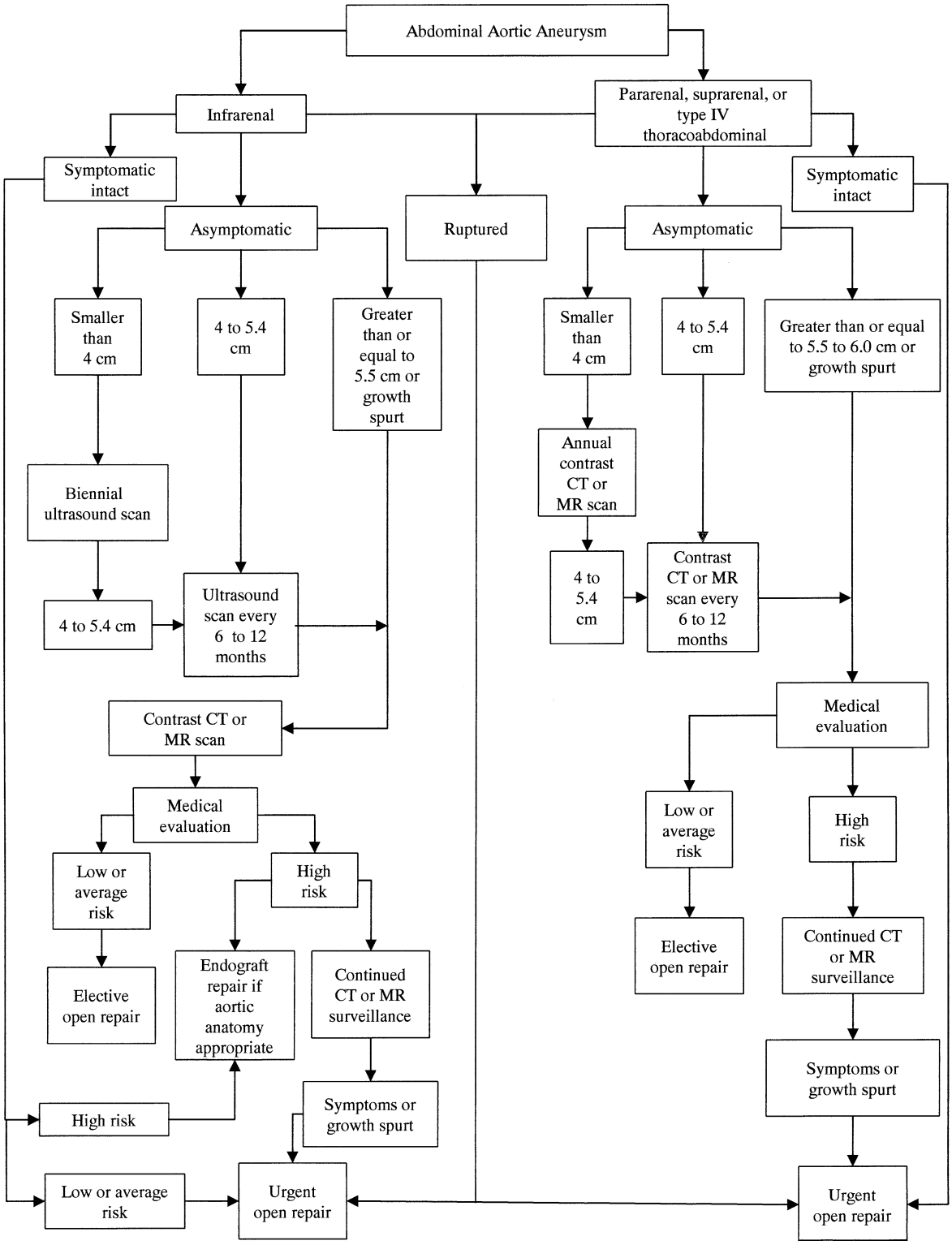

Figure 12. Management of abdominal aortic aneurysms. CT = computed tomography; MR = magnetic resonance.

in 62% of those with popliteal aneurysms (430), whereas femoral or popliteal aneurysms are present in 3% to 7% of patients who have AAAs.

