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

The following is one of three extracted sections—lower extremity, renal/mesenteric, and abdominal aortic—of the ACC/AHA 2005 Guidelines for the Management of Patients With Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic): 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). We have separated and posted each section online to facilitate easy downloading by specialists interested in a specific portion of the guideline; however, it is important that when citing the guidelines, the full-text document of record be cited as Hirsch AT, Haskal ZJ, Hertzer NR, et al. Peripheral Arterial Disease: ACC/AHA 2005 Guidelines for the Management of Patients With Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic): 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). J Am Coll Cardiol 2006;47:1239-312. The full-text guidelines are available at http://www.acc.org/clinical/guidelines/pad/index.pdf, and an executive summary is available at http://www.acc.org/clinical/guidelines/pad/index.pdf. Please note that these sections were not written as stand-alone documents and therefore may reference tables and figures not appearing in this section. Readers should make a concerted effort to ensure that they have reviewed any pertinent information related to this subject that may be located in another section.

A classification of recommendation and a level of evidence have been assigned to each recommendation. Classifications of recommendations and levels of evidence are expressed in the ACC/AHA format as follows.

Classification of Recommendations

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

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

Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy.

Class IIb: Usefulness/efficacy is less well established by evidence/opinion.

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

Level of Evidence

- Level of Evidence A: Data derived from multiple randomized clinical trials or meta-analyses.
- Level of Evidence B: Data derived from a single randomized trial or nonrandomized studies.
- Level of Evidence C: Only consensus opinion of experts, case studies, or standard-of-care.

The ACC/AHA Task Force on Practice Guidelines makes every effort to avoid any actual, potential, or perceived conflicts of interest that might arise as a result of an outside relationship or personal interest of a member of the writing panel. Specifically, all members of the 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 each meeting, and updated and reviewed by the writing committee yearly and as changes occur. Appendixes 1 and 2 contain information on author relationships with industry for authors and peer reviewers, respectively, and are attached to this extracted section for the convenience of the reader. The complete reference list of the full-text guidelines is also included in this document.

The Committee to Develop Guidelines for Peripheral Arterial Disease conducted comprehensive searching of the scientific and medical literature relevant to peripheral arterial disease (PAD). Please see the Preamble to the full-text guidelines for information on the ACC/AHA methodology specific to this guideline.

These guidelines were approved for publication by the governing bodies of the American College of Cardiology (ACC) and the American Heart Association (AHA) and have been officially endorsed by the following collaborating organizations: Society for Cardiovascular Angiography and Interventions; Society for Vascular Medicine and Biology; Society for Vascular Surgery; and Society of Interventional Radiology; as well as 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.

TABLE OF CONTENTS

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

Although their causes may be diverse, 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. For all of these reasons, current guidelines for the diagnosis and management of arterial aneurysms may be useful to clinicians irrespective of their primary care or specialty training.

5.1. Definition

According to some sources, the diagnosis of AAA should be determined by formulas that adjust for age or body surface area or by calculating the ratio between normal and dilated aortic segments (859-863). Generally, however, an AAA is considered to be present when the minimum anteroposterior diameter of the aorta reaches 3.0 cm. The size of the aorta can be measured in any plane that is perpendicular to the vessel axis, but in practice, the anteroposterior diameter is measured most easily and reproducibly. Accordingly, most screening studies define AAA in this manner (859).

There is abundant information concerning normal diameters of the abdominal aorta and its branches in healthy adults, which indicates enlargement with age and body size and larger diameters in men than in women (Table 43) (864-866). A diameter of 2.7 cm represents the 95th percentile for the nonaneurysmal infrarenal aorta in men 65 to 83 years of age (867), and 2.9 cm exceeds the upper limit of normal irrespective of age, gender, or body surface area (868). Women have slightly smaller normal aortic diameters than men (862), and although this difference in baseline aortic diameter between women and men is not great enough to influence the minimum size of 3.0 cm that customarily is used to

Table 43. Dimensions of Normal Arteries

First Author and Procedure	Females		Males		Assessment Method
	Mean Diameter, cm, Range	Standard Deviation, cm, Range	Mean Diameter, cm, Range	Standard Deviation, cm, Range	
Abdominal aorta, supraceliac	2.10 to 2.31	0.27	2.50 to 2.72	0.24 to 0.35	Computed tomography
Abdominal aorta, suprarenal	1.86 to 1.88	0.09 to 0.21	1.98 to 2.27	0.19 to 0.23	Computed tomography
Abdominal aorta, infrarenal	1.66 to 2.16	0.22 to 0.32	1.99 to 2.39	0.30 to 0.39	Computed tomography, IV arteriography
Abdominal aorta, infrarenal	1.19 to 1.87	0.09 to 0.34	1.41 to 2.05	0.04 to 0.37	B-mode ultrasound, computed tomography, IV 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 to 1.02	0.15 to 0.19	1.17 to 1.23	0.20	Computed tomography
Internal iliac	0.54	0.15	0.54	0.15	Arteriography
Common femoral	0.78 to 0.85	0.07 to 0.11	0.78 to 1.12	0.09 to 0.30	Computed tomography, 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

IV indicates intravenous; NA, not available.

Adapted from J Vasc Surg, 13, Johnston KW, Rutherford RB, Tilson MD, et al. 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, 452-58, Copyright © 1991, with permission from Elsevier (863).

define a small AAA, it may influence recommendations for the size at which larger aneurysms should be repaired.

5.2. Abdominal Aortic and Iliac Aneurysms

5.2.1. Prevalence

The prevalence of AAA varies with a number of demographic factors (Table 44), including advancing age, family history, male gender, and tobacco use. A necropsy study in Malmo, Sweden, where autopsies are performed after nearly all hospital deaths, revealed that the incidence of AAAs larger than 3.0 cm in diameter increased at ages over 50 years, reaching a maximum prevalence of 5.9% in men 80 to 85 years of age and 4.5% for women over 90 years of age (868). Most population-based ultrasound screening surveys have been performed among white men and women, particularly those of Northern European and Scandinavian ancestry. A variety of threshold diameters have been used in these investigations, which makes it difficult to establish consistent estimates of prevalence. 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 to up to 12.5% for men 75 to 84 years of age. Comparable prevalence figures for women are 0% and 5.2%, respectively.

Race also appears to influence the prevalence of AAAs and iliac aneurysms. These aneurysms are rarely encountered in population-based screening studies in Japan, where the prevalence of traditional risk factors for atherosclerosis is lower than in white populations (876,877). In a United Kingdom community in which 14% of the population was of Asian descent, a review of medical records identified 233 cases of AAA, none of which occurred in the Asian population (878).

5.2.1.1. Generalized Arteriomegaly

Generalized arteriomegaly reflects a systemic alteration of the elastic component of the arterial wall, which results in dilation and elongation of many arteries. Patients with localized AAA are relatively unlikely to have generalized arteriomegaly (879), 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 AAA, and in 36% (5/14) of patients with generalized arteriomegaly (880).

5.2.2. Etiology

Most aortic and peripheral aneurysms represent a manifestation of aortic medial degeneration, which has complex bio-

Table 44. Prevalence of Abdominal Aortic Aneurysms in Population-Based Screening Studies

Country/Study	First Author	Reference	Number Screened	Age, y	Criteria	% Prevalence/ Gender	Relative Risk
Western Australia	Jamrozik	(869)	12 203	65 to 69	Larger than 3.0 cm	4.8/Male	Higher risk: Current or ex-smokers
				80 to 83	Larger than 3.0 cm	10.8/Male	Established PAD, CAD
				65 to 83	Larger than 5.0 cm	0.69/Male	Waist-hip ratio larger than 0.9 Lower risk: Mediterranean born versus Australian born (OR 0.6) Regular vigorous exercise
Veterans Affairs Cooperative Study	Lederle	(870)	126 196*	50 to 79	Larger than 4.0 cm	1.3/Male and female	Higher risk: Increased age per 7 years (OR 1.7) Smoking history (OR 5.17)
				50 to 79	Larger than 4.9 cm	0.45/Male and female	Family history (OR 1.9) Established atherosclerosis (OR 1.6)
				50 to 79	Larger than 5.4 cm	0.27/Male and female	Lower risk: Female (OR 0.18; 2.7% of total) Black race (OR 0.59) Diabetes mellitus (OR 0.50)
Norway	Singh	(871)	6386	25 to 84	Larger than 2.9 cm	8.9/Male; 2.2/female	Higher risk: Increased age
				45 to 54	Larger than 2.9 cm	1.9/Male; 0/female	Smoker older than 40 y vs. never-smoker (OR 8.0)
				55 to 64		6.0/Male; 1.1/female	
				65 to 74		12.8/Male; 2.8 female	
				75 to 84		18.5/Male; 4.8/female	
				55 to 64	Larger than 3.9 cm	1.1/Male; 0.1/female	
				65 to 74		4.1/Male; 0.7/female	
				75 to 84		8.6/Male; 1.0/female	
The Netherlands	Pleumeekers	(872)	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
				Older than 54	Larger than 4.0 cm	1.6/Male; 0.3/female	Established cardiovascular disease
Belgium	Vazquez	(873)	716‡	65 and 75	Larger than 3 cm Larger than 4 cm	3.8/Male 0.3/Male	Higher risk: Arterial hypertension (p less than 0.05) Prior CABG (p less than 0.01) Smoker (p less than 0.06)

Continued on Next Page

Table 44. *Continued*

Country/Study	First Author	Reference	Number Screened	Age, y	Criteria	% Prevalence/ Gender	Relative Risk
The Netherlands	Boll	(874)	2419§	60 to 80	Larger than 2.9 cm	8.1/Male	
					Larger than 4.9 cm	1.7/Male	
United Kingdom	Wilmink¶	(875)					
Oxford			426	65 to 74	Larger than 4.0 cm or 5 mm larger than SRA	5.4/Male	
				65 to 74	Larger than 4.0 cm	2.3/Male	
Liverpool				Older than 55	Larger than 3.0 cm	2.9/Male	
Gloucestershire			4232	65	Larger than 2.5 cm	8.4/Male	
				65	Larger than 4.0 cm	1.3/Male	
Birmingham			2669	65 to 75	Larger than 2.9 cm	8.4/Male	
				65 to 75	Larger than 4.0 cm	3.0/Male	
Chichester			5394	65 to 80	Larger than 2.9 cm	7.6/Male	
				65 to 80		1.3/Female	
Northumberland			628	65 to 79	Larger than 2.9 cm	6.7/Male	
Huntingdon			7493	Older than 49	Larger than 2.9 cm	5.2/Male	
Japan	Takei	(876)	348	60 to 79	—	0	
Japan	Adachi	(877)	1591	—	—	0.3/Male	

*52 745 plus prior report of 73 451.
†Of 10 215 eligible.
‡Of 1764 eligible.
§Of 2914 eligible.
¶This portion of table adapted from Wilmink and Quick (875).
CABG indicates coronary artery bypass grafting; CAD, coronary artery disease; OR, odds ratio; SRA, suprarenal aneurysm.

logical mechanisms. Traditional views held that most aneurysms were caused by degenerative atherosclerotic disease, but other data (see Section 5.2.2.3) suggest that many aneurysms form in response to altered tissue metalloproteinases that diminish the integrity of the arterial wall.

5.2.2.1. Hereditary Risk Factors

A genetic predisposition to AAA formation has been suggested by studies of familial incidence, and an analysis of 313 pedigrees confirms the importance of familial factors (881). In a series of 542 patients undergoing AAA repair during a 9-year period, 15% had first-degree relatives with aneurysms compared with 2% of a control group of similar age and gender (p less than 0.001) (882). Other series have found first-degree relatives similarly affected in up to 28% of cases (883). 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 (881), which suggests a single dominant gene effect (Table 45). Among the offspring of patients with ruptured AAA, 21% of sons older than 45 years and 4% of daughters older than 42 years had aortic enlargement to a diameter of at least 3.0 cm (884). 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. One study found that patients with familial aneurysms were more often female than those without (35% vs. 14%) (885). Familial aneurysms do not expand more rapidly than nonfamilial AAA, nor are they differently located, but they may develop at an earlier age (see Section 5.2.4.6) (886).

Polycystic kidney disease, an autosomal dominant disease that affects 0.5 million people, and 8% to 10% of long-term hemodialysis cases in the United States have been associated with abdominal aneurysms (891,892). The association of cardiovascular lesions with polycystic kidney disease suggests involvement of the extracellular matrix in this disorder, but the main cause of aortic aneurysms is degenerative. Patients with renal disease may be prone to aortic aneurysm because of hypertension and connective tissue disorders, and yet an independent association between AAA and autosomal-dominant polycystic kidney disease is unproven.

5.2.2.2. 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)***

It is widely recognized that patients with AAAs have a significantly higher prevalence of smoking, hypertension, MI, heart failure, and carotid artery and/or lower extremity PAD than do age- and gender-matched controls. The lipoprotein(a) serum level, an indicator of atherosclerosis, is elevated in patients with AAA independent of cardiovascular risk factors and the extent of atherosclerosis, whereas patients with dissecting thoracic aortic aneurysms have levels comparable to those of healthy individuals (893).

Thoracic aortic atheromata detected by transesophageal echocardiography may independently predict AAA (894). In a study of 364 patients, 14% of those with thoracic atheromata had AAAs compared with only 1.4% of those without (OR 11.4, p less than 0.0001). Another indicator of generalized atherosclerosis, common carotid arterial intima-media thickness, was 0.98 plus or minus 0.34 mm in patients with occlusive arterial disease compared with 0.91 plus or minus 0.20 mm in patients with AAAs (an age- and gender-adjusted mean difference of 0.18 mm; 95% CI 0.08 to 0.28 mm) (895). The difference remained 0.11 mm (95% CI 0.01 to 0.21 mm) after adjustments for other cardiovascular risk factors. The smaller common carotid intimal-medial thickness in patients with AAAs than in patients with occlusive disease is independent of other determinants of intimal-medial thickness and probably reflects other pathophysiological mechanisms, such as hypertension.

5.2.2.3. Collagenase, Elastase, 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 (896). 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 (897). Abnormal biochemical elastolytic and active proteolytic activity has also been identified in aneurysmal aortas (898). An abnormal accumulation of macrophages (899) and elevated levels of cytokines (900) indicate that an inflammatory process may contribute to their pathogenesis. Cultured smooth muscle cells from aneurysmal aortas produce elevated levels of the plasminogen activators urokinase plasminogen activator and tissue plasminogen activator (901), which could increase proteolysis. In aggregate, the data suggest a major role for matrix metalloproteinases and their inhibitors in the loss of aortic wall structural integrity that leads to AAA formation and expansion.

Chronic obstructive pulmonary disease (COPD) and AAA share several risk factors. In 240 patients with thoracic aneurysms or AAAs, forced expiratory volume/forced vital capacity and carbon monoxide diffusing capacity were lower than in a control group (p less than 0.01) (902). The proportion with airway obstruction (forced expiratory volume in 1 second less than 70% of normal) was higher in the AAA group (100 of 240, or 42%) than in those without overt car-

Table 45. Prevalence in Families of Patients With Abdominal Aortic Aneurysms (AAAs)

Country	First Author	Reference	Study Group	Screened With Ultrasound	Age, y (Gender)	Criteria	Incidence/ Risk Factor
United Kingdom	Adams	(887)	Relatives of 100 patients with known AAA	76 of 110 eligible	Older than 50	Larger than 4.0 cm	0
					Older than 50	2.5 to 3.9 cm	21% of male first-degree relatives; 27% of sons; 17% of brothers; 4% of sisters; 0% of daughters
Sweden	Bengtsson	(884)	Offspring of patients who died of ruptured AAA	62 of 90 eligible	45 to 75 (males)	Larger than 2.9 cm	21% of sons
					45 to 80 (female)	Larger than 2.9 cm	4% of daughters
					45 to 80 (female)	Larger than 5.0 cm	3% (1 male aged 53 y)
Ireland	Fitzgerald	(888)	Siblings of patients with known AAA	125 of 234 eligible	Older than 80	3.1 to 6.8 cm	22% of brothers; 3% of sisters
Netherlands	Van Der Graf	(889)	Brothers of patients having elective surgery for AAA	210 of 571 eligible	Older than 50	New AAA	12.30%
					Older than 50	Larger than 4.9 cm	3.80%
Finland	Jaakkola	(890)	Families of patients with surgery for AAA	123 of 172 eligible	41 to 82	Larger than 2.9 cm or history of repair or rupture	10% of brothers; 3% of sisters
United States	Webster	(883)	First-degree relatives of patients with surgery for AAA	103 of 202 eligible	Older than 55	Larger than 3.0 cm or I/S diameter ratio greater than 1.5	16% of first-degree relatives; 25% of men; 6.9% of women

diovascular disease (51 of 223, or 23%) or in patients with coronary artery disease matched for age, gender, smoking, and other atherosclerotic risk factors (43 of 238, or 18%). By multiple logistic regression analysis, the presence of AAA (OR 2.928, 95% CI 1.722 to 4.979) and male gender (OR 1.622, 95% CI 1.055 to 2.493) were most strongly associated with COPD.

The association between AAA and COPD 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 COPD had aortic aneurysms (903). The overall mean annual expansion rate was 2.7 mm per year irrespective of COPD, but it 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). There was a negative correlation between the forced expiratory volume in 1 second and concentrations of serum elastin peptide and plasma elastase–alpha 1–antitrypsin complexes in patients with COPD, and the concentration of serum elastin peptide, therapy with beta-agonist bronchodilator medication, and forced expiratory volume in 1 second correlated with the degree of expansion. The high prevalence of AAA among patients with COPD might therefore be related more to medication use and coexisting diseases than to a common pathogenic mechanism.

Upregulation of genes involved in oxidative stress (e.g., heme oxygenase, inducible nitric oxide synthase, 12-lipoxygenase, and heart cytochrome c oxydase subunit VIa) and the downregulation of antioxidant genes (e.g., superoxide dismutase, reduced nicotinamide adenine dinucleotide-cytochrome b-5 reductase, and glutathione S-transferase) may play a role in the progression of AAAs (904). In patients with small, asymptomatic AAAs, prolonged administration of doxycycline was associated with reduced plasma matrix metalloproteinase (MMP-9) levels (905), but further studies are needed to evaluate the long-term effects of doxycycline on the rate and extent of aneurysm growth and the potential use of plasma MMP-9 levels as a biomarker of aneurysm disease progression.

The HMG coenzyme-A reductase inhibitors (statins) reduce the expression of matrix metalloproteinases independently of their cholesterol-lowering effect. One such agent (cerivastatin, 0.001 to 0.1 micromoles per liter) significantly reduced tissue levels of both total and active MMP-9 (p less than 0.001) (906). Cerivastatin suppressed MMP-9 production by inhibiting the activation of neutrophils and macrophages. It remains to be determined whether statin therapy could be useful for prevention or treatment of AAA.

5.2.2.4. Congenital Aneurysms

Over the course of normal aging, degenerative changes occur throughout most of the length of the aorta, which leads to a mild form of cystic medial necrosis. Although physiological, this process develops more rapidly in patients with bicuspid aortic valves and during pregnancy, and very markedly in the Marfan syndrome, in which more than 11% of patients sustain dissections of the aorta. The mechanisms by which the medial layer of the aorta is subject to accelerated degeneration are a topic of molecular genetic investigation. Gsell (in 1928) and Erdheim (in 1929) first described cystic medial necrosis, which is associated with histological evidence of severe elastic fiber degeneration, necrosis of muscle cells, and cystic spaces filled with mucoid material (907, 908). This is most often encountered in the ascending aorta between the aortic valve and the innominate artery, although similar changes can also occur in the remainder of the aorta. The Marfan syndrome, an inherited disorder characterized by dolichostenomelia, ligamentous redundancy, ectopia lentis, ascending aortic dilatation, and incompetency of the aortic and/or mitral valve (909), is frequently associated with cystic medial necrosis of the aorta. The syndrome is linked to an autosomal dominant anomaly in fibrillin type 1 (910), a structural protein that directs and orients elastin in the developing aorta (911-917). The Marfanoid aorta has markedly abnormal elastic properties and increased pulse wave velocities, with progressive stiffening and dilatation (918). Single-gene mutations have been identified that cause aneurysm formation in the Marfan syndrome and in Ehlers-Danlos syndrome type IV (919), but polygenic factors are probably involved in many cases.

Abnormalities associated with the Marfan syndrome typically affect the entire length of the aorta, although dissection most often involves the thoracic portion (920). Histologically, 10% to 21% of aortic dissections and 43% of all dissections in patients with Marfan syndrome have severe degeneration of the medial layer; more than 50% of the wall area shows features of cystic necrosis. Although most often encountered in the ascending aorta, cystic medial necrosis may occur in the abdominal aorta as well. Cystic medial degeneration may also be associated with other connective tissue disorders, such as the Ehlers-Danlos syndrome.

5.2.2.5. 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. This entity was first described in 1972 by Walker et al. and has since been described by Rasmussen and Hallett as an extreme manifestation of inflammation present in all aortic aneurysms (921). Abnormal accumulation of macrophages and cytokines in aneurysmal aortic tissue supports an association with inflammation (899,900). 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 (922). In another series of 355 patients undergoing surgical repair of

AAA, 5.6% had inflammatory clinical features and 11% had histological evidence of inflammation (923), but the early and late results of surgery were no different between the 2 groups.

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 (924). 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. Additional aneurysms were discovered in half of these patients, including iliac aneurysms in 55, thoracic or thoracoabdominal aneurysms in 17, femoral aneurysms in 16, and popliteal aneurysms in 10. Excretory urographic findings of medial ureteral displacement or obstruction suggested the diagnosis of inflammatory AAA in 31% of the cases. 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).

5.2.2.6. Infectious Aneurysms

Primary infection of the aortic wall is a rare cause of aneurysms, which are more often saccular than fusiform. Infectious, or "mycotic," aneurysms may arise secondarily from infection of pre-existent aneurysm (925). Staphylococcus and Salmonella are the most frequent pathogens that cause primary aortic infections (926), and tuberculosis has been described in association with aortic pseudoaneurysms (927).

An infectious etiology also has been postulated for conventional atherosclerotic aneurysms. Antibodies against Chlamydia pneumoniae have been detected by polymerase chain reactions in conjunction with atherosclerosis and expanding AAA (928), but it has not been possible to document that C pneumoniae antigens react with anti–C pneumoniae membrane proteins. Sixty-six percent of specimens from atherosclerotic arteries collected during various peripheral arterial operations (including AAA repair in 28 patients) revealed severe atherosclerosis and positive immunohistochemical staining for specific antibodies against C pneumoniae (929). Because there were no differences in cardiovascular risk factors, the prevalence of coronary heart disease or previous vascular surgery, or inflammatory serum markers between patients with and without C pneumoniae antibodies, this organism has been considered a concomitant phenomenon rather than a causative factor for atherosclerosis.

Although secondary prevention benefits of antibiotic therapy have been demonstrated in some studies, negative studies have also emerged. In a randomized study, 92 subjects with small AAAs received the macrolide antibiotic roxithromycin (300 mg orally daily for 28 days) or a matching placebo. The mean expansion rate of the AAA during the first year of observation in the intervention group (1.6 mm) was reduced by 44% compared with the placebo group (2.8 mm, p equals 0.02). During the second year, however, the difference favoring roxithromycin was only 5% (930). When adjusted for smoking, diastolic blood pressure, and the immunoglobulin A level, roxithromycin treatment and the initial size of the aneurysm were related to AAA expansion. Logistic regression analysis confirmed a significant difference in expansion rates exceeding 2 mm annually between the intervention and placebo groups (OR 0.09, 95% CI 0.01 to 0.83). The results of larger prospective, human antibiotic intervention trials may help to establish whether or not there is a causal link between C pneumoniae infection and atherosclerotic aortic aneurysms.

5.2.3. 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.