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

Table 32. Presentation and Mortality Rates for Visceral Artery Aneurysms

First Author	Reference	Year	Patients and/or Aneurysms, n	Symptomatic and/or Ruptured on Presentation	Initial Treatment	Complications With Observation Alone	Mortality Rate
All visceral							
Carmeci	(421)	2000	31 (20 Women)	74%	Open: 25; endo: 9	NA	3%
Carr	(422)	2001	26/34	Ruptured: 42%	Open: 19	14% Ruptured	Total: 12%; ruptured: 25%
Splenic							
Trastek	(416)	1982	100 (87 Women)	17%; 3% ruptured	Open: 81	None at 7.4 yrs	1%
Lee	(420)	1999	34 (21 Women)	Ruptured: 44%	Open: 34	NA	Elective: 0%; ruptured: 40%
Superior mesenteric							
Stone	(423)	2002	21 (7 Women)	52%; 38% ruptured (50% of men)	Open: 13; endo: 3	None (mean, 1.8 cm)	Elective: 0%; ruptured: 38%
Renal							
Tham	(424)	1983	83/89	None	Open: 14	None	0%
Henriksson	(425)	1985	21/34 (16 Women)	None	Open: 8	None	0%

NA = not available.

RECOMMENDATION

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)*

A. POPLITEAL ARTERY ANEURYSMS. Popliteal aneurysms account for 70% of all aneurysms in the lower extremities and have an estimated incidence of 0.1% to 2.8% (431,432). Approximately 5% of small aortic aneurysms are discovered because of lower extremity ischemia caused by distal embolization of mural thrombus (433). However, thromboembolic complications are much more common with popliteal aneurysms, which often are bilateral and also may be associated with aneurysms involving the aorta and the femoral and superficial femoral arteries (Table 33).

The unfavorable consequences of popliteal aneurysms suggest that even when asymptomatic with good distal runoff, they should be repaired electively, although there is a lack of prospective studies to support an unqualified recommendation in this regard, especially for aneurysms measuring less than 2.0 cm in diameter. In fact, there is a published consensus that small popliteal aneurysms rarely become symptomatic and that elective surgical intervention should be considered only for those measuring at least 2.0 cm in diameter (431,440,441).

B. FEMORAL ARTERY ANEURYSMS. Femoral artery aneurysms may be discovered incidentally as a pulsatile mass in the thigh, or they may present with distal ischemia, and even more rarely, with rupture and bleeding.

3. Management.

RECOMMENDATIONS

Class I

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

Class IIa

1. **Surveillance by annual ultrasound imaging is suggested for patients with asymptomatic femoral artery true aneurysms smaller than 3.0 cm in diameter. *(Level of Evidence: C)***
2. **In patients with acute ischemia and popliteal artery aneurysms and absent runoff, catheter-directed thrombolysis or mechanical thrombectomy (or both) is suggested to restore distal runoff and resolve emboli. *(Level of Evidence: B)***
3. **In patients with asymptomatic enlargement of the popliteal arteries twice the normal diameter for age and gender, annual ultrasound monitoring is reasonable. *(Level of Evidence: C)***
4. **In patients with femoral or popliteal artery aneurysms, administration of antiplatelet medication may be beneficial. *(Level of Evidence: C)***

A. POPLITEAL ANEURYSMS. A popliteal mass should be studied by duplex ultrasonography to distinguish an

Table 33. Presentation and Complication Rates for Popliteal Aneurysms, Since 1990

First Author	Reference	Year	Patients and/or Aneurysms, n	Bilateral Popliteal or Other Aneurysms	Symptoms Before Presentation	Initial Surgical Treatment	Complications With Observation Alone	Related Amputation Rate
Case series								
Dawson	(434)	1991	50/71	42% Bilateral; 32% other	NA	65%	54%	NA
Carpenter	(435)	1994	33/54	62% Bilateral; 61% other	61%; 39% Ischemic	83%	NA	11%
Dawson	(436)	1994	42/42	NA	All asymptomatic	None	60%	7%
Lowell	(437)	1994	106/161 (103 Men)	52% Bilateral	42%	31%	22%	7%
Schroder	(438)	1996	217/349	61% Bilateral	45%	63%	47%	NA
Duffy	(431)	1998	24/40 (23 Men)	66% Bilateral	58%	75%	None (smaller than 2 cm)	None
Collective reviews								
Dawson	(439)	1997	1673/2445 (95% Men)	50% Bilateral; 37% other	67%	NA	36%	NA

NA = not available.

aneurysm from other soft-tissue lesions, especially if the patient has a history of other arterial aneurysms involving the contralateral lower extremity or the abdominal aorta. Nonoperative observation with periodic noninvasive surveillance may be appropriate if the aneurysm measures less than 2.0 cm in diameter or contains no thrombus or if the patient is at high surgical risk or has limited longevity because of medical comorbidities. If symptoms develop or the aneurysm enlarges on follow-up duplex scans, the risk of thromboembolic complications and limb loss then must be weighed against whatever factors originally may have influenced the decision to postpone surgical treatment.