5.2.3.1. 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)***
2. **Patients with infrarenal or juxtarenal AAAs measuring 4.0 to 5.4 cm in diameter should be monitored by ultrasound or computed tomographic scans every 6 to 12 months to detect expansion. *(Level of Evidence: A)***

Class IIa

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

Class III

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)***

Rupture is the most widely recognized complication of arterial aneurysms and primarily is associated with those involving the abdominal aorta, the common iliac arteries, and the visceral arteries. Before the introduction of B-mode ultrasonography in the 1970s and computed tomographic scanning in the 1980s, the expansion rate of aortic, iliac, and visceral aneurysms could only be determined by standard plain-film roentgenograms in the presence of mural calcification. Modern imaging techniques, which now have been further supplemented by magnetic resonance imaging/MRA, currently permit more accurate estimates of expansion rates that can be used to monitor the growth of aneurysms and to select patients for preemptive intervention before rupture occurs. Growth rates have been most widely documented for aortic aneurysms, several examples of which are presented in Table 46. These data confirm similar observations (931,932) that large aneurysms tend to expand more rapidly than small aneurysms and thus require closer surveillance. According to the available information, average annual expansion rates are approximately 1 to 4 mm for aortic aneurysms measuring less than 4.0 cm in diameter at the time of their discovery, 4 to 5 mm for those measuring 4.0 to 6.0 cm in diameter, and as much as 7 to 8 mm for larger aneurysms (933,934). An observed rate of expansion that exceeds these figures usually is considered to represent a "growth spurt" that may justify early elective aneurysm repair.

High operative mortality rates alone do not fully reflect the catastrophic nature of ruptured aortic aneurysms. Given the number of patients who do not survive even to reach the operating room, the overall mortality rate for this complication may be as high as 90% (942-944). In a classic report, Szilagyi et al. (945) were among the first to recognize that the risk for spontaneous rupture was a direct function of aneurysm size. Others have since discovered that additional factors also may influence the rupture rate, such as hypertension (946,947), COPD and/or tobacco abuse (946-949), female gender (882,947), and a family history of aortic aneurysms, particularly when a woman with an aortic aneurysm is present in the proband (882). Nevertheless, aneurysm size remains the single most important predictor not only for aneurysm rupture, but also for unrelated death from other cardiopulmonary events (932,950).

Table 47 contains representative data regarding aneurysm rupture rates and long-term patient survival rates according to the baseline diameter of AAAs at the time of their discovery. These data suggest that the eventual risk for rupture is approximately 20% for aneurysms that measure larger than 5.0 cm in diameter, 40% for those measuring at least 6.0 cm in diameter, and higher than 50% for aneurysms that exceed 7.0 cm in diameter. Taylor and Porter interpreted earlier data to indicate that the annual rupture rates for aneurysms of these sizes were in the range of 4%, 7%, and 20%, respectively (938). 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. Watson et al. found that more patients with small aneurysms died of other causes than ever required surgical treatment for enlarging aneurysms (951). Bengtsson et al. have recommended only 1 annual follow-up scan for aneurysms less than 3.5 cm in diameter because the unrelat-

Table 46. Annual Rates of Expansion for Abdominal Aortic Aneurysms

First Author	Reference	Year	No. of Patients	Initial Aneurysm Diameter	Mean Annual Expansion, mm
Case series					
Nevitt	(935)	1989	103	3.5 to 5 cm	2.1
Cronenwett	(936)	1990	73	Smaller than 6 cm	4 to 5
Bengtsson	(937)	1993	155	Smaller than 4 cm Larger than or equal to 4 cm	0.8 5.3
Collective reviews					
Taylor	(938)	1986	—	Larger than or equal to 5 cm	5
Hollier	(939)	1992	—	3 to 3.9 cm 4 to 5.9 cm Larger than 6 cm	2.7 4.3 7.5
Hallin	(940)	2001	—	Smaller than 4 cm 4 to 5 cm Larger than 5 cm	2 to 4 3 to 5 3 to 7
Randomized trials					
Veterans Affairs Small Aneurysm Trial (nonoperated cohort)	(941)	2002	—	4 to 5.5 cm	3.2

Table 47. Rupture and Survival Rates for Patients With Abdominal Aortic Aneurysms

First Author	Reference	Year	No. of Patients	Baseline Aneurysm Diameter	Follow-Up Interval	Aneurysm Rupture Rate (%)	Survival Rate (%)
Case series							
Szilagyi	(945)	1966	82	Less than or equal to 6 cm	Mean 34 mo	19	45
			141	Larger than 6 cm	Mean 17 mo	43	10
Hertzer	(955)	1987	24	Smaller than 6 cm	5 y	20	38 Overall
			18	At least 6 cm	5 y	69	
Nevitt	(935)	1989	130	Smaller than 5 cm	5 y	0	NA
			46	At least 5 cm	5 y	25	NA
Bengtsson	(937)	1993	155	Median 4 cm	Median 3.4 y	14	30
Perko	(956)	1993	63	Smaller than 6 cm		less than 5	NA
				At least 6 cm		10 to 15	NA
Galland	(957)	1998	267	Smaller than 4 cm	5 y	4	NA
				4 to 5.5 cm	5 y	21	NA
Jones	(958)	1998	25	5 to 5.9 cm	3 y	28	NA
			32	At least 6 cm	3 y	41	NA
Scott	(953)	1998	218	3 to 4.4 cm	7 y	2.1 per year and/or operation	NA
				4.5 to 5.9 cm	7 y	10 per year and/or operation	NA
Conway	(950)	2001	23	5.5 to 5.9 cm	10 y	22	39
			62	6 to 7 cm	10 y	34	32
			21	Larger than 7 cm	10 y	52	5
Biancari	(959)	2002	41	2.5 to 4 cm	Median 7.3 y	7.3	59
Collective reviews							
Taylor	(938)	1986		5 cm	NA	4.1 per year	NA
				5.7 cm	NA	6.6 per year	NA
				7 cm	NA	19 per year	NA
Hollier	(939)	1992	349	Smaller than 5 cm	5 y	4.6	NA
			90	Larger than 5 cm	5 y	30	NA
Hallin	(940)	2001	54 048	Smaller than 4 cm	4 y	2	NA
				4 to 5 cm	4 y	10	NA
				Larger than 5 cm	4 y	22	NA
Randomized trials							
UK Small Aneurysm	(960)	1998	213	4 to 4.4 cm	Mean 4.6 y		
Trial (nonoperated			169	4.5 to 4.8 cm	Mean 4.6 y	NA	75%
cohort)			145	4.9 to 5.5 cm	Mean 4.6 y	NA	72%
						NA	64%
UK Small Aneurysm	(961)	1999	NA	3 to 3.9 cm	7 y	2.1	NA
Trial (nonoperated			NA	4 to 5.5 cm	7 y	4.6	NA
cohort)			NA	At least 5.6 cm	7 y	20	NA

NA indicates not available; UK, United Kingdom.

ed mortality rate in such patients is so high that relatively few live long enough to incur sufficient aneurysm growth to warrant elective surgical treatment (931). 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 (952,953). Katz et al. concluded from a Markov predictive model that early intervention to repair aneurysms that measure 4.0 cm in diameter could be justified if operative mortality rates were 4.6% or lower, but their estimates were confounded by the low reported rupture rate for untreated aneurysms of this size (954).

5.2.3.1.1. Randomized Trials. Prospective randomized trials comparing early intervention versus expectant observation for infrarenal AAAs 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 (947,961-963). 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 in size on serial imaging studies. Selected data from both investigations are summarized in Table 48, with updated information from the UK trial at a mean follow-up interval of 8 years (963) 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 composed 17% of 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 (see Table 49). 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 had 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 (960). 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 (963). 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 (OR 4.0; 95% 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.

Collectively, these 2 randomized trials provide a wealth of information that otherwise has not been available. For instance, the finding that rupture has been significantly more likely to occur among women in the nonoperative cohort of the UK trial adds further perspective to the lingering controversy concerning whether the indications for elective aneurysm repair should be slightly more liberal in women than in men because of the smaller size of the normal aorta in women. On the basis of the data regarding gender differences in the UK trial, a guidelines subcommittee of the American Association for Vascular Surgery and the Society for Vascular Surgery now has recommended that a diameter of 4.5 to 5.0 cm is an appropriate threshold for elective repair of asymptomatic infrarenal aortic aneurysms in women (964).

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.

5.2.3.2. 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 (965,966). 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 (966). Therefore, isolated common iliac aneurysms that are smaller than 3.0 cm probably can be monitored safely with serial noninvasive imaging. Contrast-enhanced computed tomographic 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.

5.2.3.3. Local Compression or Erosion

Exceptionally large or inflammatory aortic aneurysms occasionally can be associated with early satiety or gastric outlet symptoms on the basis of duodenal compression. More catastrophically and just as infrequently, an aortic aneurysm may cause either sudden upper gastrointestinal bleeding on the basis of a primary aortoenteric fistula or acute congestive heart failure on the basis of an aortocaval fistula. Far more commonly, approximately 20% of patients who have large popliteal aneurysms also have signs of venous insufficiency

Table 48. Outcomes of Early Elective Repair Versus Nonoperative Surveillance of Asymptomatic Abdominal Aortic Aneurysms*

	UK Trial (2002)	VA Trial (2002)
Total patients, n	1090	1136
Early elective repair, n	563	569
Open	536	567
Endovascular	27	2
Nonoperative surveillance, n	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, y	Range 6 to 10; mean 8	Range 3.5 to 8.0; mean 4.9
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	OR 1.0 (reference set)	NA
Women	OR 4.0 95% CI 2.0 to 7.9 (*p* less than 0.001)	NA
Eventual aneurysm repair, n (%)		
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%

NA indicates not available.
*Results of 2 prospective randomized trials conducted in the United Kingdom (960, 963) and by the United States Department of Veterans Affairs (941).

Table 49. Operative Mortality Rates for Open Repair of Intact Abdominal Aortic Aneurysms

First Author	Reference	Year (Study Period)	No. of Patients	Mortality Rate (%)
Case series				
Crawford	(1061)	1981 (1955-1980)	Asymptomatic: 531	3.8
			Symptomatic intact: 329	6.4
			Total: 860	4.8
Hertzer	(955)	1987 (1978-1982)	246	4.4
Reigel	(1063)	1987	499	2.8
Golden	(1065)	1990	500	1.6
Sicard	(1071)	1995	145	1.4
Lloyd	(1079)	1996 (1980-1995)	1000	2.4
Starr	(1060)	1996 (1983-1989)	Men: 490	5.1
			Women: 92	4.3
			Total: 582	5.0
Aune	(1058)	2001 (1985-1999)	Age less than 66 y: 118	1.7
			Age 66 y and older: 333	6.0
			Total: 451	4.9
Hertzer	(1068)	2002 (1989-1998)	1135	1.2
Menard	(1080)	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)	(960)	1998	563	5.8
Lederle (U.S. Veterans Affairs Small Aneurysm Trial; surgical cohort)	(941)	2002	569	2.7
Collective reviews				
Ernst	(1081)	1993 (1981-1992)	6488	4.0
Zarins	(973)	1997 (1987-1992)	2162	2.1
Blankensteijn	(1074)	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 multicentered studies				
Johnston (Canadian Aneurysm Group)	(1082)	1988	Elective: 541	3.9
			Symptomatic intact: 125	7.2
			Total: 666	4.5
Richardson (Kentucky Medicare)	(1083)	1991	136	5.9
Hannan (New York statewide)	(1084)	1992 (1982-1987)	6042	7.6
Johnston (Canadian Aneurysm Group)	(1085)	1994	Men: 545	4.4
			Women: 134	5.2
			Total: 679	4.6
Katz (Michigan statewide)	(1086)	1994 (1980-1990)	8185	7.5
Kazmers (Veterans Affairs)	(1087)	1996 (1991-1993)	3419	4.9
Wen (Ontario Aneurysm Study)	(1088)	1996 (1988-1992)	5492	3.8
Kantonen (Finland Vascular Registry)	(1089)	1997	929	5.1
Koskas (French AURC)	(1057)	1997 (1989)	1107	4.8
Bradbury (Edinburgh Vascular Registry)	(1090)	1998 (1976-1996)	492	6.1
Manheim (California statewide)	(1091)	1998 (1982-1994)	35 130	7.6
Dardik (Maryland statewide)	(1092)	1999 (1990-1995)	2335	3.5
Pearce (Florida statewide)	(1093)	1999 (1992-1996)	13 415	5.7
Sollano (New York statewide)	(1094)	1999 (1990-1995)	9847	5.5
Kazmers (Veterans Affairs)	(1095)	2001 (1991-1995)	5833	4.5
Axelrod (Veterans Affairs)	(949)	2001 (1997-1998)	1001	3.7
U.S. hospital databases				
Lawrence (National Hospital Discharge Survey)	(1075)	1999 (1994)	32 387	8.4
Heller (National Hospital Discharge Survey)	(1076)	2000 (1979-1997)	358 521	5.6
Huber (Nationwide Inpatient Sample)	(1096)	2001 (1994-1996)	16 450	4.2
Dimick (Nationwide Inpatient Sample)	(1078)	2002 (1996-1997)	13 887	3.8

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

in the lower leg on the basis of compression of the adjacent popliteal veins (967,968).

5.2.4. Diagnosis

5.2.4.1. 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)***

Most AAAs are asymptomatic and are discovered incidentally on routine physical examination or on an abdominal roentgenogram (969) or an ultrasound scan that has been performed for other indications. Younger patients are more likely to be symptomatic at the time of diagnosis (970). 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. In contrast to musculoskeletal back pain, 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 (971). 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.

Hemorrhagic shock may ensue rapidly and is manifested by hypotension, vasoconstriction, mottled skin, diaphoresis, mental obtundation, and oliguria. and terminally, by arrhythmias and cardiac arrest. In a few patients who survive with contained ruptures, the retroperitoneal hematoma may be accompanied by ecchymosis in the flanks (Grey-Turner sign) and groin. Free rupture into the peritoneal cavity produces obvious abdominal distention and often is rapidly fatal, whereas rupture into the duodenum is manifested by massive gastrointestinal hemorrhage.

5.2.4.2. 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. A longitudinal cohort study involving 4734 men and women older than 65 years of age in 4 US communities correlated abdominal aortic diameter by ultrasonography with incidental cardiovascular disease, mortality, and repair or rupture during a mean follow-up period of 4.5 years (972). The prevalence of aneurysms was 8.8%, of which 88% were at least 3.5 cm in size. The rates of total mortality (65 vs. 33 per 1000 person-years), cardiovascular mortality (34 vs. 14 per 1000 person-years), and incidental cardiovascular disease (47 vs. 31 per 1000 person-years) were higher in participants who had aneurysms than in those who did not. After adjustment for age, risk factors, and the presence of other cardiovascular disease, the respective relative risks were 1.32, 1.36, and 1.57, respectively. In comparison, the rates of repair and rupture were low in this series.

Elective surgical repair improves the survival rate for patients with large aneurysms (945), and approximately 50 000 operations are performed annually for this condition in the United States, with operative mortality rates that are reported to be as low as 2% in some centers (973). Even before the results of randomized trials were available, however, it generally was accepted that watchful waiting with serial imaging was a better long-term treatment strategy than early surgical repair for aneurysms less than 5.0 cm in diameter (939). Up to 13% of patients with aortic aneurysms have multiple aneurysms elsewhere (974), and 25% to 28% of those with thoracic aortic aneurysms have concomitant AAAs (975,976). 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.

5.2.4.3. 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 (977). 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. The positive and negative likelihood ratios were 12.0 (95% CI 7.4 to 19.5) and 0.72 (95% CI 0.65 to 0.81), respectively, for AAAs that were 3.0 cm or larger and 15.6 (95% CI 8.6 to 28.5) and 0.51 (95% CI 0.38 to 0.67) for AAAs that were larger than 4.0 cm. The positive predictive value of palpation was 43% for AAAs that were documented to be at least 3.0 cm in diameter. Intuition and limited data suggest that abdominal obesity reduces the sensitivity of palpation. In summary, careful abdominal palpation is moderately sensitive for the detection of AAAs that are large enough to be referred for surgical intervention, but the physical examina-

tion alone may not be sufficiently reliable for the detection of smaller AAAs, especially if rupture already is suspected.

In a 3-year retrospective study of 198 patients with AAAs that was conducted by Alcorn et al. (860) in a general hospital setting, 48% of the aneurysms had been discovered clinically, 37% represented incidental findings during radiographic investigation of another condition, and 15% were encountered during unrelated abdominal operations. Of those that initially were detected by radiography, 38% were palpable on subsequent physical examination. The average size of the AAAs that were discovered clinically (6.5 plus or minus 1.3 cm) was larger than those that were found by radiography (5.47 plus or minus 1.4 cm, *p* less than 0.001) or at operation (5.4 plus or minus 1.5 cm, *p* equals 0.039). 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).

5.2.4.4. Incidental Radiological Findings

5.2.4.4.1. Plain Films. It is not the current standard of care to use plain radiographic studies for follow-up surveillance of AAAs, but 15% to as many as 85% of these aneurysms initially are discovered because of curvilinear aortic wall calcification that represents an incidental finding on a plain abdominal film that was obtained for other purposes. The plain film also may demonstrate a soft tissue mass with obliteration of the psoas margin and/or disruption of mural calcification with extension into a periaortic soft tissue mass, occasionally suggesting that the aneurysm has ruptured. In addition, smaller calcified rings sometimes suggest the presence of visceral artery aneurysms (978-981).

5.2.4.4.2. Ultrasound and Other Scans. Asymptomatic AAAs also may be discovered incidentally on ultrasound, computed tomography, and nuclear scans that have been performed for unrelated indications; conversely, computed tomography or ultrasound may demonstrate incidental nonvascular lesions during AAA evaluation, notably malignancy (982-991). The existence of incidental findings is not surprising given the advanced age of many patients undergoing imaging studies.

Phillips and King reported that 3.1% of male urologic patients (65 to 80 years of age) undergoing urinary tract ultrasonography were documented to have unsuspected aortic aneurysms; with deliberate augmentation of the scan to include the aorta (i.e., opportunistic screening), the incidence rose to 9.1%, a figure that appeared to exceed random discovery rates (985). Akkersdijk et al. found that incidental aneurysms with a diameter of at least 3.0 cm, or 1.5 times the diameter of the proximal aorta, were present in 4.9% of 1687 patients older than 50 years who underwent some form of abdominal ultrasonography, comprising 8.8% of men, 2.1% of women, and 11% of men over 60 years of age (988). Because the symptoms of expanding aneurysms can mimic urologic symptoms, additional scanning to include the aorta may be especially prudent in some specific clinical situations (991).

5.2.4.4.3. Opportunistic Screening. In the paradigm of "opportunistic" screening, abdominal ultrasound studies that primarily have been performed to obtain information regarding disease states other than aortic aneurysms (e.g., a urologic evaluation) are extended to include an examination of the nearby abdominal aorta (985,988,992-994). Studies in this area of interest have reported the prevalence of incidental aortic aneurysms to range from 6.5% to 12%, but these studies have not been rigorously controlled for age or other high-risk factors, such as tobacco use or a family history of aneurysms. Some believe that unlike a dedicated screening program, opportunistic screening can be done at little additional cost because most of the expense of the aortic imaging is borne by the baseline ultrasound scan. However, Wolf et al. noted that the addition of an aortic ultrasound scan to other unrelated studies in the vascular laboratory prolongs each examination by 5 minutes per patient and requires 83 minutes of scanning time for each aortic aneurysm that is detected (36 minutes per male smoker), at a cost of $240 to $553 per patient (994). In fact, this happens to be in the cost range of conventional population-based ultrasound screening (873). Furthermore, at least 1 investigation has indicated that opportunistic screening successfully demonstrates the aorta in only 89% of patients (less than the expected rate for most dedicated screening programs), perhaps because of inadequate patient preparation or operator skill (994). Therefore, because the ultrasound scan represents only a small fraction of the total expense that is associated with the detection and treatment of aortic aneurysms, the cost savings of opportunistic screening may be quite small in the general population in which the prevalence of such aneurysms is low.

There are multiple strategies for utilizing ultrasonography in a screening program for AAAs. Together with the data that already are available with respect to the prevalence rate of these aneurysms in various populations, the publication of 2 large randomized trials regarding aneurysm size and its influence on surgical indications may encourage computer modeling to determine the benefit, risks, and cost-effectiveness of ultrasound screening in targeted patient populations (947,961-963). This kind of information might also influence the decisions to be made by third-party payers.

5.2.4.4.4. Unrelated Arteriography. Catheter-based arteriography is not used as a primary diagnostic modality for aortic aneurysms, especially since mural thrombus makes it impossible to determine the true size of the aneurysm with the diameter of the contrast column. Arteriography instead is reserved to answer specific anatomic questions before endovascular management or, increasingly less frequently, before open AAA repair. However, several incidental findings during unrelated arteriographic studies may suggest the presence of an AAA, such as mural calcification, slow and/or turbulent flow, a widened interior lumen that is paradoxically smooth because of laminated thrombus and occlusions of

its branch vessels (e.g., the inferior mesenteric and lumbar arteries), "draping" of the superior mesenteric artery over the contour of the aneurysm, and a thickened aortic wall or soft tissue mass (995).

5.2.4.5. Diagnostic Imaging

5.2.4.5.1. ULTRASONOGRAPHY. B-mode or real-time ultrasound is excellent for imaging many aortic aneurysms because it has no risk to the patient and is less expensive than computed tomographic scanning (996-999). Its accuracy for measuring the aortic diameter below the level of the renal arteries approaches that of direct intraoperative measurements (997-999). In comparison, the accuracy of duplex ultrasound can be operator-dependent, and therefore, its results may vary between or even within centers, especially with small AAAs (1000,1001). This variability can be decreased with appropriate quality control and credentialing, but duplex scanning is more frequently used to evaluate the femoral or popliteal arteries to distinguish aneurysms from other vascular and nonvascular masses in these particular anatomic areas (1002-1008).

Infrarenal Aortic Aneurysms. Ultrasound scanning has been used in large screening and surveillance programs for both the initial assessment and subsequent follow-up of small aneurysms that are not repaired immediately. Multiple studies have suggested that ultrasound is an appropriate means to determine the presence or absence of an infrarenal aortic aneurysm in more than 95% of candidates (870,1009,1010). The maximum anteroposterior aortic diameter usually is determined after overnight fasting to aid visualization (859,1009). Ultrasonography should be performed in the plane perpendicular to the arterial axis, because oblique measurements tend to overestimate the true size of the aorta (863) and represent one source for potential variability.

Diagnostic specificity for the presence of an aneurysm is nearly 100% (859,873,1011), with sensitivity ranging from 92% to 99% (859,873,1011). The reproducibility and intraobserver variability of ultrasound measurements are quite satisfactory and are similar to those for computed tomographic scanning (961,1011,1012), although intraobserver correlation appears to be better near the aortic bifurcation than in the proximal infrarenal aorta (1011). Thus, ultrasonography is an excellent tool for screening and surveillance, both for individual patients and for screening programs. Modalities such as computed tomographic or MRA scanning usually are reserved for anatomic mapping before aneurysm repair because they are more expensive than ultrasound scanning and have some risk related to contrast and radiation.

Suprarenal Aortic and Iliac Aneurysms. Despite its utility in establishing the size of infrarenal aortic aneurysms, ultrasonography usually does not provide dependable imaging of aneurysms that extend close to the origins of the renal arteries or into the suprarenal segment of the abdominal aorta (969,996,998,1013-1015). In one prospective study, the upper and lower limits of AAAs were accurately demonstrated by ultrasound in only 47% and 41% of cases, respectively (1015). In another prospective study of 79 patients with AAAs, ultrasound reliably determined the length of the infrarenal aortic "neck" in only 20% of inflammatory aneurysms and 28% of noninflammatory aneurysms. Furthermore, standard B-mode ultrasound is suboptimal for imaging the common and internal iliac artery segments in the context of aneurysm disease, and duplex scanning is able to detect iliac artery involvement only about 50% of the time. A spiral computed tomographic scan of the abdomen and pelvis with 3D reconstruction in special instances is superior to ultrasonography for this purpose (1016).

5.2.4.5.2. CONTRAST-ENHANCED SPIRAL COMPUTED TOMOGRAPHIC SCANNING. For many years, transcatheter arteriography, including intra-arterial digital subtraction arteriography, was the "gold standard" for the preoperative assessment of AAAs. Early studies reported a high radiation dose and contrast load with computed tomography compared with digital subtraction arteriography (1017), but computed tomography provided additional information about adjacent veins and soft tissue and eventually supplanted digital subtraction arteriography as the preoperative study of choice. Because of improved techniques, their relatively noninvasive nature, and their cost advantage over transcatheter angiography, CTA and MRA have emerged as current "gold standards" in the preoperative and postoperative evaluation of AAAs (1018). In comparison, arteriography may be warranted to optimally define collateral or variant artery anatomy, such as the arterial supply to a horseshoe kidney, or the location and severity of occlusive disease or associated aneurysms in the visceral, renal, iliac, or peripheral arteries (997,1019). The decision to use either CTA or MRA is often locale-specific. Operator proficiency and the availability of suitable equipment and protocols may determine which modality is preferred.