In the setting of acute ischemia related to popliteal artery aneurysm thrombosis or thromboembolism, catheter-directed thrombolytic therapy is useful to reestablish patency of the popliteal and tibial trunks, which allows for more effective definitive aneurysm treatment and limb salvage. Largely because of previous and often unrecognized emboli, one of the obstacles to a successful surgical outcome is the absence of adequate arterial outflow in the calf and foot. Because limb salvage rates can be correlated directly with the number of available runoff vessels, as much thrombus as possible must be cleared from the tibioperoneal and plantar arteries in conjunction with bypass grafting to exclude the popliteal aneurysm from the circulation. In the past, this has been done strictly with thromboembolectomy balloon catheters in the operating room, often after preoperative arteriograms or MRA scans have failed to determine whether a target vessel for revascularization even is present. Some series now have been reported, however, in which preoperative intra-arterial thrombolytic therapy has been a valuable adjunct for restoring runoff in the presence of recent thromboembolic events (270,436,437,442).

The algorithm presented in Figure 13 summarizes the management options for either symptomatic or asymptomatic popliteal aneurysms. In the presence of mural thrombus, the diameter of a popliteal aneurysm will appear to be smaller on an arteriogram than its true diameter on duplex ultrasound or CT imaging, but the value of an arteriogram is to determine the adequacy of tibioperoneal outflow and whether the use of catheter-directed thrombolytic therapy should be considered to restore runoff. The decision to proceed with elective surgical treatment in the absence of limb-threatening ischemia is not predicated on aneurysm size alone. It must also take into account the overall clinical situation, the severity of symptoms in the leg, and the surgical or endovascular facilities that are available.

B. FEMORAL ANEURYSMS. The cause of femoral artery aneurysms may be arterial degeneration (i.e., true aneurysms) or false aneurysms related to previous vascular reconstructions or arterial injury. Femoral artery pseudoaneurysm represents a pulsatile mass that is contained by incomplete elements of the arterial wall and surrounding subcutaneous/fibrous tissue and may result from disruption of a previous femoral suture line, femoral artery access for a catheter-based procedure, or injury resulting from puncture due to self-administered drug abuse. Regardless of the cause, a pulsatile groin mass should be evaluated by duplex ultrasound and/or contrast-enhanced CT scan. The clinical presentation of true femoral artery aneurysms is summarized in Table 34 (443). Most reports encourage a policy of elective surgical treatment for symptomatic patients if their operative risk is low and the patient has a reasonable life expectancy. In 2 series, however, nonoperative observation has been used twice as often as elective intervention for asymptomatic femoral aneurysms and appears

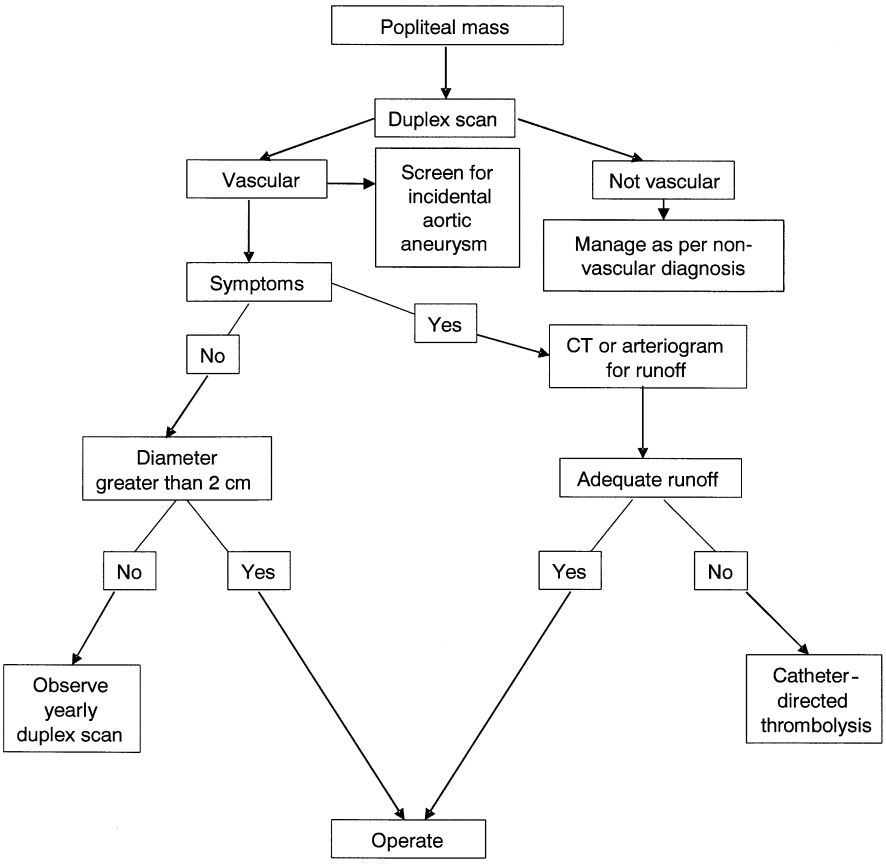

Figure 13. Diagnostic and treatment algorithm for popliteal mass. CT = computed tomography.

to be associated with a relatively low risk for complications during follow-up periods of 28 to 52 months (393,444). Therefore, the stable femoral artery aneurysm presents a therapeutic dilemma, because its complication rate appears to be substantially lower than that for popliteal aneurysms of similar size. A wide range of normal dimensions makes it difficult to determine an arbitrary size at which true femoral aneurysms should be repaired. By convention, femoral aneurysms measuring 3.0 cm or larger appear most likely to cause compressive symptoms and therefore also are most likely to be treated surgically. Although the presence of mural thrombus

Table 34. Clinical Presentation of Femoral Aneurysms

First Author	Reference	No. of Patients	Aneurysms (n)	Male: Female (n)	Bilateral (%)	AAA/PAA Associated (%)	Asymptomatic (%)	Presenting Symptoms	Complications at Presentation
Cutler	(445)	45	63	40:5	47	51/27	29	Local: 29%	Acute thrombosis: 16%; chronic thrombosis: 16% rupture: 14%
Adiseshiah	(446)	16	27	15:1	62	25/31	70		Embolization: 4% thrombosis: 7% rupture: 15%
Baird	(447)	30	36	30:0	20	40/17	27	Local: 23% ischemic: 50%	Acute thrombosis/ embolization: 13%; rupture: 0%
Graham	(429)	100	172	100:0	72	85/44	40	Local pain: 11% mass: 16% venous: 8% ischemic: 42%	Embolization: 8% acute thrombosis: 1% chronic thrombosis: 1% rupture: 2%
Sapienza	(448)	22	31	21:1	41	50/—	64	Local: 5% ischemic: 35%	

Reprinted from Vascular Surgery (5th ed), Graham L, Femoral and popliteal aneurysms, 1345–56, Copyright 2000, with permission from Elsevier (443).
AAA = abdominal aortic aneurysm; PAA = popliteal artery aneurysm.

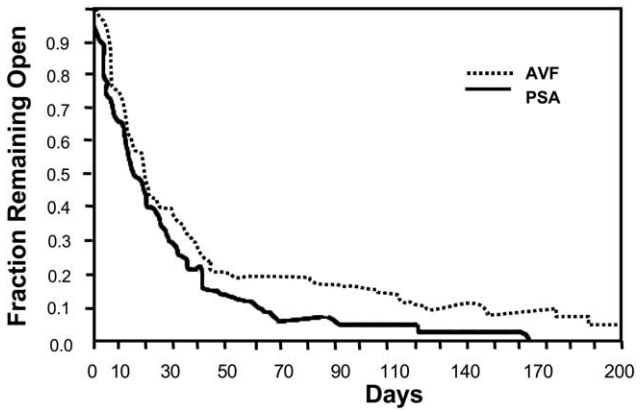

Figure 14. Spontaneous closure rates of selected pseudoaneurysms. Reprinted from J Vasc Surg, 25, Toursarkissian B, Allen BT, Petinec D, et al., Spontaneous closure of selected iatrogenic pseudoaneurysms and arteriovenous fistulae, 803–8, Copyright 1997, with permission from Elsevier (448a). AVF = arteriovenous fistula; PSA = pseudoaneurysm.

conceivably could represent a risk for distal emboli unless elective repair is performed, the actual magnitude of this risk is unknown.