Preoperative Aortic Aneurysm Assessment. The preoperative assessment of AAAs before open or endovascular repair includes defining the maximum transverse diameter and the relation of the aneurysm to the renal arteries. The length of normal-caliber aorta below the renal arteries before the aneurysm is commonly referred to as the infrarenal neck of the aneurysm. The length of this segment of normal caliber aorta as well as its diameter and angulation are particularly important when endovascular aneurysm repair is contemplated. In addition, preoperative imaging should demonstrate iliac or hypogastric aneurysms, serious occlusive disease in the iliac or renal arteries, the presence of vascular abnormalities (e.g., accessory renal arteries, duplicate vena cavae, or a retro-aortic left renal vein), or nonvascular soft tissue anomalies, such as horseshoe kidney (1020,1021). If endovascular AAA repair is under consideration, it is even more important to obtain precise measurements regarding the diameter and length of the proximal neck and the tortuosity of the aorta and the iliac arteries. Contrast-enhanced computed tomo-

graphic scanning provides baseline information in all of these areas. In select cases, contrast arteriography may be necessary in defining complicated arterial anatomy before endovascular aneurysm repair.

For accurate imaging of the length and diameter of the infrarenal AAA neck, narrow collimation (i.e., 3 mm or less) should be used (997,1021-1023). Because narrow collimation limits the aortic length that can be scanned and slows reconstruction time, typical computed tomography protocols call for narrow collimation around the renal arteries to define the superior extent of the aneurysm, combined with 10-mm collimation for the rest of the abdomen and pelvis (997). New multidetector computed tomography instrumentation promises to improve accuracy by being able to acquire more images in a faster time, with a single breath hold and less contrast medium (239). Recent helical computed tomographic techniques and protocols with 3D reconstruction displays should position computed tomography as a possible sole imaging modality for either open or endovascular AAA repair in the future (1024).

5.2.4.5.3. Magnetic Resonance Scanning. The presence of heavy mural calcification is sometimes important, because it may alter the planned repair. Computed tomography can accurately demonstrate vascular calcification, but it requires ionizing radiation and relatively large volumes of iodinated contrast. The presence of mural calcification can preclude successful computed tomographic evaluation of the peripheral arteries, so either adjunct arteriography or MRA may be needed. Magnetic resonance angiography presently has the disadvantage of being a slower scanning procedure than computed tomography and usually is not appropriate for use in patients who are claustrophobic or have metal implants. However, the coronal acquisition mode of current magnetic resonance techniques may expand its applications in the future.

Early MRA protocols depended on 3D time-of-flight imaging, which has a high signal-to-noise ratio but requires multiple slices and long imaging time because of in-plane flow saturation. Time-of-flight imaging is performed perpendicular to flow. The development of breath-held dynamic contrast-enhanced MRA has broadened the applicability of magnetic resonance by allowing rapid acquisition of images in any plane independent of flow (1025-1028). By imaging on the first pass during a breath hold, vascular signals can be obtained before leakage of contrast into the surrounding soft tissues, yielding an angiogram with high signal-to-noise ratio and enhanced detail. Images can be synchronized or subtracted for further enhancement (1028,1029). Similar protocols can be used to enhance contrast between the vessels and the background fatty tissue and have proven to be better than 3D time of flight for imaging the aortic branch vessels and the iliac arteries (1030).

In an early, blinded comparison of MRA versus conventional arteriography before elective aortic aneurysm repair, MRA was thought to be superior for defining the proximal extent of the AAA and for depicting venous anatomy, intraluminal thrombus, and coexistent iliac aneurysms (998). Subsequent improvement in magnetic resonance technique has yielded more accurate imaging of the renal arteries (209,981), a feature that eventually may make MRA as useful as spiral computed tomographic scanning for preoperative assessment before endovascular AAA repair (1012,1025,1031). In conclusion, the rapid development of both CTA and MRA makes their respective use for preoperative AAA assessment in large part dependent on local experience and the availability of the latest scanner. There presently is no consensus to indicate the superiority of either technique.

5.2.4.6. Screening High-Risk Populations

RECOMMENDATIONS

Class I

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

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 (1032,1033). Screening by this method has the potential to reduce the incidence of aortic rupture and has increasingly become the focus of population-based screening programs that have examined the efficacy of targeted AAA detection strategies. The effectiveness of ultrasound screening studies has been evaluated in several countries, with specific targeting of high-risk groups, such as those with hypertension, coronary disease, or tobacco use. A study of screening for AAAs in 3000 of 6058 males aged 64 to 81 years was underpowered to demonstrate a reduction in mortality through selective rescreening or surgical intervention for AAAs (1034). 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 larger 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 ORs 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 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. The risk factor associations for smaller AAAs (3.0 to 3.9 cm) were similar but less robust (870). According to one estimate, if the risk for AAA were based on age alone, it would be necessary to examine over half of the elderly male population to obtain 80% of the total

potential benefit among men. If age and smoking were included, the proportion needed to screen would fall to 35%. Even if other risk factors, such as coronary disease or hyperlipidemia, were included, it still would be necessary to screen 15% to 20% of the population, and the cost would be prohibitive (1035).

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 (1036). Men in whom aortic aneurysms at least 3.0 cm in diameter were detected underwent 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 1cm 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. The 30-day mortality rate was 6% (24 of 414) after elective aneurysm repair compared with 37% (30 of 81) after emergency operations. 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 US dollars). After an adjustment for censoring and a discount of 6%, the mean additional cost of screening was 63£ or $98 (95% CI 53.31£ to 73£ or $84 to $116) per patient. 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£ or $45 000 per life-year gained, a figure that is equivalent to approximately 36 000£ or $57 000 per quality-adjusted life-year. After 10 years, this figure was estimated to decline to approximately 8000£ or $12 500 per life-year gained (1037).

These values of cost-effectiveness for AAA screening are at the margin of acceptability according to most current health services thresholds. Over a longer period, however, cost-effectiveness is expected to improve substantially, decreasing to about one fourth of the 4-year figure at 10 years. How to set policy in relation to these values depends on national and regional health standards. A Canadian cohort analysis that used a multiprovince life-table model determined that the most cost-effective rate at which latent AAAs should be detected is 20% per year, which corresponds to a screening interval of 5 years by abdominal ultrasonography for patients over 50 years of age (1038), but the aortic dimensions at which intervention was recommended were larger than those that recently have been used in influential randomized trials (962,963). In Finland, 74% (238 of 322) of first-degree relatives of 150 consecutive AAA patients were screened at a central university hospital to evaluate the effectiveness and costs of treatment (1039). Outcomes were assessed with the national discharge registry and from survival analysis of AAA patients who underwent elective or emergency surgery. The incremental effectiveness in life-years gained by the screening of male siblings was 92 years, with an incremental cost-effectiveness ratio of 33 000 Finnish marks or $6200. Given these data, screening of male siblings of AAA patients was recommended because it appeared to be associated with improved survival at low cost.

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%, or men who smoke) and the use of a limited ultrasound scan are more cost-effective than conventional abdominal imaging of unselected populations. In a small pilot study, the average time required to perform a limited screening scan was one sixth that of a conventional study (4 vs. 24 minutes), with comparable accuracy for the diagnosis of AAA alone (1040). Reducing the cost of screening tests from $259, which represents the approximate Medicare reimbursement for conventional abdominal ultrasound imaging, to $40 for the limited scan would improve cost-effectiveness.

A meta-analysis of the currently published international data that might support the use of screening programs to detect AAA has been completed recently and was summarized by the United States Preventive Services Task Force (USPSTF). This summary provides a concise focus on the potential benefit and harm that might be associated with such targeted AAA screening programs, balancing detection efficacy, interventional risk reduction, and cost-effectiveness (1041). A version prepared for the Agency for Healthcare Research and Quality in February 2005 is available online at www.ahrq.gov/clinic/serfiles.htm. The USPSTF 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). It is notable that the data do not support the application of AAA screening for men who have never smoked or for women. The USPSTF analysis balanced the efficacy of AAA detection and potential diminution of AAA-associated death by surgical repair with the potential psychological harm and increased morbidity and mortality of AAA surgery performed in low risk populations.

There are important caveats to be applied to any screening recommendations. These include the need for the screening intervention to be performed in individuals whose life expectancy is adequately long for benefit to accrue (thus, decreasing benefit is gained in more elderly populations with ages greater than 75 years) and that the use of endovascular (vs. open surgical) aortic repair is likely no more beneficial in the long-term risk-benefit calculation, because there are inadequate data to demonstrate that use of endovascular techniques would be associated with any greater benefit than with operative repair. Finally, AAA screening has not been proven to be linked to an improvement in all-cause mortality, even when AAA-associated death is diminished. These limitations may have significant impact on the willingness of

screening candidates to participate in this screening pathway. Finally, the USPSTF analysis suggested that screening performed as per the Multicentre Aneurysm Screening Study (MASS) would be associated with a cost-effectiveness ratio for population-based AAA screening (compared with no screening) in the range of $14 000 to $20 000 per quality-adjusted life-year. Although this estimate is promising, additional data are required to confirm that these estimates are accurate over longer periods of time in actual (vs. clinical trial) practice (1042).

5.2.5. Observational Management

5.2.5.1. Blood Pressure Control and Beta-Blockade

RECOMMENDATIONS

Class I

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

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

Preclinical models of aneurysm progression have suggested that beta-adrenergic antagonist agents may reduce the risk of aneurysm development and expansion. Brophy et al. (1043) demonstrated that propranolol delays the development of aneurysms in a mouse model that is prone to spontaneous aortic aneurysms. In that model, drug efficacy appeared to be independent of reductions in blood pressure or diminution of the force of left ventricular ejection (dP/dt) and may have resulted from actions on the connective tissue structure of the aortic wall. In another animal model in which AAAs were induced both in normotensive and in genetically hypertensive rats by perfusion of the isolated infrarenal aorta with elastase for 2 hours, the aneurysms were significantly larger in hypertensive rats, with a mean expansion rate (mm per day) that was nearly twice that of normotensive animals (1044). In comparison, the aneurysms in the study by Brophy et al. were significantly smaller in hypertensive propranolol-treated rats than in placebo-treated controls (p less than 0.05).

Retrospective clinical studies have suggested that beta-adrenergic antagonist agents might reduce the risk of aneurysm expansion and rupture (1045), but these data have been inconsistent. In one small retrospective analysis, the mean aneurysm growth rate was 0.17 cm per year in treated patients versus 0.44 cm per year in untreated patients (1046). Eight percent of the patients in the beta-blockade group exhibited a growth rate that exceeded the mean for the overall study population, compared with 53% of the patients who received no treatment. The mean rate of aneurysm expansion was slower in treated patients, a difference that was most pronounced in those with large aneurysms. Lindholdt et al. reported another study of 54 patients who had small AAAs who were randomized to receive 40 mg of propranolol twice daily or placebo and were followed up for 2 years (1047). Sixty percent of the subjects in the propranolol group and 25% of those in the placebo group ultimately withdrew from this trial, with many subjects in the propranolol group reporting problems with dyspnea. Reductions in pulmonary function, ABI, and quality of life were also observed in the propranolol group. The mortality rate was 17% in the propranolol group compared with 4.2% in the placebo group (risk reduction 1.6; 95% CI 1.02 to 2.51). However, the relative risk of aneurysm expansion at an annual rate of more than 2 mm in the placebo group was 1.17 (95% CI 0.74 to 1.85) by intention-to-treat analysis and 2.44 (95% CI 0.88 to 6.77) according to on-treatment analysis. Only 22% of the treated patients continued to take propranolol for the full 2 years. In another trial, asymptomatic patients with AAAs measuring 3.0 to 5.0 cm in diameter were randomized in a double-blind fashion to receive either propanolol (n equals 276) or placebo (n equals 272) and then observed for a mean of 2.5 years (601). Forty-two percent of the patients in the propranolol group discontinued their medication compared with 27% of those in the placebo group (p equals 0.0002). The annual aneurysm growth rate was similar for the propanolol (0.22 cm per year) and placebo (0.26 cm per year, p equals 0.11) groups. There was a slight trend towards more elective surgical intervention in the placebo group (27% vs. 20%, p equals 0.11), but there was no difference in mortality rates (propanolol 12%, placebo 9%; p equals 0.36). Patients in the propranolol group had significantly poorer quality-of-life scores. Finally, one prospective randomized trial found that the expansion rate of AAAs was not attenuated by use of beta-adrenergic blockers (601).

Long-term prophylactic beta-blockade appears to be effective in slowing the rate of aortic dilation and decreasing the incidence of aortic complications in some patients with Marfan syndrome by reducing the heart rate and the impulse (i.e., the rate of pressure change in the aortic root) of left ventricular ejection. An open-label, randomized trial of propranolol (mean dose 212 plus or minus 68 mg daily) in adolescent and adult patients with classic Marfan syndrome determined that the rate of aortic root dilation was significantly lower in the treatment group than in the control group (0.023 vs. 0.084 cm per year, p less than 0.001) (1048). Clinical end points were reached in 5 patients in the treatment group and 9 in the control group. The Kaplan-Meier survival curve for the treatment group differed significantly from that for the control group during the middle years of the trial and remained better for the treatment group throughout the study. It is not clear whether these observations apply to aneurysms in the abdominal aorta, because patients with Marfan syndrome develop aneurysms less commonly in this location than in the thoracic aorta.

Aside from their effects on aneurysm size, the perioperative administration of beta-blockers may reduce the risk of adverse cardiac events and death in patients with cardiac risk factors who undergo AAA repair and other noncardiac vascular surgery (1049-1051).

5.2.5.2. 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 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, respectively (1052). Given the usual slow rate of expansion for truly small aneurysms, however, Grimshaw et al. and Santilli et al. have recommended that those measuring less than 4.0 cm in diameter can be followed up safely with ultrasound scans every 2 to 3 years (933,934).

The available evidence does not support a lower size threshold for the endovascular repair of AAAs than for conventional surgical repair (1053,1054). No recommendations currently are available for patients whose aortic diameter is ectatic but less than 3.0 cm in diameter and thus not truly aneurysmal. Screening of 12 500 people at a university-affiliated VA medical center yielded 223 patients whose aortic diameters were 2.5 to 2.9 cm (1055). On the basis of serial ultrasound imaging over 7 years, these ectatic aortas expanded slowly, rupture did not occur, and criteria for operative repair were infrequently met. No risk factors linked to the development of aneurysms were identified on multivariate analysis. Therefore, in patients with ectatic but nonaneurysmal aortas, repeat ultrasound imaging was recommended no more often than 5 years after the initial study. Because of the potential for late dissection or aneurysm in other areas of the aorta, however, patients with Marfan syndrome should undergo serial imaging of the aorta indefinitely after surgical repair of aneurysmal disease or dissection.

5.2.6. Open Aortic Aneurysm Repair

The management of patients who have AAAs that are large enough to represent a predictable risk for fatal rupture often is guided by several considerations. First, the survival rate of this patient population generally is acknowledged to be significantly lower than that for a normal population of the same age (1056-1059), and Aune has reported that unfavorable late survival is particularly evident among patients who are 65 years of age or younger at the time that their aortic aneurysms are discovered (1058). 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 (945,1060). 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, FDA-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, the contemporary clinician is faced with an array of choices in the management of aortic aneurysms, each of which must be tailored to the individual patient.

5.2.6.1. Infrarenal AAAs

5.2.6.1.1. 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 (955,1061-1064). Glance constructed a Markov predictive model in which patients at high cardiac risk underwent coronary arteriography, those at intermediate risk received noninvasive assessment with dipyridamole-thallium scanning, and those at low risk proceeded directly to aneurysm repair (1064). The conclusion of this exercise was that selective screening "may improve 5-year survival and be cost effective." Several large clinical series have been reported in which a similar clinical approach has been used (1065-1068). According to these reports, 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 (1069). However, the role of coronary artery revascularization in the context of contemporary medical management appears to be less than has been traditionally assumed. Intensive medical therapy and coronary revascularization (including percutaneous coronary intervention and coronary artery bypass grafting), when offered to individuals anticipated to undergo lower extremity or AAA revascularization surgery, resulted in equal postoperative rates of cardiovascular ischemic events in a prospective investigation (1069). A comprehensive discussion of this topic may be found in a previous guidelines document sponsored by the ACC/AHA (484).

5.2.6.1.2. Open Surgical Approaches. Open aortic aneurysm repair can be performed by a midline transabdominal approach or an extraperitoneal incision in the left flank, and Darling et al. have recommended that the flank approach also be used to gain expeditious suprarenal aortic control for

ruptured infrarenal aneurysms (1070). There is no clear consensus, however, regarding the superiority of either of these incisions on the basis of prospectively randomized studies. Sicard et al. found that the extraperitoneal approach was associated with fewer postoperative complications, a shorter length of stay, and lower hospital charges (1071). Other randomized institutional trials (1072,1073) have failed to demonstrate any material advantage to the routine use of the extraperitoneal approach and have suggested that it may result in a higher incidence of muscular atony, incisional hernias, and wound discomfort than a standard transabdominal incision (1073).

5.2.6.1.3. 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 (1074). Such factors undoubtedly account for some of the variability in the representative early outcomes that are summarized in Table 49. 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 United States and elsewhere generally have been associated with slightly higher mortality rates, ranging from 5% to 7%. Exceptionally large databases, such as the National Hospital Discharge Survey (NHDS) and the Nationwide Inpatient Sample, are intriguing because of their potential sample size but often require considerable editing to distinguish between infrarenal and suprarenal aortic aneurysms, both of which are classified under the same ICD-9 (International Classification of Diseases, 9th Revision) code. Lawrence et al. (1075) used the NHDS to calculate an operative mortality rate of 8.4% for 32 387 patients in 1994, but as indicated in Table 49, conflicting results can be generated from the NHDS and the Nationwide Inpatient Sample during similar periods of study (1075-1078). In comparison, the operative mortality rate for open repair of ruptured AAAs is uniformly grim, 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 (Table 50).

Table 50. Operative Mortality Rates for Open Repair of Ruptured Abdominal Aortic Aneurysms

First Author	Reference	Year (Study Period)	No. of Patients	Mortality Rate (%)
Case series				
Johansen	(943)	1991 (1980-1989)	180	69
Panneton	(1097)	1995 (1980-1992)	112	49
Seiwert	(1098)	1995 (1986-1993)	119	45
Darling	(1070)	1996 (1988-1995)	104	28
Barry	(1099)	1998 (1982-1993)	258	43
Noel	(1100)	2001 (1980-1998)	413	37
Collective reviews				
Taylor	(938)	1987	5 Reports	42
Hollier	(939)	1992 (1985-1991)	1040	48
Ernst	(1081)	1993 (1981-1992)	1731	49
Zarins	(973)	1997 (1988-1996)	1618	42
Regional or multicentered studies				
Hertzer (Northeastern Ohio)	(1101)	1984 (1978-1981)	213	33
Johnston (Canadian Aneurysm Group)	(1102)	1994	147	50
Katz (Michigan statewide)	(1086)	1994 (1980-1990)	1829	50
Kazmers (Veterans Affairs)	(1087)	1996 (1991-1993)	268	47
Wen (Ontario Aneurysm Study)	(1088)	1996 (1988-1992)	1203	40
Kantonen (Finland Vascular Registry)	(1089)	1997	454	46
Bradbury (Edinburgh Vascular Registry)	(1090)	1998 (1976-1996)	673	37
Manheim (California statewide)	(1091)	1998 (1982-1994)	7327	48
Axelrod (Veterans Affairs)	(949)	2001	52	31
Kazmers (Veterans Affairs)	(1095)	2001 (1991-1995)	427	46
U.S. hospital databases				
Lawrence (National Hospital Discharge Survey)	(1075)	1999 (1994)	6623	68
Heller (National Hospital Discharge Survey)	(1076)	2000 (1979-1997)	67 751	46
Dimick (National Inpatient Sample)	(1078)	2002	13 887	47

The clinical variables that significantly influence the mortality rate for ruptured aneurysm repair generally reflect a sudden loss of blood volume, as well as the physiological resilience of individual patients to withstand such a catastrophe. These include a low initial hematocrit, hypotension that requires resuscitation, cardiac arrest, a high APACHE (Acute Physiological And Chronic Health Evaluation) score, and advanced age (1097,1100,1101,1103-1105). In comparison, certain patient demographics and organ-specific factors take precedence over hemodynamic instability in the determination of the surgical risk for elective repair of intact aneurysms. Some of these considerations are listed below.

Age. Not surprisingly, higher patient age has been shown to be directly related to higher operative mortality rates in typical case series (1058,1060), collective reviews (1106), regional or statewide audits (1086,1092), and the UK Small Aneurysm Trial (1107). Although the operative mortality rate for urgent repair of ruptured aortic aneurysms is no higher among octogenarians than in younger patients (1099,1108), the results of 2 relatively large series indicate that the mortality rate for elective aneurysm repair in octogenarians is only slightly less than 10% (1108,1109). These findings also are supported by data from the NHDS (1075,1076) and the Nationwide Inpatient Sample (1077). Nevertheless, the mortality rate for elective operations is so much lower than for ruptured aneurysms that octogenarians should not be dismissed as surgical candidates merely on the basis of their age, provided their aneurysms are sufficiently large by contemporary standards to justify intervention (1108-1110).

Gender. Patient gender did not influence early mortality or late survival rates in series of approximately 600 patients from the Canadian Aneurysm Group (1102) or the Cleveland Clinic (1060), but this experience is far from universal. According to larger, population-based data sets in Michigan (1086); Maryland (1092); and Ontario, Canada (1088), the mortality rate for elective aneurysm repair may be as much as 50% higher among women and appears to be higher than in men for ruptured aneurysm repair (1076,1099,1100).

Race. Patient race has not been found to be an independent predictor of early mortality after elective aneurysm repair in the VA system (1111), but another large database from the NHDS suggests that the elective morality rate is significantly higher among blacks (1076). Similarly, Dardik et al. found that the elective mortality rate for blacks (6.7%) was higher than the comparable figure for other races (3.2%, p equals 0.046) in the state of Maryland during the early 1990s (1092).

Organ-Specific Risk Factors. Reports (1068,1076,1077) have confirmed the conclusions of countless previous studies that the mortality rate for elective aneurysm repair is closely related to the presence of preoperative cardiac risk factors and the severity of pre-existing renal impairment. In comparison, COPD is associated with increased morbidity, the need for prolonged ventilatory support, and longer lengths of stay in the hospital but has been shown not to be a predictor of operative mortality (949).

Volume/Outcome Relationship. 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 51. Other studies have reconfirmed these observations with respect to hospital volume (1094,1111), surgeon experience (1089), or both (1112). Manheim et al. (1091) and Dimick et al. (1078) have estimated that the operative mortality rate for elective aneurysm repair is reduced by approximately 50% in high-volume hospitals in the United States, and Wen et al. (1088) 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, Canada. Pearce et al. (1093) 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. (1092) 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.

5.2.6.1.4. Late Survival Rates. Representative late survival rates after open surgical repair of intact and ruptured AAAs are summarized in Table 52. Five-year survival rates after intact aneurysm repair generally have ranged from 60% to 75%, with 10-year survival rates of approximately 40% to 50%. Several other studies (1085,1095,1102,1114,1115) have determined that the long-term mortality rate is substantially higher after ruptured aneurysm repair even among operative survivors, possibly because some of these patients may have serious medical comorbidities that discouraged earlier elective intervention for their aneurysms. Several risk factors have been shown to be significant in more than 1 of these studies, including advanced age, ischemic heart disease manifested by congestive heart failure or electrocardiographic evidence of myocardial ischemia, an elevated serum creatinine level, COPD, and cerebrovascular disease (1057,1068,1085,1095,1102,1116).

5.2.6.1.5. Late Graft Complications. Late graft complications (e.g., aortic pseudoaneurysms, graft infections and/or enteric fistulas, and graft limb occlusions) are exceedingly unusual after open aortic aneurysm repair. Hallett et al. (1120) reported graft-related complications in only 9.4% of a population-based series of 307 patients who underwent open aneurysm repair at the Mayo Clinic between 1957 and 1990, which included anastomotic pseudoaneurysms in 3.0%, graft thrombosis in 2.0%, enteric fistulas in 1.6%, and graft infections in 1.3%. In another long-term study that included a substantial number of aortofemoral grafts, Biancari et al. (959) calculated survival rates free from graft complications of

Table 51. Volume/Outcome Relationships for Open Aortic Abdominal Aneurysm Repair

First Author	Reference	Year (Study Period)	No. of Patients	Overall Mortality Rate (%)	Annual Volume	
					Hospital	Surgeon
Intact aneurysms						
Hertzer (Northeastern Ohio)	(1101)	1984 (1978-1981)	840	6.50	NA	Low: 4.7%; medium: 16%; high: 2.9% (p less than 0.001)
Amundsen (Norway)	(1113)	1990	279	NA	Low: 11%; high: 4.8% (p equals 0.05)	NA
Hannan (New York statewide)	(1084)	1992 (1982-1987)	6042	7.60	Low: 12%; medium: 6.8%; high: 5.6%	Low: 11%; medium: 7.3%; high: 5.6%
Katz (Michigan statewide)	(1086)	1994 (1980-1990)	8185	7.50	Low: 8.9% High: 6.2% (p less than 0.001)	NA
Kazmers (Veterans Affairs)	(1087)	1996 (1991-1993)	3419	4.90	Low: 6.7%; high: 4.2% (p less than 0.05)	NA
Dardik (Maryland statewide)	(1092)	1999 (1990-1995)	2335	3.50	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						
Hertzer (Northeastern Ohio)	(1101)	1984 (1978-1981)	213	33	NA	Low: 32%; medium: 39%; high: 27% (p equals NS)
Amundsen (Norway)	(1113)	1990	165	NA	Low: 73%; high: 52% (p equals 0.03)	NA
Katz (Michigan statewide)	(1086)	1994 (1980-1990)	1829	50	Low: 54%; high: 46% (p equals 0.0026)	NA
Dardik (Maryland statewide)	(1092)	1999 (1990-1995)	527	47	Low: 46%; medium: 49%; high: 47% (p equals NS)	Low: 51%; medium: 47%; high: 36% (p equals 0.05)

NA indicates not available; NS, not significant.

Table 52. Late Survival Rates After Open Aortic Abdominal Aneurysm Repair

First Author	Reference	Year	No. of Patients	Survival Rates 1 Year	3 Years	5 Years	10 Years	Other
Intact aneurysms								
Case series								
Crawford	(1061)	1981	816			63%	38%	15 y: 18%
Hertzer	(955)	1987	236			72%		
Hallett	(1056)	1993	130			61%		
Stonebridge	(1117)	1993	311					8 y: 45%
Soisalon-Soininen	(1114)	1995	706			67%		
Cho	(1115)	1998	116	97%		74%	43%	
Aune	(1058)	2001	Younger than age 66 y: 118				8 y: 69%	
			66 y or older: 333				8 y: 47%	
			Total: 451				15 y: 18%	
Biancari	(959)	2002	208					
Hertzer	(1068)	2002	1135		67%	39%		
Menard	(1080)	2003	Low risk: 444	94%		75%	49%	
			High risk: 128		68%			
			Total: 572		74%			
					46%			
Collective reviews or multicenter studies								
Ernst (collective review)	(1081)	1993	3226	92%		67%	40%	
Johnston (Canadian Aneurysm Group)	(1085)	1994	680	91%	81%	68%		6 y: 60%
Feinglass (Veterans Affairs	(1116)	1995	280	89%		64%		
		1995	280	89%		64%		
Koskus (French AURC)	(1057)	1997	794	94%	84%	67%		
Norman (collective review)	(1118)	2001	32 Reports		70%			
Ruptured aneurysms								
Case series								
Stonebridge	(1117)	1993	227				8 y: 40%	
Soisalon-Soininen	(1114)	1995	Operative survivors: 364		60%			
Cho	(1115)	1998	Operative survivors: 116	86%		64%	33%	
Evans	(1119)	1999	Operative survivors: 115	88%		59%	26%	
Collective reviews or multicenter studies								
Johnston (Canadian aneurysm study)	(1102)	1994	147				6 y: 22%	

AURC indicates Association for Academic Research in Vascular Surgery.

94% at 5 years, 88% at 10 years, and 74% at 15 years. Only 2.9% of the patients in that series developed aortic pseudoaneurysms, and the higher rates of distal anastomotic pseudoaneurysms (8.7%) and graft limb occlusions (5.3%) that occurred in the series almost certainly were related to the fact that the majority (55%) of the replacement grafts extended below the inguinal ligament. Hertzer et al. (1068) reported a modern series of 1135 open aneurysm procedures that were collected from 1989 through 1998, were performed with monofilament suture material, and included relatively few aortofemoral grafts (5%). Only 0.4% of these patients have required reoperations for graft complications.

5.2.6.2. 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 (1121). Open repair of juxtarenal or pararenal aortic aneurysms may be accomplished through a midline transabdominal incision with or without medial visceral rotation of the spleen, the pancreas, and sometimes the left kidney, depending on the preference of the surgeon. These aneurysms also can be repaired with a thoracoretroperitoneal approach, which almost always is necessary for type IV thoracoabdominal aneurysms. 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.

5.2.6.2.1. Early Mortality and Complication Rates Juxtarenal Aortic Aneurysms. 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. This is not always evident on preoperative imaging because of angulation of the aorta or superimposition of the aneurysm over the infrarenal cuff (1121). 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. Taylor et al. encountered no postoperative deaths after juxtarenal aneurysm repair, but 7% of their patients experienced at least transient renal failure (1013). In a series of 53 juxtarenal aneurysms and 376 infrarenal aneurysms, Ayari et al. reported early mortality rates of 11% and 3% (p less than 0.01) and morbidity rates of 51% and 26% (p less than 0.01), respectively (1122). Faggioli et al. described a series of 50 juxtarenal or pararenal aneurysms in which the operative mortality rate of 12% was significantly worse (p less than 0.02) than the comparable figure for all infrarenal aneurysm procedures that were done at the same center (1123).

Pararenal/Suprarenal and Type IV Thoracoabdominal Aortic Aneurysms. Selected but representative data regarding the operative mortality and complication rates for all upper AAAs involving the renal arteries are presented in Table 53. In aggregate, the mortality for elective repair of type IV thoracoabdominal aneurysms is approximately twice as high as that for pararenal or "low" suprarenal aneurysms. All of these aneurysms share the requirement for suprarenal aortic cross-clamping and usually for additional reconstruction of the left renal artery, either by reimplantation or with the use of an independent renal artery graft that originates from the aortic prosthesis. Accordingly, a period of renal ischemia is unavoidable unless continuous kidney perfusion is used, and for this reason, postoperative renal insufficiency is the most common organ-specific complication that is generic to the repair of any aortic aneurysm arising at or above the level of the renal arteries. A transient elevation in the serum creatinine can be expected in 20% to 30% of these patients, with temporary hemodialysis support being necessary in 3% to 15%. Fortunately, however, permanent renal failure generally has been reported in fewer than 5% of patients. The risk of spinal cord ischemia with paraplegia is less than 5% for type IV thoracoabdominal aneurysms but otherwise is distinctly uncommon.

The operative mortality rate for aneurysms that involve the upper abdominal aorta has been shown to be related to patient age and the presence of coronary artery disease (1123), as well as to whether the aneurysm extends to the level of the diaphragm and/or requires urgent rather than elective surgical treatment (1133). The risk for postoperative renal insufficiency can be correlated with the severity of intrinsic renal artery disease and the extent of revascularization that is necessary to correct it, particularly when both renal arteries require additional reconstruction (1124,1125).

5.2.6.2.2. Late Survival Rates. According to the data that are available, the late survival rate after repair of juxtarenal, pararenal, or suprarenal aortic aneurysms may be slightly lower than after operations for infrarenal aortic aneurysms. Schwartz et al. (1131) and Martin et al. (1133) have reported 5-year survival rates of 50%, whereas the 5-year survival rate was only 40% in the series described by Faggioli et al. (1123).

Table 53. Operative Mortality and Postoperative Complication Rates for Open Repair of Pararenal, Suprarenal, and Type IV Thoracoabdominal Aortic Aneurysms

First Author	Reference	Year (Study Period)	No. of Patients	Mortality Rate (%)	Postoperative Complication Rates (%)		
					Renal	Paraplegia	Other
Pararenal or suprarenal							
Qvarfordt	(1124)	1986	77	1.3	Transient: 23 Dialysis: 2.5	NA	5
Nypaver	(1125)	1993 (1985-1992)	53	3.8	Transient: 23 Dialysis: 5.7	NA	NA
Faggioli	(1123)	1998	50	12	NA	NA	NA
Jean-Claude	(1126)	1999 (1977-1997)	257	5.8	Transient: 30 Sustained: 9.3 Dialysis: 7.0	0.4	31
Anagnostopoulos	(1127)	2001 (1986-1999)	65	0	Total: 42 Dialysis: 9.2 Permanent: 1.5	0	NA
Type IV thoracoabdominal							
Crawford	(1121)	1986 (1960-1985)	145	4.8	Dialysis: 5.5	2.1	NA
Cox	(1128)	1992 (1966-1991)	42	Total: 31 Elective: 12 Urgent: 55	NA	Total: 11 Elective: 4.3 Urgent: 20	NA
Svensson	(1129)	1993 (1960-1991)	346	5.8	Total: 22	4.3	NA
Coselli	(1130)	1995 (1984-1993)	35	14 (reoperations)	None permanent	2.9	NA
Schwartz	(1131)	1996 (1977-1994)	58	5.3	Transient: 31 Sustained: 28 Dialysis: 8.8 Permanent: 1.9	1.8	42
Dunning	(1132)	1999 (1995-1998)	26	12	Dialysis: 3.8	3.8	42
Martin	(1133)	2000 (1989-1998)	165	Total: 11 Elective: 7.2 Urgent: 22	Transient: 19 Dialysis: 14 Permanent: 3.0	3.6	56

NA indicates not available.

5.2.7. Endovascular Aortic Aneurysm Repair

5.2.7.1. Introduction

The technique of transfemoral catheter-based repair of infrarenal AAAs was first reported by Parodi et al., originally as an alternative for the management of patients whose medical comorbidities made them poor candidates for conventional surgical treatment (1134). A variety of proprietary stent grafts and delivery systems now have been used for more than a decade throughout the world, 4 of which presently have market approval by the FDA and remain commercially available in the United States. Open exposure of the common femoral arteries conventionally is used for sheath placement in most patients, and extraperitoneal incisions occasionally are necessary to construct temporary access conduits to 1 or both iliac arteries if the external iliac arteries are too small or tortuous for transfemoral cannulation. Endovascular AAA repair can avoid 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 AAA who have severe cardiopulmonary disease or other risk factors, such as advanced age, morbid obesity, or a hostile abdomen from multiple previous operations. Once its feasibility had been demonstrated in such patients, however, endovascular repair also has been offered at many centers to low- or average-risk patients who have no 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 (1135).

Driven by necessity and a competitive medical marketplace, the design of aortic stent grafts has passed through several iterations. 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. Thus, the aortic stem and a contiguous iliac limb are inserted through 1 femoral artery, with the opposite iliac limb then being positioned by a separate delivery system through the contralateral femoral artery. 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. Better graft stability with a transrenal attachment will likely improve results but does not necessarily mean that patients with aneurysms with shorter necks can be treated, because the proximal seal of the endovascular graft continues to be infrarenal in all currently approved devices. In aggregate, modular externally supported bifurcation endografts are more widely applicable, less prone to migrate 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 (1136,1137), these include anatomic limitations, intrasac endoleaks, graft occlusion, and aortic neck expansion.

5.2.7.1.1. Anatomic Limitations. Even with suprarenal fixation of its metallic exoskeleton, the fabric component of an endograft obviously cannot be permitted to overlap the origins of the renal arteries. Accordingly, at least 1 cm of proximal aortic cuff (1.5 cm for commercially available grafts) presently is optimal for elective endograft repair below the renal arteries. For devices without a suprarenal fixation device, the optimum infrarenal aortic diameter at the time of this writing is 25 mm or less, and for devices with a suprarenal fixation component, it is 28 mm or less. 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 men (1138,1139). 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) (1140). Becker et al. (1141) also found that significantly fewer women qualified for endovascular aneurysm repair (26% of women vs. 41% of men), and Mathison et al. (1142) 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 in men (57%), but the women in that 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 (1143).

5.2.7.1.2. Intrasac Endoleaks. Endoleaks represent sources of continued blood flow into the excluded aneurysm sac and are of such importance that they justified a consensus conference of experts in endovascular aneurysm repair in 2000 (1144). 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 proce-

dure 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 all 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 (1145,1146), they do not yet appear to influence the risk for rupture during 18 to 36 months of surveillance in large series of patients (1147,1148). If an intervention is necessary for the few type II endoleaks that persist or are associated with aneurysm expansion, therapeutic embolization of feeding branches through a translumbar approach to the aneurysm sac has been successful. Type III endoleaks are caused by midgraft defects from fabric tears or the junctional disruption of modular graft components, especially if these components become 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 computed tomographic scans.

In summary, it is largely because of the uncertainties related to intrasac endoleaks that clinical investigators and the FDA 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 (1144,1149). If persistent endoleaks or continued aneurysm expansion is demonstrated, further studies are necessary to determine the cause. Perhaps the most active area of current interest in this regard is related to the management of type II endoleaks, largely because of the frequency with which they occur and both the inconvenience and expense of their treatment. According to European collaborators registry on stent-graft techniques for abdominal aortic aneurysm repair (EUROSTAR) data for follow-up intervals as long as 6 years, the presence of type II endoleaks has not been associated with a significant incidence of any adverse clinical events other than the secondary interventions that are performed at the discretion of the attending physicians (1150). Similar findings have led Steinmetz et al. to conclude that selective intervention should be considered only for type II endoleaks that have persisted for at least 6 months on serial noninvasive imaging (1151).

5.2.7.1.3. Graft Occlusion. Occlusion of the iliac limbs of bifurcation endografts was not uncommon with early devices, occurring in 10% of some series (1152). After finding that further intraluminal stenting was necessary to eliminate torsion or kinking in 36% of all unsupported grafts, Amesur et al. adopted the use of routine intraoperative intravascular ultrasonography to identify these potential problems and to correct them before thrombosis occurred (1153). Graft occlusion may become a less frequent complication in the future, because the stability of a metallic skeleton tends to prevent the kinds of graft distortion that can lead to subsequent thrombosis. Although Baum et al. encountered limb kinking in a total of 12% of grafts in their series, they were able to document this finding in only 5% of externally supported grafts compared with 44% of unsupported grafts (1154). In a multicenter study of 242 unsupported bifurcation endografts that were implanted from 1995 through 1998, Fairman et al. reported an overall primary patency rate of 62% at a mean follow-up interval of 31 months (1155). The primary-assisted and secondary patency rates for this series were 94% and 97%, however, because of successful intraoperative (28%) or postoperative (12%) graft limb interventions that were necessary in 40% of the 242 patients.

5.2.7.1.4. Aortic Neck Expansion. Endograft migration from the proximal attachment site has been reported in a wide range of 1.5% to 16% of patients (1024,1156,1157). One of the factors that could lead to graft migration or delayed type I endoleaks is further expansion of the proximal aorta, a finding that Makaroun et al. have documented by serial imaging studies in 13% of patients at 1 year after endovascular aneurysm repair, in 21% at 2 years, and in 19% at 3 years (1158). According to Matsumura et al., the mean increase in aortic neck diameter after endografting is 0.7 plus or minus 2.1 mm at 1 year and 0.9 plus or minus 1.9 mm at 2 years (1159). Even when device diameters are purposefully oversized by as much as 20% in an attempt to accommodate future aortic neck expansion, Conners et al. have found that endograft migration still can occur (1157). The implications of these observations are a source of some concern, but the maximum follow-up period of approximately 3 years for most reported endograft series is too short for their influence on late clinical outcomes to be known.

5.2.7.2. 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. In an unselected series of 83 endovascular and 63 open repairs in patients who had an identical number of Eagle Criteria risk factors, de Virgilio et al. found no differences in the incidence of postoperative cardiac events (6% and 4.8%, respectively) or mortality rates (3.6% and 4.8%, respectively) (1160). Among patients who received endografts, the only predictors of cardiac events were a history of congestive heart failure (p equals 0.005) or the presence of a Q wave on the preoperative electrocardiogram. More recent-

ly, Aziz et al. have reported that perioperative cardiac events were associated with certain Eagle risk factors, such as age 70 years or older (p equals 0.026) and a history of either MI (p equals 0.024) or congestive heart failure (p equals 0.001), after aortic endografting in 365 patients (1161). Moreover, the lack of preoperative beta-blockade was associated with a higher risk for perioperative events in this nonrandomized series (p equals 0.007).

At least one study appears to confirm the intuitive impression that endografting should have less cardiac risk than a major transabdominal operation. In a concurrent series of 71 open and 49 endovascular aneurysm repairs, Cuypers et al. found that endovascular procedures were associated with a higher intraoperative cardiac index (p less than 0.01) and a lower intraoperative stroke work index (p equals 0.04) than open procedures (1162). Although the number of adverse cardiac events was comparable, postoperative electrocardiograms and transesophageal echocardiograms revealed significantly more evidence of myocardial ischemia after open operations (57% vs. 33% after endograft repair; p equals 0.01). 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 (484).

5.2.7.3. Early Mortality and Complication Rates

Table 54 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 FDA- and industry-sponsored device trials in the United States, and from 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 54 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. With the exception of the specific device trials, most of these reports describe results with a wide variety of proprietary endografts, each of which appears to be associated with a declining complication rate after sufficient experience has been accumulated with its use at individual centers (1141,1163-1166). Data regarding volume/outcome relationships are not yet available for endovascular aneurysm repair.

The early mortality rate for endograft repair generally has been less than 3%, but May et al. (1165) 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, however, 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-, average-, and low-risk patients. Using a scoring system for preoperative risks that ranged from zero (low) to 3 (high) in a large series of 305 patients, Becker et al. (1141) calculated the mortality rates for endovascular repair to be 2.5%, 0.8%, 3.4%, and 6.5%, respectively. 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 (1163,1166,1192), as well as among those who needed adjunctive procedures in addition to the placement of an aortic stent graft (1163). Walker et al. also found significant differences between mortality rates for endovascular repair in high- and low-risk patients (16% vs. 3.7%, p equals 0.02) (1193). 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) (1135).

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 (1194-1196). Nevertheless, these and other studies (1197-1199) 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 US) of most endografts. Despite its shorter length of stay and an earlier return to normal activity, aortic endografting does not appear to be associated with superior late functional outcome or longer quality-adjusted life expectancy than open surgical treatment (1200,1201).

5.2.7.4. Late Survival and Complication Rates

Representative data regarding late survival rate and the incidence of aneurysm rupture, delayed or persistent endoleaks, and endograft reinterventions are provided in Table 55. The follow-up interval is 3 years or less for much of the information in Table 55, and the methods that were used to calculate outcomes (i.e., crude vs. cumulative) are inconsistent. In addition, according to a 1999 report (1202), only 45% of the expected 18-month follow-up results for the first 899 aortic endografts in the EUROSTAR experience had been submitted to its central registry office. The current acquisition rate for this database is not known.

Table 54. Representative Early Results for Endovascular Repair of Infrarenal Aortic Abdominal Aneurysms

First Author (Study/Sponsor)	Reference	Year (Study Period)	No. of Patients	Immediate Open Conversion (%)	Postoperative Complication Rates (%)		
					Total	Persistent	Procedural Mortality Rate (%)
Case series							
Blum	(1167)	1997 (1994-1996)	295	1.7	8.1	NA	0.7
Stelter	(1152)	1997 (1994-1997)	201	2	9	NA	3.5
May	(1168)	1998 (1992-1996)	Endo: 108 Open: 195	12	14	11	5.6 5.6
Amesur	(1169)	1999 (1996-1998)	54	NA	39	13	NA
Becquemin	(1170)	2000 (1995-1999)	Endo: 73 Open: 195	None	23	9.6	2.7 2.8
Chuter	(1164)	2000 (1996-1999)	High risk: 116	None	NA	10	1.7
Zarins	(1171)	2000 (1996-2000)	149	1.30	36	18	1.3
Blum	(1172)	2001 (1994-2001)	1994-1996: 111 1996-1997: 159 1998-2001: 28	3.6 0.6 None	14 3.1 11	NA NA NA	Total: 8.1
Becker	(1141)	2001 (1994-2001)	305	1.30	23	17	2.6
Fairman	(1173)	2001 (1998-1999)	75	None	44	20	0
Holzenbein	(1174)	2001	173	1.2	4.6 (Type I)	NA	2.8
Howell	(1175)	2001	215	None	42	11	0
Mathison	(1142)	2001 (1994-2000)	305	1.3	23	NA	2.6
May	(1165)	2001 (1995-1998)	Endo: 148 Open: 135	0.7	6.8	5.4	2.7 5.9
Sicard	(1176)	2001 (1997-2000)	Endo: 260 Open: 210	0.8	13	3	1.9 2.9
Abraham	(1146)	2002 (1998-2001)	116	None	15	11	0.9
Dattilo	(1177)	2002 (1994-2000)	362	1.40	NA	NA	1.5
Sampram	(1178)	2003 (1996-2002)	703	NA	NA	NA	1.7
Ouriel	(1179)	2003 (1996-2002)	606 Men 98 Women	NA NA	NA NA	NA NA	1.3 3.1
Shames	(1180)	2003 (1999-2001)	302 Men 42 Women	0.5 14	NA NA	NA NA	1.5 2.3
Anderson (New York State)	(1135)	2004 (2000-2002)	Endo: 1706 Open: 3063	NA	NA	NA	1.1 4.0

Continued on Next Page

Table 54. *Continued*

First Author (Study/Sponsor)	Reference	Year (Study Period)	No. of Patients	Immediate Open Conversion (%)	Postoperative Complication Rates (%)		
					Total	Persistent	Procedural Mortality Rate (%)
Device trials							
Moore (Endovascular Technologies)	(1181)	1996 (1993-1994)	46	15	44	21	0
Coppi (Stentor, Mintec)	(1182)	1998 (1995-1996)	66	6.10	6.1	3	1.5
Matsumura (Endovascular Technologies)	(1159)	1998 (1993-1995)	68	13	47	24	0
Becquemin (Vanguard, Boston Scientific)	(1183)	1999 (1996-1997)	75	None	31	9.3	0
Zarins (AneuRx, Medtronic)	(1184)	1999 (1996-1997)	Endo: 190 Open: 60	None	21	8.9	2.6 0
Zarins (AneuRx, Medtronic)	(1185)	2000 (1997-1998)	425	1.20	Centers: 38; core lab: 50	13	1.4
Beebe (Vanguard, Boston Scientific)	(1186)	2001 (1997-1998)	Endo: 268 Open: 98	1.90	5.70	2.7	1.5 3.1
Greenberg (Zenith, Cook)	(1156)	2001 (1995-2000)	528	0.80	16	5.5	0.2
Faries (Talent, Medtronic/AVE-Worldmedical)	(1187)	2002 (1999-2001)	368	1.10	12	4.8	1.9
Matsumura (Excluder; WL Gore & Associates	(1188)	2003 (2000-2002)	Endo: 235 Open: 99	None	22	17	0.9 0
EUROSTAR							
Buth	(1163)	2000 (1994-1999)	1554	1.70	16	0.9	2.6
Harris	(1189)	2000 (1996-2000)	2464	1.30	17	8.3	3.2
Vallabhaneni	(1190)	2001 (1994-2000)	2812	NA	NA	NA	2.9
Buth	(1166)	2002 (1996-2001)	3075	1.70	17	NA	2.5
Peppelenbosch	(1191)	2004 (1996-2002)	1962 (4.0 cm to 5.4 cm) 1528 (5.5 cm to 6.4 cm) 902 (over 6.4 cm)	1.1 1.4 2.3	3.7 (Type I) 6.8 (Type I) 9.9 (Type I)	NA NA NA	1.6 2.6 4.1

EUROSTAR indicates European collaborators registry on stent-graft techniques for abdominal aortic aneurysm repair; NA, not available.