Anastomotic pseudoaneurysms occur with an incidence of 2% to 5%, are encountered most commonly as a late complication of synthetic aortofemoral bypass grafting, inevitably continue to enlarge if left untreated, and may require arteriography before repair. Infected femoral pseudoaneurysms may occur as the result of arterial puncture during drug abuse and must be treated by extensive operative debridement, often in conjunction with either autogenous in situ reconstruction or extra-anatomic bypass grafts to avoid CLI. Skin erosion or expanding rupture into adjacent soft tissue obviously is an unstable situation for which urgent surgical repair is necessary irrespective of the etiology of the femoral artery aneurysm or pseudoaneurysm.

C. CATHETER-RELATED FEMORAL ARTERY PSEUDOANEURYSMS.

RECOMMENDATIONS

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)***

Although a pulsatile mass is an obvious indication that a pseudoaneurysm may be present, a diagnostic duplex scan should be obtained whenever the diagnosis is even suspected. In the absence of antithrombotic therapy, several studies have indicated that catheter-related pseudoaneurysms that are less than 2.0 cm in diameter tend to heal spontaneously and usually require no treatment. Figure 14 illustrates the spontaneous closure rate of selected pseudoaneurysms that were not immediately repaired, 90% of which resolved within 2 months. Accordingly, small asymptomatic pseudoaneurysms probably can be managed conservatively unless they are still present on a follow-up duplex scan 2 months later.

At the opposite extreme, large pseudoaneurysms can rupture into the retroperitoneal space or the upper thigh or cause

Table 35. Ultrasound-Guided Compression of Femoral Pseudoaneurysms

First Author	Reference	Patients (n)	Closure (n)	Surgery (n)	Comments
Chatterjee	(449)	38	37	1	FemoStop used
Coughlan	(450)	10	9	1	
Cox	(451)	100	94	2	10 Recurrences, 1 to 35 days
Dean	(452)	77	56	14	Size less than 4 cm; twice as successful at closure
Feld	(453)	15	10	2	
Fellmeth	(454)	29	27	—	
Hajarizadeh	(455)	57	54	2	2 Recurrences, 2 to 10 days
Hertz	(456)	41	36	3	Large catheter sheath size problematic
Kazmers	(457)	33	25	3	2 Pseudoaneurysm ruptures
Kumins	(458)	60	52	—	7 Recurrences
Langella	(459)	36	27	—	3 Recurrences
Paulson	(460)	48	37	—	
Perkins	(461)	13	10	—	
Schaub	(462)	124	104	5	
Sorrell	(462)	11	10	1	
Steinkamp	(463)	98	96	2	
Weatherford	(464)	11	8	3	

Table 36. Thrombin-Injection Closure of Femoral Pseudoaneurysms

First Author	Reference	Patients	Thrombin Dose (U)	Closure (n)	Surgery (n)	Comments
Hughes	(468)	9	1000 to 2000	8	0	1 recurrence at 4 days
Kang	(469)	21	500 to 1000	20	1	
La Perna	(467)	70	1000	66	2	94% overall success rate Success maintained in patients with antithrombotic medications
Liau	(470)	5	1000	5	0	
Mohler	(471)	91	500 to 1000	87	0	98% overall success rate Second injection required for 3 patients
Reeder	(472)	26	50 to 450	25	0	1 recurrence at 4 days
Sacket	(473)	30	100 to 2000	27	3	
Taylor	(474)	29	600	27	1	

venous thrombosis or painful neuropathy by compressing the adjacent femoral vein or the femoral nerve. Urgent surgical repair clearly is necessary if any of these serious complications occur, and until recently, it was the mainstay of treatment for most catheter-related femoral artery injuries. Many reports now have demonstrated, however, that the majority of uncomplicated pseudoaneurysms can be managed nonoperatively with either ultrasound-guided compression therapy or the injection of miniscule amounts of thrombin directly into the pseudoaneurysm cavity. Problems with ultrasound-guided compression therapy include pain at the site of compression, long compression times, and incomplete closure, each of which is more problematic with large pseudoaneurysms. Table 35 contains information from 17 series of patients who underwent ultrasound-guided compression therapy with a primary success rate of 86% and surgical treatment in only 4.9%. Recurrences usually responded to further compression and most frequently were associated with pseudoaneurysms that exceeded 4.0 cm in size in patients who had required larger-diameter delivery sheaths or periprocedural anticoagulation.