Table 55. Representative Late Results for Endograft Repair of Infrarenal Abdominal Aortic Aneurysms

First Author (Study/Sponsor)	Reference	Year (Study Period)	No. of Patients	Aneurysm Rupture	Late Endoleaks	Endograft Reinterventions		Survival Rate
						Endo	Open	
Case series								
Stelter	(1152)	1997 (1994-1997)	201	None	9.50%	11%	10%	NA
May	(1168)	1998 (1992-1996)	Endo: 108 open: 195	None	6.30%	Total 7.4% (median, 29 mo)	NA	
Amesur	(1169)	1999 (1996-1998)	54	None	13%	17%	None	NA
Amesur	(1153)	2000 (1996-1999)	130 Limbs	NA	NA	36% of limbs	None	NA
Becquemin	(1170)	2000 (1995-1999)	Endo: 73; open: 107	4.1%	NA	Total 21% cumulative (1 y)		Endo: 82%; open: 96% (1 y)
Baum	(1154)	2000	Unsupported: 27 limbs; supported: 122 limbs	NA	NA	Unsupported: 44%; supported: 5% (*p* less than .001)	NA	NA
Chuter	(1164)	2000 (1996-1999)	High risk: 116	0.9%	7.8%	15%	2.6%	82% (Mean, 16 mo)
Zarins	(1147)	2000 (1996-2000)	149	None		Total 17% (median, 11 mo)		90%
Becker	(1141)	2001 (1994-2001)	305	0.7%	NA	Total 9.8%	70% (5 y)	
Holzenbein	(1174)	2001	173	0.6%	NA	Total 22% (median, 18 mo)	NA	
Howell	(1175)	2001	215	None	12%	Total 10% (maximum, 2 y)	94%	
May	(1165)	2001 (1995-1998)	Endo: 148; open: 135	1.4%	5.4%	4.7%	2.7%	Endo: 96%; open: 85% (3 y)
Ohki	(1203)	2001 (1992-2000)	239	0.8%	8.8%	5.9%	3.8%	78% (Mean, 16 mo)
Sicard	(1176)	2001 (1997-2000)	Endo: 260; open: 210	None	4.2%	2.7%	1.2%	Endo: 91%; open: 86% (3 y)
Abraham	(1146)	2002 (1998-2001)	116	0.9%	4.3%	2.6%	2.6%	NA (mean, 10 mo)
Datillo	(1177)	2002 (1994-2000)	362	0.8%	NA	11%	2.2% Late conversion	NA
Sampram	(1178)	2003 (1996-2002)	703	0.4%	23%	15% (Total)		70% (3 y)
Ouriel	(1204)	2003 (1996-2002)	416 (Size less than 5.5 cm)	0.2%	1.4% (Type I)	NA	1.4% Conversion	86% (24 mo)
			284 (Size 5.5 cm or more)	0.7%	6.4%(Type I)	NA	8.2% Conversion	71% (24 mo)
Ouriel	(1179)	2003 (1996-2002)	606 Men; 98 women	Men: 0.3%; women: 1.0%	Men: 30%; women: 35% (12 mo)	Men: 24%; women: 21% (total)		Men: 80%; women 78% (24 mo)
Shames	(1180)	2003 (1999-2001)	203 Men; 42 women	None	Men: 11%; women: 21%	Men: 9%; women: 29% (total)		Men: 95%; women: 90% (mean, 11 mo)

Continued on Next Page

Table 55. *Continued*

First Author (Study/Sponsor)	Reference	Year (Study Period)	No. of Patients	Aneurysm Rupture	Late Endoleaks	Endograft Reinterventions		Survival Rate
						Endo	Open	
Device trials								
Becquemin (Vanguard; Boston Scientific)	(1183)	1999 (1996-1997)	75	1.3%	6.70%	24%	4%	86% (25 mo)
Zarins (AneuRx; Medtronic)	(1184)	1999 (1996-1997)	Endo: 190; open: 60	None	9.00%	5.9%	None	Endo: 96%; open: 97% (1 y)
Zarins (AneuRx; Medtronic)	(1185)	2000 (1996-1999)	1046	0.7% (mean, 16 mo)	NA	NA	NA	NA
Zarins (AneuRx; Medtronic)	(1171)	2000 (1997-1998)	398	0.3%	13% (Centers) 20% (core lab)	4%	2%	95% (18 mo)
Beebe (Vanguard; Boston Scientific)	(1186)	2001 (1997-1998)	Endo: 268; open: 98	None	16% Cumulative (24 mo)	Total 31%; cumulative (24 mo)		Endo: 85%; open: 80% (24 mo)
Zarins (AneuRx; Medtronic)	(1148)	2001 (1996-1999)	1192	0.8%	NA	Total 12%; cumulative (3 y)		86% (3 y)
Faries (Talent; Medtronic/AVE-Worldmedical)	(1187)	2002 (1999-2001)	368	0.5%	4.8% (12 mo)	3%	3%	89% (7.3 mo)
Matsumura (Excluder; WL Gore)	(1188)	2003 (2000-2002)	Endo: 235; open: 99	None	20% (24 mo)	11%	1.7%	Endo: 87%; open: 93% (24 mo)
Zarins (AneuRx; Medtronic)	(1137)	2003 (1996-1999)	1193	1.3%	14%	NA	4.1% Late conversion	62% (4 y)
EUROSTAR								
Cuypers (endoleak study)	(1202)	1999 (1994-1998)	899	NA	26% total 10% persistent	NA	NA	88% (18 mo)
Cuypers (conversion study)	(1205)	2000 (1994-1999)	1871	NA	NA	NA	2.6% overall conversion	NA
Harris	(1189)	2000 (1996-2000)	2464	1% annual	15%	NA	2.1% annual conversion	75% (4 y)
Laheij	(1206)	2000 (1996-1999)	1023	NA	NA	14%	4%	NA
Vallabhaneni	(1190)	2001 (1994-2000)	2464	0.01% annual	NA	NA	2.1% annual conversion	NA
Buth	(1166)	2002 (1996-2001)	3075	0.7%	NA	NA	3.1% conversion	No risk: 88%; high risk: 75% (2 y)
Harris	(1150)	2004 (1996-2003)	4242	1.4%	30% total 10% persistent		Total 22% cumulative (5 y)	80% (5 y)
Peppelenbosch	(1191)	2004 (1996-2002)	1962 (4.0 to 5.4 cm); 1528 (5.5 to 6.4 cm); 902 (over 6.4 cm)	0.4% 0.6% 1.8%	5.3% (Type I) 4.9% (Type I) 10% (Type I)	NA NA NA	6.6% conversion 6.8% conversion 14% conversion	84% (5 y) 70% (5 y) 62% (5 y)

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

5.2.7.4.1. Survival Rates. Intermediate-term survival rates after endovascular aortic aneurysm repair primarily are influenced by antecedent risk factors, being lowest in series for which high surgical risk was a criterion for patient selection (1164,1170). Again using their scoring system (0 to 3) for stratifying incremental risk, Becker et al. (1141) calculated actuarial 1-year survival rates of 98%, 94%, 87%, and 81%, respectively. On the basis of EUROSTAR data, Buth et al. found that the cumulative 3-year survival rate was significantly lower for patients who had been deemed unfit for open repair or for general anesthesia than for the remainder of the registry population (68% vs. 83%, *p* equals 0.0001) (1166).

5.2.7.4.2. Endograft-Related Complications. Secondary interventions are common after endovascular aortic aneurysm repair and often are performed within months for limb ischemia, within 1 year for endoleaks, and after 2 years or more for graft migration (1207). Aneurysm rupture is a rare event in most series, possibly because of the recognized importance of serial computed tomography scanning to detect continued aneurysm expansion. Delayed rupture has occurred at a rate of 1% per year in the EUROSTAR population; has been significantly associated with the presence of type I or type III endoleaks, graft migration, or postoperative endograft kinking; and has a postoperative mortality rate of 58% (1189,1190). Persistent and/or delayed endoleaks occur in a wide range of approximately 5% to more than 20% of patients and are the indication for most reinterventions after endografting. Becker et al. documented endoleaks in 23% of their series (1141). Nearly half (43%) of these required intervention, whereas the remainder either resolved spontaneously (24%) or remain untreated (31%). Holzenbein et al. also reported reinterventions in 22% of their series, of which 46% were performed within 1 year of the index procedure and 74% within 2 years (1174). Ninety percent of these reinterventions were necessary to control endoleaks, whereas the remaining 10% were done to restore endograft patency. Some sources in the United States have found that graft-related complications appear to occur with greater frequency after specific devices that previously were used only in the setting of clinical trials receive market approval by the FDA. The proposed explanation for this finding is that the stringent anatomic criteria that were necessary for inclusion in the clinical trials, especially those concerning the allowable length, diameter, and angulation of the proximal infrarenal neck, may be interpreted more liberally once these devices become commercially available (1208,1209).

Zarins et al. have described further aneurysm enlargement after endograft repair in 46 (12%) of the 383 patients who entered the phase II AneuRx clinical trial from 1997 through 1998 (1210). Not surprisingly, patients with aneurysm enlargement were more likely to undergo secondary interventions (21 [46%] of 46 patients) than those with either no change (33 [17%] of 199 patients) or a reduction in postendograft aneurysm size (16 [12%] of 138 patients; *p* equals 0.0001). Open surgical conversion was performed in a total of 18 (4.7%) of the 383 patients, including 9 (20%) of the 46 patients who had experienced aneurysm enlargement after their original endograft procedures (*p* less than 0.0001). The postoperative mortality rate after open conversion was 33% in these 9 patients. According to EUROSTAR data, the annual incidence of late endograft conversion to an open operation is 2.1%, with a postoperative mortality rate of 24% (1189,1190). Overall, the crude rate of device-related complications submitted to the EUROSTAR registry declined from 22% in 1994 to 7.3% in 2000. Nevertheless, patients who had these complications were nearly 14 times more likely to require conversion procedures and were 2.4 times more likely to die than patients who did not have device-related complications (1211).

Ouriel and associates have made several observations regarding late complication rates in a large series of 703 patients who underwent endovascular repair of AAAs with either investigational or commercially available stent grafts at a single center during a 6-year period of study beginning in 1996 (1204). First, certain complications (i.e., graft limb occlusions, fabric tears, and type II endoleaks) appeared to occur more commonly with some grafts than with others and therefore may be device-specific (1204). Second, endograft repair of aneurysms that were larger than 5.4 cm in diameter was associated with a higher incidence of type I endoleaks (6.4% vs. 1.4%, *p* equals 0.011), device migration (13% vs. 4.4%, *p* equals 0.006), and conversion to open surgical repair (8.2% vs. 1.4%, *p* equals 0.031) than was the case with smaller aneurysms. Patients with larger aneurysms also had a lower survival rate (71% vs. 86%, *p* less than 0.001) and a higher risk for aneurysm-related death (6.1% vs. 2.6%, *p* equals 0.011) at 24 months of follow-up (1212). Finally, although there were no gender differences in the overall incidence of secondary interventions, graft limb occlusions occurred more frequently in women than in men (11% vs. 3.3%, *p* equals 0.022) (1179).

Others have reported similar data with respect to aneurysm size and patient gender. Peppelenbosch et al. found that EUROSTAR patients with aneurysms larger than 5.4 cm in diameter were more likely to be older and to have more preoperative risk factors, early complications, and late unrelated deaths than patients with smaller aneurysms (1191). In addition, large aneurysms often were associated with arterial anatomy (such as angulated or ectatic infrarenal necks and iliac aneurysms) that was less favorable for endograft repair and probably contributed to the significantly higher overall incidence of type I endoleaks, conversion to open surgical repair, and late rupture and/or aneurysm-related deaths that were documented in the group of patients who had large aneurysms. In another study of endograft repair in 245 patients (42 women), Shames et al. also determined that graft limb occlusions were more common among women (12% vs. 2.5%, *p* equals 0.05) (1180). Unlike Ouriel and associates (1179), however, these investigators found that women also had a higher incidence of all technical complications (17% vs. 8.3%, *p* less than 0.05) and secondary procedures (29% vs. 9.0%, *p* equals 0.001).

5.2.7.4.3. 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 56 summarizes the early and intermediate-term technical success rates from 16 previous reports. These data reconfirm 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. 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 (1213).

5.2.8. 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 computed tomography/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. Brown et al. have shown in a prospective but nonrandomized study that observation alone is a safe approach until an aneurysm undergoes a growth spurt or attains a threshold diameter of 5.0 cm (952). The success of watchful waiting is predicated on patient cooperation, however. In a similar study of 101 patients with aneurysms measuring less than 5.0 cm in diameter, Valentine et al. encountered no ruptures among patients who complied with their follow-up program compared with a 10% rupture rate among those who did not (1217). If continued surveillance is recommended, measures should be taken to control hypertension and to discourage smoking, because these risk factors are associated with accelerated rates of aneurysm growth (936,961). 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.

5.2.8.1. 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

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

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 19. This algorithm incorporates the results of the randomized UK and VA trials and takes into account the relatively limited information that yet 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 be convinced that infrarenal aneurysms should continue to be repaired at a size of only 5.0 cm or larger, 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 believe that the present technology of endovascular repair is at a state of development that justifies its general use in low- 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

Table 56. Technical Success Rates for Endograft Repair of Infrarenal Abdominal Aortic Aneurysms

Author Device/Vendor	Reference	Year (Study Period)	n	Criteria for Technical Success	Technical Success Rates Early	 Late
Case Series						
Blum	(1167)	1997 (1994-1996)	154	Successful deployment No endoleaks	87%	
Stelter	(1152)	1997 (1994-1997)	201	NA	89%	
Coppi	(1182)	1998 (1995-1996)	66	Successful deployment No endoleaks No deaths	86% (30 days)	
Hausegger	(1214)	1999	30	Successful deployment No endoleaks	83% primary 93% secondary	
Becquemin	(1170)	2000 (1995-1999)	Endo: 73 Open: 107	No endoleaks No re-intervention		74% (p=.001) 94% (1 year)
Chuter	(1164)	2000 (1996-1999)	High risk: 116	Successful deployment No endoleaks	86% (2 weeks)	
Howell	(1215)	2000	56	NA		83% primary 85% secondary (6 months)
Blum	(1172)	2001 (1994-2001)	111 (1994-1996) 159 (1996-1997) 28 (1998-2001)	Successful deployment No endoleaks	82% 96% 89%	
Ohki	(1203)	2001 (1992-2000)	239	Successful deployment No endoleaks	89%	
Device Trials						
Zarins AneuRx™ /Medtronic	(1184)	1999 (1996-1997)	190	Successful deployment No endoleaks No deaths	77%	
Zarins AneuRx™/ Medtronic	(1171)	2000 (1997-1998)	398	Survival free of aneurysm rupture, open conversion, or re-intervention for endoleaks or graft thrombosis		88% (18 months)

Continued on Next Page

Table 56. *Continued*

Author Device/Vendor	Reference	Year (Study Period)	n	Criteria for Technical Success	Technical Success Rates Early	 Late
Device Trials *(Continued)*						
Beebe Vanguard™/ Boston Scientific	(1186)	2001	240	Successful deployment No endoleaks Graft patent No deaths	89% (30 days)	
Criado Talent™/ Medtronic World Medical	(1216)	2001 (1997-2001)	High risk: 127 Low risk: 151	Successful deployment No endoleaks	86% (96% at 30 days) 88% (97% at 30 days)	
EUROSTAR						
Cuypers	(1202)	1999 (1994-1998)	899	Endoleak-free survival		79% (18 months, cumulative)
Buth	(1163)	2000 (1994-1999)	1,554	Successful deployment No endoleaks No deaths	72% (30 days)	
Laheij	(1206)	2000 (1996-1999)	1,023	Freedom from any secondary intervention		1 yr: 89% 3 yrs: 67% 4 yrs: 62%

EUROSTAR indicates European collaborators registry on stent-graft techniques for abdominal aortic aneurysm repair; n, number of patients; NA, not available.

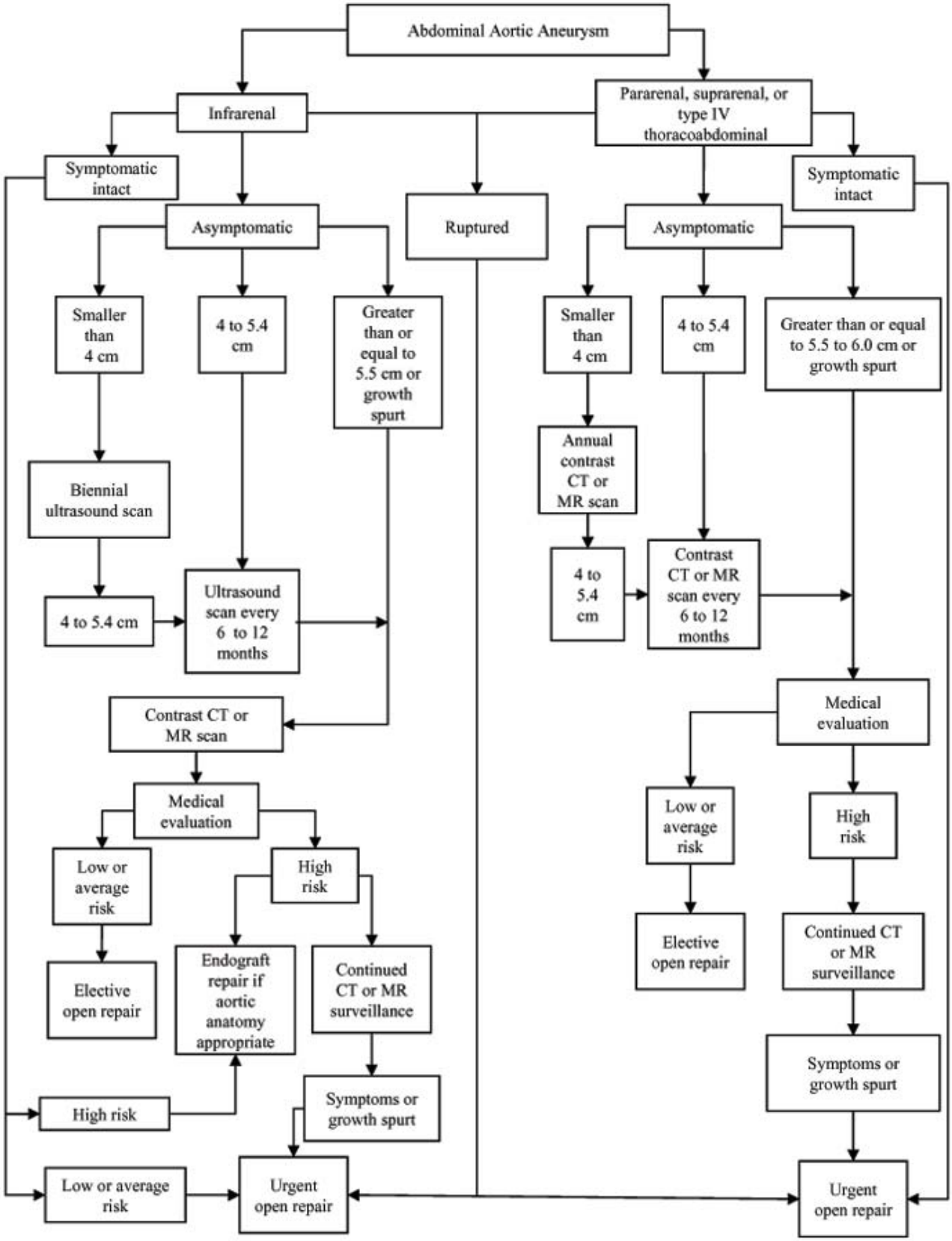

Figure 19. Management of abdominal aortic aneuryms. CT indicates computed tomography; MR, magnetic resonance imaging.

a 30-day mortality rate of 1.2% (1218). However, this report shares the current liability of many studies concerning aortic stent grafts. The mean follow-up period for these patients was only 9.6 months, during which another 8% of them required reintervention.

5.3. Visceral Artery Aneurysms

RECOMMENDATIONS

Class I

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

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 computed tomography/magnetic resonance imaging scanning. Not surprisingly, therefore, several studies have indicated that approximately half of all visceral artery aneurysms present with rupture (Table 57). 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 (1219,1220). 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 (1221-1223). 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.

5.3.1. Splenic Artery Aneurysms

Splenic artery aneurysms historically have been considered to be the most common visceral artery aneurysms (Table 58), but an increasing incidence of hepatic artery pseudoaneurysms has been described in relation to percutaneous and laparoscopic biliary procedures, as well as improved imaging

Table 57. 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	(1224)	2000	31 (20 Women)	74%	Open: 25; Endo: 9	NA	3
Carr	(1225)	2001	26/34	Ruptured: 42%	Open: 19	14% Rupture	Total: 12; ruptured: 25
Splenic							
Trastek	(1219)	1982	100 (87 Women)	17%; Ruptured: 3%	Open: 81	None at 7.4 y	1
Lee	(1223)	1999	34 (21 Women)	Ruptured: 44%	Open: 34	NA	Elective: 0; ruptured: 40
Superior mesenteric							
Stone	(1226)	2002	21 (7 Women)	52%; Ruptured: 38% (50% of men)	Open: 13; Endo: 3	None (mean size 1.8 cm)	Elective: 0; ruptured: 38
Renal							
Tham	(1227)	1983	83/89	None	Open: 14	None	0
Henriksson	(1228)	1985	21/34 (16 Women)	None	Open: 8	None	0

Endo indicates endovascular repair; NA, not available.

Table 58. Site of Visceral Artery Aneurysms

Aneurysm	%
Splenic	60
Hepatic	20
Superior mesenteric	6
Celiac	4
Others	10

Reprinted from Semin Vasc Surg, 8, Hallett JW, Jr., Splenic artery aneurysms, 321-6, Copyright 1995, with permission from Elsevier (1229).

techniques (1229,1230). Most splenic artery aneurysms are asymptomatic at the time they are recognized as an incidental finding during some type of abdominal imaging, but approximately 20% of patients present with either chronic upper abdominal pain or acute rupture (Table 59). An increasing number of splenic artery aneurysms also are being discovered in women undergoing ultrasound evaluations during pregnancy. The mortality rate for ruptured splenic artery aneurysms in patients who are not pregnant ranges from 10% to 25%, but the risk of maternal death from rupture during pregnancy has been estimated to be as high as 70%, with a fetal mortality rate of more than 90% (1231). The natural history of splenic artery aneurysms followed up through pregnancy is unknown because no large series of such patients has been collected. Nevertheless, the literature contains many case reports of pregnant women who were known to have splenic artery aneurysms that were at least 2.0 cm in diameter and that eventually ruptured during their pregnancies.

Table 59. Demographics of Splenic Artery Aneurysms (n=100)

Characteristic	Value (Range)
Gender Women:men	87:13
Mean age (years)	58.2 (16 to 81)
Mean number of pregnancies	4.5 (1 to 16)
Aneurysm size (cm)	2.1 (0.6 to 30)
Symptoms (%)	
Asymptomatic	83
Chronic	13
Rupture	4

n indicates number of patients.
Reprinted from Semin Vasc Surg, 8, Hallett JW, Jr., Splenic artery aneurysms, 321-6, Copyright 1995, with permission from Elsevier (1229).

5.3.2. Superior Mesenteric Artery Aneurysms

Superior mesenteric artery aneurysms represent only 6% to 7% of all visceral aneurysms (1226,1229). Stone et al. have described the largest series of superior mesenteric aneurysms, comprising just 21 patients who were collected from 2 large institutions during a 19-year study period (1226). Men and those patients with noncalcified aneurysms appeared to have the highest risk for rupture. Interestingly, no ruptured aneurysms happened to occur among patients who were receiving beta-blockade. The operative mortality rate for ruptured aneurysms was 38%, but there were no deaths after elective intervention (e.g., ligation, catheter embolization, or prosthetic replacement grafting) in 8 patients. None of the patients who underwent elective ligation or catheter embolization developed intestinal ischemia. This probably implies that these patients were selected very carefully on the basis of the collateral circulation that was demonstrated by their initial arteriograms, but it could also suggest that revascularization after ligation or catheter embolization sometimes can be deferred unless there is clinical evidence of ischemia. Five patients in this series who had small (diameter of 1.0 to 2.4 cm) aneurysms have been followed up with computed tomographic or ultrasound scans for 2 to 147 months without complications.

5.3.3. 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 (850,1232,1233). 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, a point that appears to be especially important in the treatment of mesenteric artery aneurysms, for which there is potential risk for bowel ischemia. Therefore, patients undergoing catheter-based intervention for visceral artery aneurysms should be watched closely after the procedure for the development of abdominal pain in the setting of mesenteric or splenic artery aneurysms and flank pain in the setting of renal artery aneurysms.

5.4. Lower Extremity Aneurysms

5.4.1. Etiology

As illustrated in Figures 20 and 21, the diameters of peripheral arteries increase approximately 20% to 25% between the ages of 20 and 70 years (865,1234). Coexistent AAAs

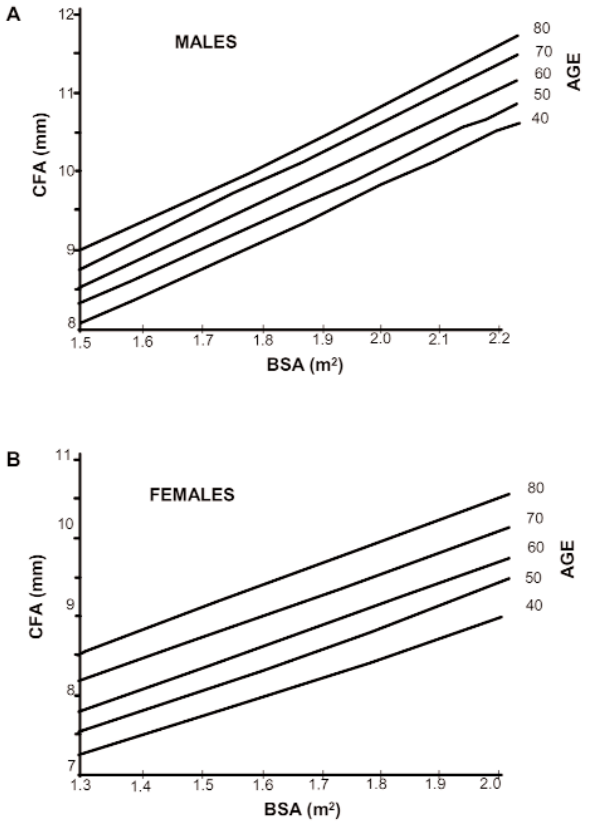

Figure 20. Predicted diameter of common femoral artery (CFA) in male and female subjects. Select appropriate curve for age marked on right and follow curve to appropriate body surface area (BSA) on horizontal axis. Predicted diameter is shown on vertical axis. Reprinted from J Vasc Surg, 29, Sandgren T, Sonesson B, Ahlgren R, et al., The diameter of the common femoral artery in healthy human: influence of sex, age, and body size, 503-10, with permission from Elsevier (1234).

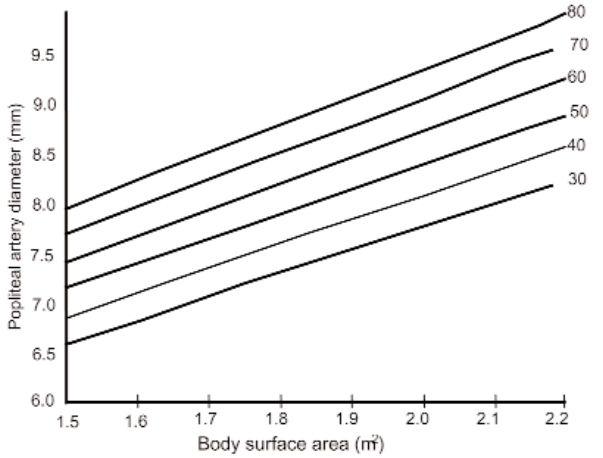

Figure 21. Predicted diameter of popliteal artery in males. To use this nomogram, select the appropriate age curve marked on the right and follow the curve to the appropriate body surface area (BSA) on the horizontal axis. The vertical axis shows the predicted diameter. Sandgren T, Sonesson B, Ahlgren AR, et al. Factors predicting the diameter of the popliteal artery in healthy humans. J Vasc Surg. 1998;28:284-9, with permission from Elsevier (865).

have been reported in 85% of patients with femoral aneurysms (1235) and in 62% of those with popliteal aneurysms (1236), whereas femoral or popliteal aneurysms are present in 3% to 7% of patients who have AAAs. It is not known whether these patients are specifically prone to diffuse aneurysm disease because of genetic or other factors or whether certain aneurysms are associated with generalized arterial ectasia elsewhere (1237-1239). The possibility that arterial aneurysm disease is a generalized process in the vascular system is supported by studies showing defective mechanical properties in the walls of distant arteries that usually do not undergo dilatation (1240,1241). When dilatation of the peripheral arteries was described in patients with AAAs more than a decade ago, the normal diameters of the studied regional arteries were unknown (1242,1243).

In an angiographic study in which arterial luminal diameters were measured, dilatation in the iliac artery was identified in patients with AAA, but the peripheral arteries in the leg were not affected (1244). The tunica media of the femoral and popliteal arteries consists largely of smooth muscle cells. The mechanical properties (and thus the integrity) of arterial walls are based on the matrix components, elastin and collagen, whereas smooth muscle cells have the potential to modulate wall mechanics. Therefore, the systemic implications of an aortic aneurysm may be different in central arteries than in peripheral arteries. In another investigation by Sandgren et al., ultrasound measurements of the anteroposterior diameters of the peripheral arteries of the right legs of 183 consecutive patients who were referred for elective repair of AAA revealed 8 common femoral aneurysms and 4 popliteal aneurysms, all in men (879). Of those in whom femoral and popliteal aneurysms were identified, occlusive PAD was present in 46% and 49%, respectively. After exclusion of those with either peripheral aneurysms or occlusive disease, no dilating diathesis was found in the limb vessels of the remaining patients with AAA.

5.4.2. Natural History

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

RECOMMENDATION

Class I

In patients with femoral or popliteal aneurysms, ultrasound (or computed tomography or magnetic resonance) imaging is recommended to exclude contralateral femoral or popliteal aneurysms and AAA. ***(Level of Evidence: B)***

5.4.2.1 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% (1245,1246). Approximately 5% of small aortic aneurysms are discovered because of lower extremity ischemia caused by distal embolization of mural thrombus (1247). However, thromboembolic complications are much more common with popliteal aneurysms, which also may be associated with arteriomegaly involving the common femoral and superficial femoral arteries. Before the introduction of modern arterial bypass grafting, Gifford et al. reported a series of 69 patients with 100 popliteal aneurysms, of which 45% were bilateral and 65% were symptomatic (1248). Only 21% of these aneurysms were treated surgically. Very few (7%) of the remaining aneurysms eventually ruptured, but 21% ultimately were associated with ischemic complications, and 23% of the 69 patients required amputations.

Although rupture has continued to be distinctly unusual in some studies, the data in Table 60 confirm many of the other observations that were made by Gifford et al. (1248). The vast majority of popliteal aneurysms occur in men, and approximately half are bilateral. Approximately half of popliteal aneurysms also are associated with other aneurysms, principally involving the abdominal aorta. At least 40% of popliteal aneurysms are symptomatic on discovery because of thrombosis-in-situ of the popliteal artery or distal emboli to the calf or foot. According to a collective review of the literature that was conducted by Dawson et al. (1249), these complications still occur in 36% of patients whose popliteal aneurysms are merely placed under observation, a figure that is remarkably similar to the late complication rate of 34% that was reported by Gifford and his associates more than 40 years earlier. Furthermore, Dawson et al. also found that the cumulative incidence of ischemic complications was as high as 70% during 5 to 10 years of follow-up for popliteal aneurysms that were evaluated at their own center (1250,1251).

According to data reported by Roggo et al., as many as 50% of previously asymptomatic popliteal aneurysms may be expected to become symptomatic within 2 years after their discovery and 75% within 5 years (1254) (Figure 22). Symptomatic popliteal aneurysms generally exceed 2.0 cm in diameter, often contain a substantial amount of mural thrombus on B-mode ultrasound imaging, and frequently are associated with distal tibioperoneal arterial occlusions that suggest previous emboli (1252,1253,1255). Probably because of prior emboli with thrombosis of downstream outflow vessels, Poirier et al. reported that 56% of patients continued to experience distal ischemia despite surgical repair of symptomatic popliteal aneurysms, and 19% eventually required amputation (1256).

The unfavorable consequences of popliteal aneurysms suggest that even asymptomatic popliteal aneurysms with good distal runoff 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 (1245,1254,1255). Stiegler et al. have reported a series of 46 patients who had 65 popliteal artery aneurysms with a mean diameter of 1.9 cm (range 0.8 to 4.0 cm); the aneurysms were occluded at the time of their discovery in only 8 patients (mean diameter 2.4 cm, range 1.4 to 4.0 cm) (1257). Thirty-six patients with 46 aneurysms were observed over a period of 2.5 years. The total complication rate was 6.5%, with a higher incidence in patients whose aneurysms were larger than 2.0 cm in diameter (14% vs. 3.1%). Complications also appeared to occur more frequently (14% vs. 0%) in the 19 patients who were treated with platelet-inhibitor drugs than in 16 others who received coumarin anticoagulation. The mean increase in diameter during follow-up was 1.5 mm per year for aneurysms larger than 2.0 cm versus 0.7 mm per year for smaller aneurysms. In another regional survey of 19 vascular surgeons who contributed data for 200 popliteal aneurysms in 137 patients during a 4-year period of study, Varga et al. determined that 31% of small, untreated aneurysms eventually required surgical intervention because of the onset of new symptoms or expansion to a diameter that exceeded 2.0 cm while under surveillance (968).

Thrombosis of popliteal arterial aneurysms accounts for approximately 10% of acute arterial occlusions in elderly men. Commonly mistaken for an embolic event, the diagnosis is often made intraoperatively at the time of an attempted embolectomy (518,1258). Severe ischemia usually occurs because thrombosis occurs suddenly in the absence of collateral enhancement and because the popliteal artery is the sole axial artery traversing the knee. Given that half of all popliteal aneurysms are bilateral, the presence of a prominent popliteal pulse in the opposite leg may be a valuable clue to the underlying etiology of the acute ischemia. Once suspected, ultrasound imaging is the most rapid means to confirm the diagnosis. In a series of 33 patients with 54 popliteal artery aneurysms that were followed up over 62 months, thrombosis occurred in 39%, most often in larger aneurysms (967).

5.4.2.2 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. In a series of 13 aneurysms of the superficial femoral artery reported by Jarrett et al., 11 (85%) occurred in men, 9 (69%) were associated with aortic or iliac aneurysms, and 7 (54%) were contiguous with common femoral or popliteal aneurysms (1259). Six patients (46%) presented with distal ischemia and 4 (31%) with a thigh mass, whereas the remaining 3 (23%) were discovered during investigations for other

Table 60. Presentation and Complication Rates for Popliteal Aneurysms

First Author	Reference	Year	No. of Patients and/or Aneurysms	Bilateral Popliteal or Other Aneurysms	Previous Symptoms Before Presentation	Initial Surgical Treatment	Complications With Observation Alone	Related Amputation Rate
Case series								
Gifford	(1248)	1953	69/100 (66 men)	45% bilateral; 25% other	65% (34% ischemic; 12% ruptured	21%	34% (21% ischemic; 7% ruptured)	23% (7% early; 16% late)
Dawson	(1250)	1991	50/71	42% bilateral; 32% other	NA	65%	54%	NA
Carpenter	(967)	1994	33/54	62% bilateral; 61% other	61% (39% ischemic)	83%	NA	11%
Dawson	(1251)	1994	42/42	NA	All asymptomatic	None	60%	7%
Lowell	(1252)	1994	106/161 (103 men)	52% bilateral	42%	31%	22%	7%
Schroder	(1253)	1996	217/349	61% bilateral	45%	63%	47%	NA
Duffy	(1245)	1998	24/40 (23 men)	66% bilateral	58%	75%	None (smaller than 2 cm)	None
Collective reviews								
Dawson	(1249)	1997	1673/2445 (95% men)	50% bilateral; 37% other	67%	NA	36%	NA

NA indicates not available.

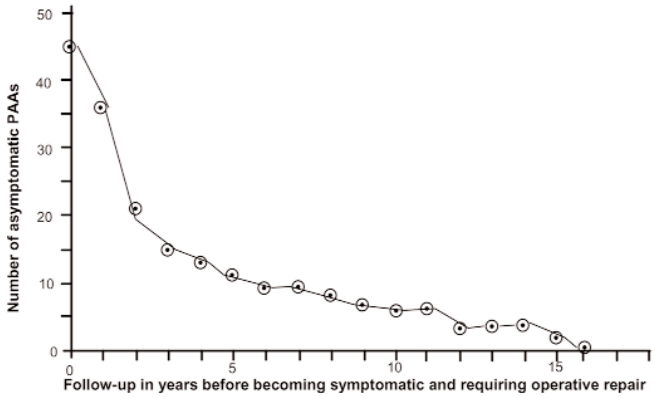

Figure 22. Follow-up evaluation of asymptomatic popliteal artery aneurysm (PAAs). Reprinted from Roggo A, Brunner U, Ottinger LW. The continuing challenge of aneurysms of the popliteal artery. Surg Gynecol Obstet. 1993;177:565-72 (1254).

vascular conditions. None of these aneurysms had ruptured. Aneurysms of the deep femoral artery usually are found in conjunction with an adjacent aneurysm of the common femoral artery, but isolated aneurysms of the deep femoral artery account for just 0.5% of peripheral aneurysms and for only 1% to 2.6% of femoral aneurysms (1260,1261). Twenty percent of patients with deep femoral aneurysms in 1 series had 3 or more peripheral aneurysms. The rate of rupture of deep femoral aneurysms appears to be higher than that of other lower extremity aneurysms, occurring in one third of the cases reported by Cutler and Darling (1260). Other complications are related to expansion, such as femoral nerve compression, venous occlusion with phlegmasia cerulea dolens, and acute leg ischemia secondary to thrombosis or embolization (1260-1264).

5.4.3. 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)***

5.4.3.1. Popliteal Aneurysms

A popliteal mass should be studied by duplex ultrasonography to distinguish an aneurysm from other soft-tissue lesions, such as a synovial (Baker's) cyst, 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. Farina et al. were unable to identify any controlled trials regarding clinical

management in their review of 29 studies comprising 1673 patients with 2445 popliteal arterial aneurysms (1265).

In the setting of acute ischemia related to popliteal artery aneurysm thrombosis or thromboembolism, catheter-directed thrombolytic therapy is useful to re-establish patency of the popliteal and tibial trunks to allow for more effective definitive aneurysm treatment and limb salvage. Largely on the basis 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 (881,882,1251,1252). Failure to attain runoff with catheter-directed thrombolysis suggests that atheroemboli are involved and/or that a fasciotomy should be considered because of high muscular compartment pressures that may be contributing to the occlusion of otherwise normal outflow vessels.

The data illustrated in Figure 23 document the 10-year graft patency, limb salvage, and patient survival rates for a series of popliteal aneurysm repairs described by Dawson et al. (1249). The survival rate was lower than that for the general population because of the medical comorbidities in such patients. Nevertheless, these data indicate that it is possible to achieve limb salvage rates exceeding 90% at 10 years when the operation is done for asymptomatic aneurysms, with graft patency rates that are as high as 80% after operations for symptomatic aneurysms. According to information collected from 14 other reports (1249), the choice of the bypass conduit may influence late results (Table 61). Saphenous vein grafts were associated with superior long-term patency and limb salvage rates compared with either polyester filament or PTFE grafts in 6 of these reports, and in several others, PTFE grafts were approximately twice as likely as polyester filament grafts to remain patent. Furthermore, in the absence of adequate runoff, surgical repair of popliteal artery aneurysms is more likely to be successful if saphenous vein is used as the conduit and fasciotomy is performed.

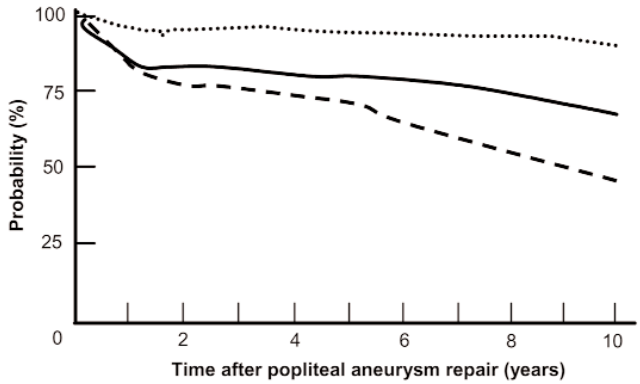

Figure 23. Long-term graft patency, limb salvage, and patient survival after popliteal aneurysm repair. —— indicates long-term graft patency; ·····, limb salvage; and - - - - -, patient survival. From Dawson RB, Sie RB, van Bockel JH. Atherosclerotic popliteal aneurysm. Br J Surg. 1997;84:293-9. ©John Wiley & Sons Limited. Reproduced with permission (1249).

The algorithm presented in Figure 24 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 or computed tomographic 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.

5.4.3.2. 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 computed tomographic scan. The clinical presentation of true femoral artery aneurysms is summarized in Table 62 (1272). Most reports encourage a policy of elective surgical treatment for symptomatic patients if their operative risk is low and if 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 to be associated with a relatively low risk for complications during follow-up periods of 28 to 52 months (1115,1156). 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 (see Figure 20) makes it difficult to determine an

Table 61. Graft Patency and Limb Salvage Rates for Popliteal Aneurysms

First Author	Reference	Follow-Up (y)	No. of Patients	Patency (%)						Limb Salvage (%)			
					Symptoms		Graft Material			Symptoms		Graft Material	
				Total	Asymptomatic	Symptomatic	SV	Others*	Total	Asymptomatic	Symptomatic	SV	Others
Anton	(1266)	5	123	–	82	57	94	43	83	93	82	98	75
		10		56	82	48	94	27	83	93	79	98	66
Carpenter	(967)	5	54	71	–	–	–	–	90	–	–	–	–
Cole	(1267)	3	59	88	94	81	–	–	–	–	–	–	–
Dawson	(1250)	5	46	75	–	—	–	–	–	–	–	–	–
		10		64	–	–	84	41	95	–	–	100	88
Duffy	(1245)	3	30	84	–	–	–	–	96	–	–	–	0
Farina	(1265)	5	50	62	80	65	100	60 A	94	–	–	–	–
		10		62	–	–	–	–	–	–	–	–	–
Inahara	(1268)	10	40	76	–	–	–	–	–	–	–	–	–
Lilly	(1268a)	5	48	74	91	54	–	–	–	–	–	–	–
Reilly	(1269)	5	167	–	–	–	77	30					
Roggo	(1254)	5	252	69	85	61	81	40 B	94	98	92	97	88
		10		–	–	–			87	96	81	94	74
Schellack	(1270)	5	95	75	93	66	92	55	94	100	91	–	–
Schroder	(1253)	4	221	–	89	–	–	–	–	100	–	–	0
Szilagyi	(1255)	5	50	60	—	—	–	–	–	–	–	–	
		10		28	–	—	–	–	–	–	–	–	–
Towne	(1271)	5	115	53	–	–	–	–	–	–	–	–	–

*A indicates 34% polyester fiber and 74 % polytetrafluorethylene (PTFE); B, 33% polyester fiber and 64% PTFE.
SV indicates saphenous vein.
Reprinted from Dawson I, Sie RB, van Bockel JH. Atherosclerotic popliteal aneurysm. Br J Surg 1997;84:293-9 (1249).

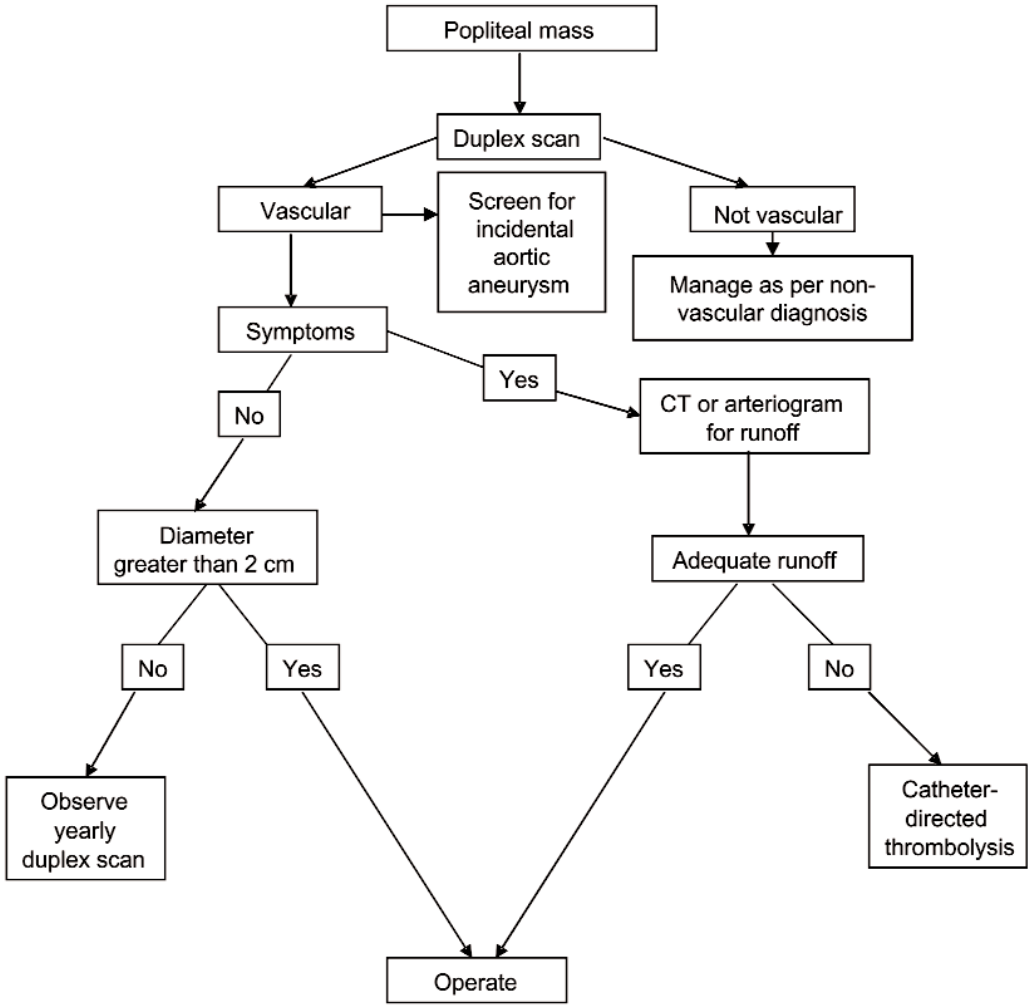

Figure 24. Diagnostic and treatment algorithm for popliteal mass. CT indicates computed tomography.

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 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 regardless of the cause of the femoral artery aneurysm or pseudoaneurysm.

5.4.3.3. 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**

Table 62. Clinical Presentation of Femoral Aneurysms

First Author	Reference	No. of Patients	Aneurysms (n)	Males:Females	Bilateral (%)	AAA/PAA Associated (%)	Asymptomatic (%)	Presentingii Symptoms	Complications at Presentation
Cutler	(1260)	45	63	40:5	47	51/27	29	Local: 29%	Acute thrombosis: 16%; chronic thrombosis: 16%; rupture: 14%
Adiseshiah	(1273)	16	27	15:1	62	25/31	70		Embolization: 4%; thrombosis: 7%; rupture: 15%
Baird	(1274)	30	36	30:0	20	40/17	27	Local: 23%; ischemic: 50%	Acute thrombosis/embolization: 13%; rupture: 0%
Graham	(1235)	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	(1275)	22	31	21:1	41	50/—	64	Local: 5%; ischemic: 35%	

AAA indicates abdominal aortic aneurysm; FAA, femoral artery aneurysm; PAA, popliteal artery aneurysm.
Reprinted from Vascular Surgery (5th ed), Graham L, Femoral and popliteal aneurysms, 1345-56, copyright 2000, with permission from Elsevier (1272).

femoral artery pseudoaneurysms smaller than 2.0 cm in diameter. ***(Level of Evidence: B)***

A pseudoaneurysm is a pulsatile hematoma that communicates with an artery through a defect in the arterial wall. Femoral pseudoaneurysms are well-recognized complications of arterial catheterization, occurring after 0.1% to 0.2% of diagnostic angiograms and after 3.5% to 5.5% of interventional procedures. Puncture-site pseudoaneurysms are most commonly associated with longer procedures, the use of larger-diameter delivery-sheath sizes catheters, systemic anticoagulation, and difficult arterial access. Some studies have suggested that more than 60% of catheter-related femoral pseudoaneurysms are overlooked on the basis of the physical examination alone. Therefore, 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. Collectively, 61% of the small pseudoaneurysms in the 9 series that are summarized in Table 63 resolved within 7 to 52 days of observation, and only 11% ultimately required surgical intervention. Figure 25 illustrates the spontaneous closure rate of selected pseudoaneurysms that were not repaired immediately, 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 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 64 contains information from 17 series of patients who underwent ultrasound-guided compression therapy with a primary success rate of 86% and surgical

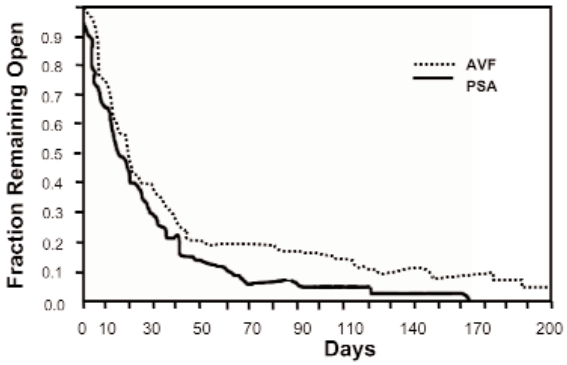

Figure 25. Spontaneous closure rates of selected pseudoaneurysms. AVF indicates arteriovenous fistula; PSA, pseudoaneurysm. Reprinted from J Vasc Surg, 25, Toursarkissian B, Allen BT, Petinec D, et al. Spontaneous closure of selected iatrogenic pseudoaneurysms and arteriovenous fistulae, 803-8, Copyright 1997, with permission from Elsevier (1283).