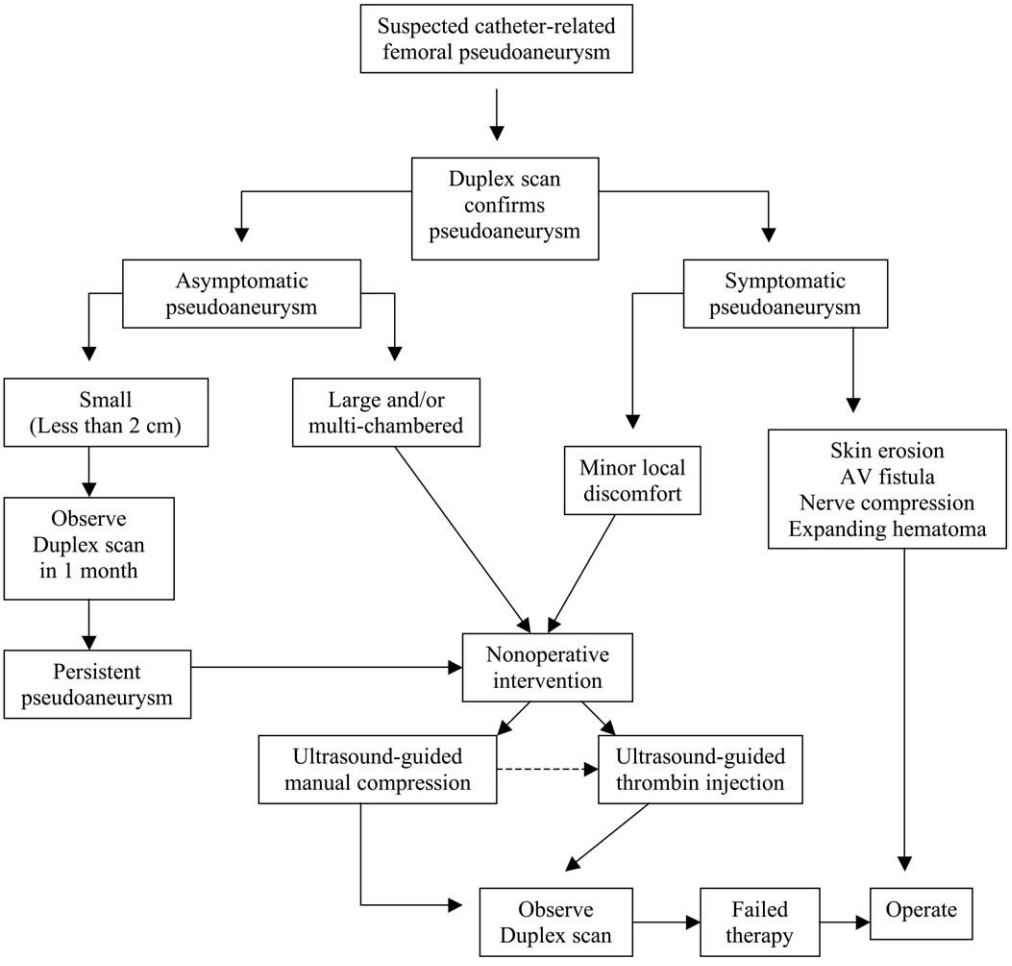

Figure 15. Diagnostic and treatment algorithm for femoral pseudoaneurysm. AV = arteriovenous; US = ultrasound.

Pseudoaneurysms ranging in size from 1.5 to more than 7.5 cm may be successfully obliterated by the injection of thrombin, 100 to 3000 international units, under ultrasound guidance. Table 36 contains data from seven institutional series in which thrombin injection was performed for catheter-related femoral pseudoaneurysms. In aggregate, the success rate was 93%, and only 4.1% of the patients needed operations. Thrombin injection can be complicated by distal arterial thromboembolism in less than 2% of cases and rarely by pulmonary embolism. The recurrence rate is approximately 5% after an initial injection, but recurrent pseudoaneurysms can be safely reinjected with a high rate of success (465–467).

The algorithm illustrated in Figure 15 presents an approach to the management of catheter-related femoral artery pseudoaneurysms that is consistent with the current literature on this topic (16).

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