Table 63. Spontaneous Thrombosis of Femoral Pseudoaneurysms

First Author	Reference	No. of Patients	Spontaneous Closure (n)	Surgery (n)	Comments
Feld	(1276)	17	3	2	
Fellmeth	(1277)	35	4	—	
Johns	(1278)	6	5	2	7 to 42 days to close
Kazmers	(1279)	53	4	3	
Kresowik	(1280)	7	7	—	Less than 28 days to close
Samuels	(1281)	11	11	—	
Schaub	(1282)	54	50	—	Approximately 52 days to close
Toursarkissian	(1283)	147	86%	14%	Approximately 23 days to close
Weatherford	(1284)	27	7	10	Median 40 days to close
Total		357	217	38	
Fractional percentage			61%	11%	

Table 64. Ultrasound-Guided Compression of Femoral Pseudoaneurysms

First Author	Reference	Patients (n)	Closure (n)	Surgery (n)	Comments
Chatterjee	(1285)	38	37	1	FemoStop used
Coghlan	(1286)	10	9	1	
Cox	(1287)	100	94	2	10 recurrences, 1 to 35 days
Dean	(1288)	77	56	14	Size less than 4 cm; twice as successful at closure
Feld	(1276)	15	10	2	
Fellmeth	(1277)	29	27	—	
Hajarizadeh	(1289)	57	54	2	2 recurrences 2 to 10 days
Hertz	(1290)	41	36	3	Large catheter sheath size problematic
Kazmers	(1279)	33	25	3	2 pseudoaneurysm ruptures
Kumins	(1291)	60	52	—	7 recurrences
Langella	(1292)	36	27	—	3 recurrences
Paulson	(1293)	48	37	—	
Perkins	(1294)	13	10	—	
Schaub	(1282)	124	104	5	
Sorrell	(1295)	11	10	1	
Steinkamp	(1296)	98	96	2	
Weatherford	(1284)	11	8	3	

Table 65. Thrombin Injection Closure of Femoral Pseudoaneurysms

First Author	Reference	Patients (n)	Thrombin Dose (U)	Closure (n)	Surgery (n)	Comments
Hughes	(1303)	9	1000 to 2000	8	0	1 recurrence at 4 days
Kang	(1304)	21	500 to 1000	20	1	
La Perna	(1299)	70	1000	66	2	94% overall success rate Success maintained in patients using antithrombotic medications
Liau	(1305)	5	1000	5	0	
Mohler	(1300)	91	500 to 1000	87	0	98% overall success rate Second injection required for 3 patients
Reeder	(1306)	26	50 to 450	25	0	1 recurrence at 4 days
Sacket	(1307)	30	100 to 2000	27	3	
Taylor	(1308)	29	600	27	1	

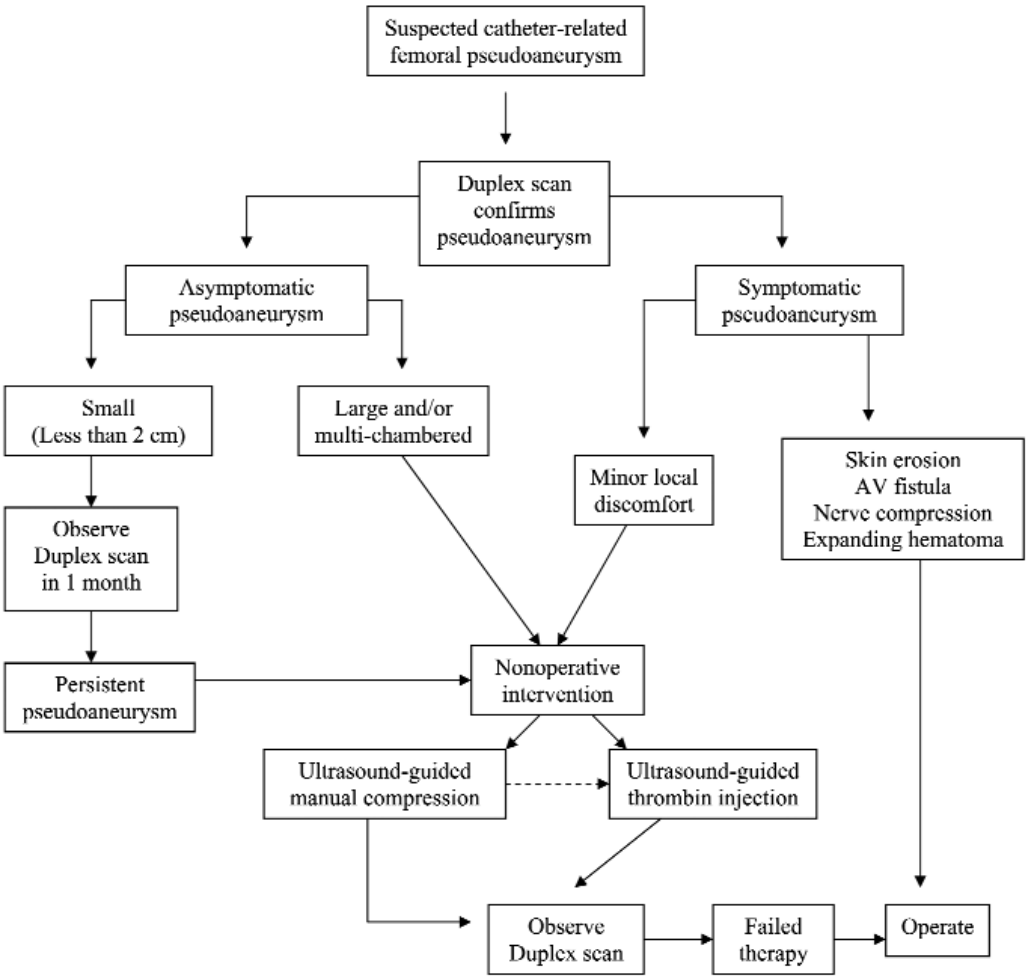

Figure 26. Diagnostic and treatment algorithm for femoral pseudoaneurysm. AV indicates arteriovenous.

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.

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 65 contains data from 7 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 (1297-1299). According to a multicenter registry of patients who have been treated with this technique, thrombin injection ultimately has provided successful treatment for 98% of pseudoaneurysms and appears to represent an improvement over ultrasound-guided compression therapy (1300,1301). One study has been reported in which thrombin injection was compared concurrently with ultrasound-guided compression therapy (1302). Thrombin injection took less time and was associated with lower vascular laboratory costs, but the overall hospital costs were equivalent in both groups of patients.

The algorithm illustrated in Figure 26 presents an approach to the management of catheter-related femoral artery pseudoaneurysms that is consistent with the current literature on this topic.

STAFF

American College of Cardiology

Tom Arend, Interim Chief Staff Officer

Marie Temple, Specialist, Knowledge Development

Joseph Allen, MS, Research Analyst, Knowledge Development

Deborah Steinbach, MA, Managing Editor, Knowledge Development

Peg Christiansen, Librarian, Knowledge Development

American Heart Association

M. Cass Wheeler, Chief Executive Officer

Rose Marie Robertson, MD, FACC, FAHA, Chief Science Officer

Kathryn A. Taubert, PhD, FAHA, Senior Science Advisor

APPENDIX 1. ACC/AHA Writing Committee to Develop Guidelines on Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic)

Committee Member	Research Grant	Speakers Bureau/ Honoraria	Stock Ownership	Consultant	Advisory Board
Dr Curtis W. Bakal	None	None	None	None	Berlex Labs Abbott Labs
Dr Mark A. Creager	Eli Lilly Otsuka Pharmaceuticals Pfizer Vasogen	Bristol Myers Squibb/ Sanofi Partnership Otsuka Pharmaceuticals	Northport Domain	None	Bristol Myers Squibb/ Sanofi Genvec Geozyme Northport Domain Otsuka Pharmaceuticalse Pfizer Vasogen
Dr Jonathan L. Halperin	None	AstraZeneca, LP Bristol Myers Squibb/ Sanofi Partnership	None	AstraZeneca, LP Bayer AG Boehringer Ingelheim Bristol Myers Squibb/ Sanofi Partnership	AstraZeneca, LP
Dr Ziv J. Haskal	Bard/Impra Boston Scientific Cook Cordis Endovascular Genentech IntraTherapeutics W. L. Gore	TransVascular W. L. Gore	None	Bard/Impra Endosurgery Ethicon Omnisonics TransVascular	TransVascular
Dr Norman R. Hertzer	None	None	None	None	None
Dr Loren F. Hiratzka	None	None	None	None	None
Dr Alan T. Hirsch	Alteon AstraZeneca Pharmaceuticals Bristol Myers Squibb/ Sanofi Aventis Partnership Kos Pharmaceuticals Otsuka America Pharmaceuticals	AstraZeneca Pharmaceuticals Bristol Myers Squibb/ Sanofi Aventis Partnership Otsuka America Pharmaceuticals Pfizer	None	Sonosite Vasogen	None
Dr William R.C. Murphy	None	None	None	None	None
Dr Jeffrey W. Olin	Bristol Myers Squibb/ Sanofi Partnership Vasogen	None	None	Aventis Bristol Myers Squibb/ Sanofi Partnership Genzyme Otsuka Vasogen	Abbott Aventis Bristol Myers Squibb/ Sanofi Partnership Genzyme

Continued on Next Page

APPENDIX 1. *Continued*

Committee Member	Research Grant	Speakers Bureau/ Honoraria	Stock Ownership	Consultant	Advisory Board
Dr Jules B. Puschett	None	None	None	None	None
Dr Kenneth A. Rosenfield	Abbott Boston Scientific Cordis Guidant	Eli Lilly	CryoVascular	Abbott Boston Scientific Cordis CryoVascular Guidant	Abbott Boston Scientific Cordis Guidant
Dr David Sacks	None	None	Angiotech	None	None
Dr James C. Stanley	None	None	None	None	None
Dr Lloyd M. Taylor, Jr	None	None	None	None	None
Dr Christopher J. White	None	Eli Lilly	None	None	None
Dr John White	None	None	None	None	None
Dr Rodney A. White	AVE Bard Baxter Cordis J & J EndoLogix EndoSonics Medtronic	Multiple relationships with commercial entities that arise and are met as needed	Several biomedical companies	None	None

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

APPENDIX 2. External Peer Reviewers for the ACC/AHA 2005 Guideline Update for Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic)*

Peer Reviewer Name*	Representation	Research Grant	Speakers Bureau/ Honoraria	Stock Ownership	Consultant/ Advisory Board
Dr Joshua A. Beckman	Content Reviewer – ACC PVD Committee	None	Merck	None	None
Dr James F. Benenati	Official Reviewer – AHA	None	None	None	None
Dr Ralph G. Brindis	Official Reviewer – ACC BOT	None	None	None	None
Dr Alan S. Brown	Official Reviewer – ACC BOG	AstraZeneca Merck Merck Schering Plough Pfizer Smith Kline Beecham	Merck Merck Schering Plough Pfizer	None	AstraZeneca Merck Merck Schering Plough
Rita C. Clark	Organizational Reviewer – SVN	None	None	None	None
Dr John P. Cooke	Content Reviewer – Individual	None	None	None	None
Dr Robert T. Eberhardt	Official Reviewer – AHA	None	None	None	None
Dr Brian S. Funaki	Content Reviewer – AHA Committee on PV Imaging and Intervention	None	None	None	None
Dr Bruce Gray	Organizational Reviewer – SVMB	None	None	None	None
Karen Hayden, MSN	Organizational Reviewer – SVN	None	None	None	None
Dr William R. Hiatt	Organizational Reviewer – TASC	None	BMS/Sanofi Otsuka	None	BMS/Sanofi Signature
Dr David Holmes, Jr	Content Reviewer – ACC BOG	None	None	None	None
Dr Sharon A. Hunt	Organizational Reviewer – ACC/AHA TF on PGL	None	None	None	None
Dr Michael R. Jaff	Organizational Reviewer – SVMB	None	OtsukaBMS/Sanofi	None	Cordis Endovascular
Dr Matthew S. Johnson	Content Reviewer – AHA Committee on PV Imaging and Intervention	Bard Access Systems Boston Scientific	None	None	Boston Scientific
Dr John A. Kaufman	Content Reviewer – AHA Atherosclerosis PVD Steering Committee	None	None	None	None
Dr Morton Kern	Content Reviewer – AHA Diag and Interv Cardiac Cath Cmte	None	None	None	None
Dr Lloyd Klein	Content Reviewer – AHA Diag and Interv Cardiac Cath Cmte	TBD	TBD	TBD	TBD

Continued on Next Page

APPENDIX 2. *Continued*

Peer Reviewer Name*	Representation	Research Grant	Speakers Bureau/ Honoraria	Stock Ownership	Consultant/ Advisory Board
Dr Frank Lederle	Content Reviewer – Individual Review	None	None	None	None
Dr Jonathan Lindner	Official Reviewer – ACCF TF on CECD	None	None	None	None
Dr Mary M. McDermott	Content Reviewer – AHA Athero PVD PVD Steering Committee	None	None	None	None
Dr Alan Matsumoto	Content Reviewer – AHA Committee on PV Imaging and Intervention	None	Genentech	None	Cordis Endovascular Medtronic W. .L. Gore
Dr Roxana Mehran	Content Reviewer – Individual Review	Boston Scientific Cordis Medtronic	The Medicines Company Tyco/Mallinckrodt	None	None
Dr Emile R. Mohler III	Content Reviewer – Individual Review	None	None	None	None
Roberta Oka, RN	Content Reviewer – AHA Atherosclerosis PVD Steering Committee	None	None	None	None
Dr Joseph P. Ornato	Official Reviewer – ACC/AHA TF on PGL, Lead Reviewer	None	None	None	Genentech Meridian Revivant Wyeth
Dr Kenneth Ouriel	Content Reviewer – ACC PVD Committee	TBD	TBD	TBD	TBD
Dr William Pearce	Official Reviewer – AHA	None	None	None	None
Carolyn A. Robinson	Organizational Reviewer – SVN	None	None	None	None
Dr Robert D. Safian	Organizational Reviewer – SCAI	None	None	None	Boston Scientific Cordis/Johnson & Johnson eV3 Medtronic
Dr Sonia I. Skarlatos	Organizational Reviewer – NHLBI	None	None	None	None
Dr Kimberly A. Skelding	Content Reviewer – AHA Diag and Interv Cardiac Cath Cmte	None	None	None	None
Dr Vincenza Snow	Organizational Reviewer – ACP/ASIM	None	None	None	None
Dr Thomas L. Whitsett	Organizational Reviewer – SVMB	None	None	None	None

This table represents the relationships of peer reviewers with industry that were disclosed at the time of peer review of this guideline. It does not necessarily reflect relationships with industry at the time of publication. Participation in the peer review process does not imply endorsement of the document.

*Names are listed in alphabetical order.

ACCF indicates American College of Cardiology Foundation; ACP, American College of Physicians; AHA Diag and Interv Cardiac Cath Cmte, AHA Diagnostic and Interventional Cardiac Catheterization Committee; ASIM, American Society of Internal Medicine; BOG, Board of Governors; BOT, Board of Trustees; NHLBI, National Heart, Lung, and Blood Institute; PV, peripheral vein; PVD, peripheral vascular disease; SCAI, Society for Cardiovascular Angiography and Interventions; SVMB, Society of Vascular Medicine and Biology; SVN, Society for Vascular Nursing; TBD, to be determined; TF on CECD, Task Force on Clinical Expert Consensus Documents; and TF on PGL, Task Force on Practice Guidelines.

APPENDIX 3. ABBREVIATIONS

ABI = ankle-brachial index
ACC = American College of Cardiology
ACE = angiotensin-converting enzyme
AHA = American Heart Association
ARIC = Atherosclerosis Risk in Communities study
bFGF = basic fibroblast growth factor
CI = confidence interval
CLI = critical limb ischemia
COPD = chronic obstructive pulmonary disease
CTA = computed tomographic angiography
DNA = deoxyribonucleic acid
DRASTIC = Dutch Renal Artery Stenosis Intervention Cooperative
EDTA = ethylenediaminetetraacetic acid
ESRD = end-stage renal disease
EUROSTAR = EUROpean collaborators on Stent-graft Techniques for abdominal aortic Aneurysm Repair
FDA = Food and Drug Administration
FMD = fibromuscular dysplasia
HDL = high-density lipoprotein
HMG = hydroxymethyl glutaryl
ICAVL = Intersocietal Commission for Accreditation of Vascular Laboratories
INR = international normalized ratio
LDL = low-density lipoprotein
MI = myocardial infarction
MMP = matrix metalloproteinases
MRA = magnetic resonance angiography
NHDS = National Hospital Discharge Survey
OR = odds ratio
p = statistical significance
PAD = peripheral arterial disease
PARTNERS = PAD Awareness, Risk and Treatment: New Resources for Survival (study)
PGE-1 = prostaglandin E1
phVEGF165 = vascular endothelial growth factor plasma DNA
PTA = percutaneous transluminal angioplasty
PTFE = polytetrafluoroethylene
PVR = pulse volume recording
RAS = renal artery stenosis
RRI = resistive index
ROS = review of symptoms
SVS/ISCVS = Society for Vascular Surgery/ International Society for Cardiac Vascular Surgery
TASC = TransAtlantic Inter-Society Consensus Working Group
TBI = toe-brachial index
3D = 3-dimensional
UK = United Kingdom
US = United States
USPSTF = United States Preventive Services Task Force
VA = Veterans Affairs
VEGF = vascular endothelial growth factor

References

1. Dormandy JA, Rutherford RB. Management of peripheral arterial disease (PAD). TASC Working Group. TransAtlantic Inter-Society Consensus (TASC). J Vasc Surg 2000;31(1 pt 2):S1-S296.
2. Ross R. Cellular and molecular studies of atherogenesis. Atherosclerosis 1997;131(suppl):S3-4.
3. Fowkes FG, Housley E, Riemersma RA, et al. Smoking, lipids, glucose intolerance, and blood pressure as risk factors for peripheral atherosclerosis compared with ischemic heart disease in the Edinburgh Artery Study. Am J Epidemiol 1992;135:331-40.
4. Ridker PM, Cushman M, Stampfer MJ, et al. Plasma concentration of C-reactive protein and risk of developing peripheral vascular disease. Circulation 1998;97:425-8.
5. Taylor LM Jr, DeFrang RD, Harris EJ Jr, et al. The association of elevated plasma homocyst(e)ine with progression of symptomatic peripheral arterial disease. J Vasc Surg 1991;13:128-36.
6. Robinson PN, Booms P. The molecular pathogenesis of the Marfan syndrome. Cell Mol Life Sci 2001;58:1698-707.
7. Pyeritz RE. The Marfan syndrome. Annu Rev Med 2000;51:481-510.
8. Parfitt J, Chalmers RT, Wolfe JH. Visceral aneurysms in Ehlers-Danlos syndrome: case report and review of the literature. J Vasc Surg 2000;31:1248-51.
9. Pope FM, Burrows NP. Ehlers-Danlos syndrome has varied molecular mechanisms. J Med Genet 1997;34:400-10.
10. Bergqvist D. Ehlers-Danlos type IV syndrome: a review from a vascular surgical point of view. Eur J Surg 1996;162:163-70.
11. Begelman SM, Olin JW. Fibromuscular dysplasia. Curr Opin Rheumatol 2000;12:41-7.
12. Luscher TF, Lie JT, Stanson AW, et al. Arterial fibromuscular dysplasia. Mayo Clin Proc 1987;62:931-52.
13. Gonzalez-Gay MA, Garcia-Porrua C. Epidemiology of the vasculitides. Rheum Dis Clin North Am 2001;27:729-49.
14. Johnston SL, Lock RJ, Gompels MM. Takayasu arteritis: a review. J Clin Pathol 2002;55:481-6.
15. Salvarani C, Cantini F, Boiardi L, et al. Polymyalgia rheumatica and giant-cell arteritis. N Engl J Med 2002;347:261-71.
16. Cid MC, Font C, Coll-Vinent B, et al. Large vessel vasculitides. Curr Opin Rheumatol 1998;10:18-28.
17. Langford CA, Sneller MC. New developments in the treatment of Wegener's granulomatosis, polyarteritis nodosa, microscopic polyangiitis, and Churg-Strauss syndrome. Curr Opin Rheumatol 1997;9:26-30.
18. Barron KS. Kawasaki disease: etiology, pathogenesis, and treatment. Cleve Clin J Med 2002;69 suppl 2:SII69-78.
19. Gedalia A. Kawasaki disease: an update. Curr Rheumatol Rep 2002;4:25-9.
20. Newburger JW, Burns JC. Kawasaki disease. Vasc Med 1999;4:187-202.
21. Olin JW. Thromboangiitis obliterans (Buerger's disease). N Engl J Med 2000;343:864-9.
22. Szuba A, Cooke JP. Thromboangiitis obliterans: an update on Buerger's disease. West J Med 1998;168:255-60.
23. Aqel MB, Olin JW. Thromboangiitis obliterans (Buerger's disease). Vasc Med 1997;2:61-6.
24. Lee R. Factor V Leiden: a clinical review. Am J Med Sci 2001;322:88-102.
25. Kottke-Marchant K. Genetic polymorphisms associated with venous and arterial thrombosis: an overview. Arch Pathol Lab Med 2002;126:295-304.
26. Segal JB, McNamara RL, Miller MR, et al. Anticoagulants or antiplatelet therapy for non-rheumatic atrial fibrillation and flutter. Cochrane Database Syst Rev 2001;(1):CD001938.
27. Hirsh J, Anand SS, Halperin JL, et al. Guide to anticoagulant therapy: heparin: a statement for healthcare professionals from the American Heart Association. Circulation 2001;103:2994-3018.
28. Lillicrap D. The genetics of venous and arterial thromboembolism. Curr Atheroscler Rep 2001;3:209-15.
29. Dormandy J, Heeck L, Vig S. Acute limb ischemia. Semin Vasc Surg 1999;12:148-53.
30. Fraenkel L. Raynaud''s phenomenon: epidemiology and risk factors. Curr Rheumatol Rep 2002;4:123-8.
31. Edwards JM, Porter JM. Upper extremity arterial disease: etiologic considerations and differential diagnosis. Semin Vasc Surg 1998;11:60-6.
32. Belch J. Raynaud's phenomenon. Cardiovasc Res 1997;33:25-30.
33. Belch JJ, Ho M. Pharmacotherapy of Raynaud's phenomenon. Drugs 1996;52:682-95.
34. Criqui MH, Fronek A, Klauber MR, et al. The sensitivity, specificity, and predictive value of traditional clinical evaluation of peripheral arterial disease: results from noninvasive testing in a defined population. Circulation 1985;71:516-22.
35. Criqui MH, Denenberg JO, Langer RD, et al. The epidemiology of peripheral arterial disease: importance of identifying the population at risk. Vasc Med 1997;2:221-6.
36. Price JF, Mowbray PI, Lee AJ, et al. Relationship between smoking and cardiovascular risk factors in the development of peripheral arterial disease and coronary artery disease: Edinburgh Artery Study. Eur Heart J 1999;20:344-53.
37. Kannel WB, McGee DL. Update on some epidemiologic features of intermittent claudication: the Framingham Study. J Am Geriatr Soc 1985;33:13-8.
38. Smith GD, Shipley MJ, Rose G. Intermittent claudication, heart disease risk factors, and mortality. The Whitehall Study. Circulation 1990;82:1925-31.
39. Bowlin SJ, Medalie JH, Flocke SA, et al. Epidemiology of intermittent claudication in middle-aged men. Am J Epidemiol 1994;140:418-30.
40. Meijer WT, Hoes AW, Rutgers D, et al. Peripheral arterial disease in the elderly: the Rotterdam Study. Arterioscler Thromb Vasc Biol 1998;18:185-92.
41. Cole CW, Hill GB, Farzad E, et al. Cigarette smoking and peripheral arterial occlusive disease. Surgery 1993;114:753-6; discussion 756-7.
42. Powell JT, Edwards RJ, Worrell PC, et al. Risk factors associated with the development of peripheral arterial disease in smokers: a case-control study. Atherosclerosis 1997;129:41-8.
43. Kannel WB, Shurtleff D. The Framingham Study: cigarettes and the development of intermittent claudication. Geriatrics 1973;28: 61-8.
44. Newman AB, Siscovick DS, Manolio TA, et al. Ankle-arm index as a marker of atherosclerosis in the Cardiovascular Health Study. Cardiovascular Heart Study (CHS) Collaborative Research Group. Circulation 1993;88:837-45.
45. Hiatt WR, Hoag S, Hamman RF. Effect of diagnostic criteria on the prevalence of peripheral arterial disease. The San Luis Valley Diabetes Study. Circulation 1995;91:1472-9.
46. Beks PJ, Mackaay AJ, de Neeling JN, et al. Peripheral arterial disease in relation to glycaemic level in an elderly Caucasian population: the Hoorn study. Diabetologia 1995;38:86-96.
47. Katsilambros NL, Tsapogas PC, Arvanitis MP, et al. Risk factors for lower extremity arterial disease in non–insulin-dependent diabetic persons. Diabet Med 1996;13:243-6.

48. Bowers BL, Valentine RJ, Myers SI, et al. The natural history of patients with claudication with toe pressures of 40 mm Hg or less. J Vasc Surg 1993;18:506-11.
49. McDaniel MD, Cronenwett JL. Basic data related to the natural history of intermittent claudication. Ann Vasc Surg 1989;3:273-7.
50. Dormandy JA, Murray GD. The fate of the claudicant—-a prospective study of 1969 claudicants. Eur J Vasc Surg 1991; 5:131-3.
51. Most RS, Sinnock P. The epidemiology of lower extremity amputations in diabetic individuals. Diabetes Care 1983;6:87-91.
52. Murabito JM, Evans JC, Nieto K, et al. Prevalence and clinical correlates of peripheral arterial disease in the Framingham Offspring Study. Am Heart J 2002;143:961-5.
53. Ingolfsson IO, Sigurdsson G, Sigvaldason H, et al. A marked decline in the prevalence and incidence of intermittent claudication in Icelandic men 1968-1986: a strong relationship to smoking and serum cholesterol—the Reykjavik Study. J Clin Epidemiol 1994;47:1237-43.
54. Murabito JM, D'Agostino RB, Silbershatz H, et al. Intermittent claudication. A risk profile from The Framingham Heart Study. Circulation 1997;96:44-9.
55. Bainton D, Sweetnam P, Baker I, et al. Peripheral vascular disease: consequence for survival and association with risk factors in the Speedwell prospective heart disease study. Br Heart J 1994; 72:128-32.
56. Sanderson KJ, van Rij AM, Wade CR, et al. Lipid peroxidation of circulating low density lipoproteins with age, smoking and in peripheral vascular disease. Atherosclerosis 1995;118:45-51. Erratum in: Atherosclerosis 1996;121:295.
57. Horby J, Grande P, Vestergaard A, et al. High density lipoprotein cholesterol and arteriography in intermittent claudication. Eur J Vasc Surg 1989;3:333-7.
58. Bradby GV, Valente AJ, Walton KW. Serum high-density lipoproteins in peripheral vascular disease. Lancet 1978;2:1271-4.
59. Greenhalgh RM, Rosengarten DS, Mervart I, et al. Serum lipids and lipoproteins in peripheral vascular disease. Lancet 1971; 2:947-50.
60. Harris LM, Armstrong D, Browne R, et al. Premature peripheral vascular disease: clinical profile and abnormal lipid peroxidation. Cardiovasc Surg 1998;6:188-93.
61. Mowat BF, Skinner ER, Wilson HM, et al. Alterations in plasma lipids, lipoproteins and high density lipoprotein subfractions in peripheral arterial disease. Atherosclerosis 1997;131:161-6.
62. Mendelson G, Aronow WS, Ahn C. Prevalence of coronary artery disease, atherothrombotic brain infarction, and peripheral arterial disease: associated risk factors in older Hispanics in an academic hospital-based geriatrics practice. J Am Geriatr Soc 1998;46:481-3.
63. Novo S, Avellone G, Di Garbo V, et al. Prevalence of risk factors in patients with peripheral arterial disease: a clinical and epidemiological evaluation. Int Angiol 1992;11:218-29.
64. Hooi JD, Stoffers HE, Kester AD, et al. Risk factors and cardiovascular diseases associated with asymptomatic peripheral arterial occlusive disease. The Limburg PAOD Study. Peripheral Arterial Occlusive Disease. Scand J Prim Health Care 1998; 16:177-82.
65. Reunanen A, Takkunen H, Aromaa A. Prevalence of intermittent claudication and its effect on mortality. Acta Med Scand 1982; 211:249-56.
66. Boushey CJ, Beresford SA, Omenn GS, et al. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: probable benefits of increasing folic acid intakes. JAMA 1995;274:1049-57.
67. Graham IM, Daly LE, Refsum HM, et al. Plasma homocysteine as a risk factor for vascular disease. The European Concerted Action Project. JAMA 1997;277:1775-81.
68. Robinson K, Arheart K, Refsum H, et al. Low circulating folate and vitamin B6 concentrations: risk factors for stroke, peripheral vascular disease, and coronary artery disease. European COMAC Group. Circulation 1998;97:437-43. Erratum in: Circulation 1999;99:983.
69. Hoogeveen EK, Kostense PJ, Beks PJ, et al. Hyperhomocysteinemia is associated with an increased risk of cardiovascular disease, especially in non–insulin-dependent diabetes mellitus: a population-based study. Arterioscler Thromb Vasc Biol 1998;18:133-8.
70. Aronow WS, Ahn C. Association between plasma homocysteine and peripheral arterial disease in older persons. Coron Artery Dis 1998;9:49-50.
71. Currie IC, Wilson YG, Scott J, et al. Homocysteine: an independent risk factor for the failure of vascular intervention. Br J Surg 1996;83:1238-41.
72. Molgaard J, Malinow MR, Lassvik C, et al. Hyperhomocyst(e)inaemia: an independent risk factor for intermittent claudication. J Intern Med 1992;231:273-9.
73. Taylor LM Jr, Moneta GL, Sexton GJ, et al. Prospective blinded study of the relationship between plasma homocysteine and progression of symptomatic peripheral arterial disease. J Vasc Surg 1999;29:8-19; discussion 19-21.
74. Pradhan AD, Manson JE, Rossouw JE, et al. Inflammatory biomarkers, hormone replacement therapy, and incident coronary heart disease: prospective analysis from the Women's Health Initiative observational study. JAMA 2002;288:980-7.
75. Burke GL, Evans GW, Riley WA, et al. Arterial wall thickness is associated with prevalent cardiovascular disease in middle-aged adults. The Atherosclerosis Risk in Communities (ARIC) Study. Stroke 1995;26:386-91.
76. Kannel WB, Skinner JJ Jr, Schwartz MJ, et al. Intermittent claudication: incidence in the Framingham Study. Circulation 1970;41:875-83.
77. Criqui MH, Fronek A, Barrett-Connor E, et al. The prevalence of peripheral arterial disease in a defined population. Circulation 1985;71:510-5.
78. Kannel WB. The demographics of claudication and the aging of the American population. Vasc Med 1996;1:60-4.
79. Hiatt WR, Marshall JA, Baxter J, et al. Diagnostic methods for peripheral arterial disease in the San Luis Valley Diabetes Study. J Clin Epidemiol 1990;43:597-606.
80. Fowkes FG, Housley E, Cawood EH, et al. Edinburgh Artery Study: prevalence of asymptomatic and symptomatic peripheral arterial disease in the general population. Int J Epidemiol 1991;20:384-92.
81. Aronow WS. Prevalence of atherothrombotic brain infarction, coronary artery disease and peripheral arterial disease in elderly blacks, Hispanics and whites. Am J Cardiol 1992;70:1212-3.
82. Aronow WS, Ahn C. Prevalence of coexistence of coronary artery disease, peripheral arterial disease, and atherothrombotic brain infarction in men and women > or = 62 years of age. Am J Cardiol 1994;74:64-5.
83. Cofan F, Nunez I, Gilabert R, et al. Increased prevalence of carotid and femoral atherosclerosis in renal transplant recipients. Transplant Proc 2001;33:1254-6.
84. Erdoes LS, Hunter GC, Venerus BJ, et al. Prospective evaluation of peripheral vascular disease in heart transplant recipients. J Vasc Surg 1995;22:434-40; discussion 440-2.

85. Hirsch AT, Halverson SL, Treat-Jacobson D, et al. The Minnesota Regional Peripheral Arterial Disease Screening Program: toward a definition of community standards of care. Vasc Med 2001;6:87-96.
86. Hirsch AT, Criqui MH, Treat-Jacobson D, et al. Peripheral arterial disease detection, awareness, and treatment in primary care. JAMA 2001;286:1317-24.
87. Coni N, Tennison B, Troup M. Prevalence of lower extremity arterial disease among elderly people in the community. Br J Gen Pract 1992;42:149-52.
88. Gallotta G, Iazzetta N, Milan G, et al. Prevalence of peripheral arterial disease in an elderly rural population of southern Italy. Gerontology 1997;43:289-95.
89. Cheng SW, Ting AC, Lau H, et al. Epidemiology of atherosclerotic peripheral arterial occlusive disease in Hong Kong. World J Surg 1999;23:202-6.
90. Binaghi F, Fronteddu PF, Cannas F, et al. Prevalence of peripheral arterial occlusive disease and associated risk factors in a sample of southern Sardinian population. Int Angiol 1994;13:233-45.
91. Al Zahrani HA, Al Bar HM, Bahnassi A, et al. The distribution of peripheral arterial disease in a defined population of elderly high-risk Saudi patients. Int Angiol 1997;16:123-8.
92. Ness J, Aronow WS. Prevalence of coexistence of coronary artery disease, ischemic stroke, and peripheral arterial disease in older persons, mean age 80 years, in an academic hospital-based geriatrics practice. J Am Geriatr Soc 1999;47:1255-6.
93. Weitz JI, Byrne J, Clagett GP, et al. Diagnosis and treatment of chronic arterial insufficiency of the lower extremities: a critical review. Circulation 1996;94:3026-49. Erratum in: Circulation 2000;102:1074.
94. Stoffers HE, Rinkens PE, Kester AD, et al. The prevalence of asymptomatic and unrecognized peripheral arterial occlusive disease. Int J Epidemiol 1996;25:282-90.
95. Dormandy J, Mahir M, Ascady G, et al. Fate of the patient with chronic leg ischaemia: a review article. J Cardiovasc Surg (Torino) 1989;30:50-7.
96. Golomb B, Criqui MH, Budens W. Epidemiology. In: Creager MA, ed. Management of Peripheral Arterial Disease. London, UK: ReMEDICA Pub; 2000:1-18.
97. Valentine RJ, Grayburn PA, Eichhorn EJ, et al. Coronary artery disease is highly prevalent among patients with premature peripheral vascular disease. J Vasc Surg 1994;19:668-74.
98. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. Lancet 1996;348:1329-39.
99. Klop RB, Eikelboom BC, Taks AC. Screening of the internal carotid arteries in patients with peripheral vascular disease by colour-flow duplex scanning. Eur J Vasc Surg 1991;5:41-5.
100. Alexandrova NA, Gibson WC, Norris JW, et al. Carotid artery stenosis in peripheral vascular disease. J Vasc Surg 1996;23:645-9.
101. Cheng SW, Wu LL, Ting AC, et al. Screening for asymptomatic carotid stenosis in patients with peripheral vascular disease: a prospective study and risk factor analysis. Cardiovasc Surg 1999;7:303-9.
102. Long TH, Criqui MH, Vasilevskis EE, et al. The correlation between the severity of peripheral arterial disease and carotid occlusive disease. Vasc Med 1999;4:135-42.
103. Leng GC, Lee AJ, Fowkes FG, et al. Incidence, natural history and cardiovascular events in symptomatic and asymptomatic peripheral arterial disease in the general population. Int J Epidemiol 1996;25:1172-81.
104. Kornitzer M, Dramaix M, Sobolski J, et al. Ankle/arm pressure index in asymptomatic middle-aged males: an independent predictor of ten-year coronary heart disease mortality. Angiology 1995;46:211-9.
105. Newman AB, Sutton-Tyrrell K, Vogt MT, et al. Morbidity and mortality in hypertensive adults with a low ankle/arm blood pressure index. JAMA 1993;270:487-9.
106. Criqui MH, Langer RD, Fronek A, et al. Mortality over a period of 10 years in patients with peripheral arterial disease. N Engl J Med 1992;326:381-6.
107. Vogt MT, Cauley JA, Newman AB, et al. Decreased ankle/arm blood pressure index and mortality in elderly women. JAMA 1993;270:465-9.
108. Zheng ZJ, Sharrett AR, Chambless LE, et al. Associations of ankle-brachial index with clinical coronary heart disease, stroke and preclinical carotid and popliteal atherosclerosis: the Atherosclerosis Risk in Communities (ARIC) Study. Atherosclerosis 1997;131:115-25.
109. McKenna M, Wolfson S, Kuller L. The ratio of ankle and arm arterial pressure as an independent predictor of mortality. Atherosclerosis 1991;87:119-28.
110. McDermott MM, Feinglass J, Slavensky R, et al. The ankle-brachial index as a predictor of survival in patients with peripheral vascular disease. J Gen Intern Med 1994;9:445-9.
111. Howell MA, Colgan MP, Seeger RW, et al. Relationship of severity of lower limb peripheral vascular disease to mortality and morbidity: a six-year follow-up study. J Vasc Surg 1989;9:691-6; discussion 696-7.
112. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet 2002;360:7-22. Summary for patients in Curr Cardiol Rep 2002;4:486-7.
113. Yusuf S, Dagenais G, Pogue J, et al. Vitamin E supplementation and cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. N Engl J Med 2000;342:154-60.
114. Luther M. The influence of arterial reconstructive surgery on the outcome of critical leg ischaemia. Eur J Vasc Surg 1994;8:682-9.
115. Ebskov B. Relative mortality and long term survival for the non-diabetic lower limb amputee with vascular insufficiency. Prosthet Orthot Int 1999;23:209-16.
116. Kazmers A, Perkins AJ, Jacobs LA. Major lower extremity amputation in Veterans Affairs medical centers. Ann Vasc Surg 2000;14:216-22.
117. Dormandy J, Heeck L, Vig S. The fate of patients with critical leg ischemia. Semin Vasc Surg 1999;12:142-7.
118. Muluk SC, Muluk VS, Kelley ME, et al. Outcome events in patients with claudication: a 15-year study in 2777 patients. J Vasc Surg 2001;33:251-7; discussion 257-8.
119. Rose GA. The diagnosis of ischaemic heart pain and intermittent claudication in field surveys. Bull World Health Organ 1962; 27:645-58.
120. Fowkes FG. The measurement of atherosclerotic peripheral arterial disease in epidemiological surveys. Int J Epidemiol 1988;17:248-54.
121. Leng GC, Fowkes FG. The Edinburgh Claudication Questionnaire: an improved version of the WHO/Rose Questionnaire for use in epidemiological surveys. J Clin Epidemiol 1992;45:1101-9.
122. Criqui MH, Denenberg JO, Bird CE, et al. The correlation between symptoms and non-invasive test results in patients referred for peripheral arterial disease testing. Vasc Med

1996;1:65-71.
123. McDermott MM, Ferrucci L, Simonsick EM, et al. The ankle brachial index and change in lower extremity functioning over time: the Women's Health and Aging Study. J Am Geriatr Soc 2002;50:238-46.
124. McDermott MM, Greenland P, Liu K, et al. Leg symptoms in peripheral arterial disease: associated clinical characteristics and functional impairment. JAMA 2001;286:1599-606.
125. Newman AB, Naydeck BL, Sutton-Tyrrell K, et al. The role of comorbidity in the assessment of intermittent claudication in older adults. J Clin Epidemiol 2001;54:294-300.
126. Hooi JD, Kester AD, Stoffers HE, et al. Incidence of and risk factors for asymptomatic peripheral arterial occlusive disease: a longitudinal study. Am J Epidemiol 2001;153:666-72.
127. Criqui MH, Denenberg JO. The generalized nature of atherosclerosis: how peripheral arterial disease may predict adverse events from coronary artery disease. Vasc Med 1998;3:241-5.
128. Simons PC, Algra A, Eikelboom BC, et al. Carotid artery stenosis in patients with peripheral arterial disease: the SMART study. SMART study group. J Vasc Surg 1999;30:519-25.
129. House AK, Bell R, House J, et al. Asymptomatic carotid artery stenosis associated with peripheral vascular disease: a prospective study. Cardiovasc Surg 1999;7:44-9.
129a. Grundy SM, Cleeman JI, Bairey Merz CN, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. Circulation 2004;110:227-39.
130. Joint National Committee on Prevention, Detection Evaluation and Treatment of High Blood Pressure. The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Bethesda, Md: US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Heart, Lung and Blood Institute; 1997. Publication No. 98-4080.
131. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Bethesda, Md: National Cholesterol Education Program, National Heart, Lung, and Blood Institute, National Institutes of Health; 2002. Publication No. 02-5215. Available at: http://www.nhlbi.nih.gov/guidelines/cholesterol/. Accessed July 16, 2005.
132. Stewart KJ, Hiatt WR, Regensteiner JG, et al. Exercise training for claudication. N Engl J Med 2002;347:1941-51.
133. Feinglass J, McCarthy WJ, Slavensky R, et al. Effect of lower extremity blood pressure on physical functioning in patients who have intermittent claudication. The Chicago Claudication Outcomes Research Group. J Vasc Surg 1996;24:503-11; discussion 511-2.
134. Breek JC, Hamming JF, De Vries J, et al. The impact of walking impairment, cardiovascular risk factors, and comorbidity on quality of life in patients with intermittent claudication. J Vasc Surg 2002;36:94-9.
135. Aquino R, Johnnides C, Makaroun M, et al. Natural history of claudication: long-term serial follow-up study of 1244 claudicants. J Vasc Surg 2001;34:962-70.
136. Mukherjee D, Lingam P, Chetcuti S, et al. Missed opportunities to treat atherosclerosis in patients undergoing peripheral vascular interventions: insights from the University of Michigan Peripheral Vascular Disease Quality Improvement Initiative (PVD-QI2). Circulation 2002;106:1909-12.
137. Grundy SM, Pasternak R, Greenland P, et al. AHA/ACC scientific statement: Assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. J Am Coll Cardiol 1999; 34:1348-59.
138. Chan AW, Bhatt DL, Chew DP, et al. Early and sustained survival benefit associated with statin therapy at the time of percutaneous coronary intervention. Circulation 2002;105:691-6.
139. McGee SR, Boyko EJ. Physical examination and chronic lower-extremity ischemia: a critical review. Arch Intern Med 1998; 158:1357-64.
140. Second European Consensus Document on chronic critical leg ischemia. Circulation 1991;84(4 suppl):IV1-26.
141. Vale PR, Isner JM, Rosenfield K. Therapeutic angiogenesis in critical limb and myocardial ischemia. J Interv Cardiol 2001; 14:511-28.
142. Boersma E, Poldermans D, Bax JJ, et al. Predictors of cardiac events after major vascular surgery: role of clinical characteristics, dobutamine echocardiography, and beta-blocker therapy. JAMA 2001;285:1865-73.
143. Mercer KG, Berridge DC. Saddle embolus—the need for intensive investigation and critical evaluation: a case report. Vasc Surg 2001;35:63-5.
144. Ha JW, Chung N, Chang BC, et al. Aortic saddle embolism. Clin Cardiol 1999;22:229-30.
145. Green RM, Ouriel K, Ricotta JJ, et al. Revision of failed infrainguinal bypass graft: principles of management. Surgery 1986; 100:646-54.
146. Bartlett ST, Olinde AJ, Flinn WR, et al. The reoperative potential of infrainguinal bypass: long-term limb and patient survival. J Vasc Surg 1987;5:170-9.
147. Belkin M, Donaldson MC, Whittemore AD, et al. Observations on the use of thrombolytic agents for thrombotic occlusion of infrainguinal vein grafts. J Vasc Surg 1990;11:289-94; discussion 295-6.
148. Kinney EV, Bandyk DF, Mewissen MW, et al. Monitoring functional patency of percutaneous transluminal angioplasty. Arch Surg 1991;126:743-7.
149. Schmidtke I, Roth FJ. Repeated percutaneous transluminal catheter-treatment: primary results. Int Angiol 1985;4:87-91.
150. Brewster DC, LaSalle AJ, Robison JG, et al. Femoropopliteal graft failures: clinical consequences and success of secondary reconstructions. Arch Surg 1983;118:1043-7.
151. Moody P, de Cossart LM, Douglas HM, et al. Asymptomatic strictures in femoro-popliteal vein grafts. Eur J Vasc Surg 1989;3: 389-92.
152. Decrinis M, Doder S, Stark G, et al. A prospective evaluation of sensitivity and specificity of the ankle/brachial index in the follow-up of superficial femoral artery occlusions treated by angioplasty. Clin Investig 1994;72:592-7.
153. Sanchez LA, Suggs WD, Veith FJ, et al. Is surveillance to detect failing polytetrafluoroethylene bypasses worthwhile? Twelve-year experience with ninety-one grafts. J Vasc Surg 1993;18:981-9; discussion 989-90.
154. Buth J, Disselhoff B, Sommeling C, et al. Color-flow duplex criteria for grading stenosis in infrainguinal vein grafts. J Vasc Surg 1991;14:716-26; discussion 726-8.
155. Idu MM, Blankenstein JD, de Gier P, et al. Impact of a color-flow duplex surveillance program on infrainguinal vein graft patency: a five-year experience. J Vasc Surg 1993;17:42-52; discussion 52-3.

155a. Hirsch AT. Recognition and management of peripheral arterial disease. In: Eugene Braunwald E, Goldman L, eds. Primary Cardiology. 2nd ed. Saunders, 2003:659-71.
156. Lijmer JG, Hunink MG, van den Dungen JJ, et al. ROC analysis of noninvasive tests for peripheral arterial disease. Ultrasound Med Biol 1996;22:391-8.
157. Feigelson HS, Criqui MH, Fronek A, et al. Screening for peripheral arterial disease: the sensitivity, specificity, and predictive value of noninvasive tests in a defined population. Am J Epidemiol 1994;140:526-34.
158. Nassoura ZE, Ivatury RR, Simon RJ, et al. A reassessment of Doppler pressure indices in the detection of arterial lesions in proximity penetrating injuries of extremities: a prospective study. Am J Emerg Med 1996;14:151-6.
158a. Hiatt WR. Medical treatment of peripheral arterial disease and claudication. N Engl J Med 2001;344:1608-21.
159. Baker JD, Dix DE. Variability of Doppler ankle pressures with arterial occlusive disease: an evaluation of ankle index and brachial-ankle pressure gradient. Surgery 1981;89:134-7.
160. Carter SA. Clinical measurement of systolic pressures in limbs with arterial occlusive disease. JAMA 1969;207(10):1869-74.
161. Strandness DE Jr, Dalman RL, Panian S, et al. Effect of cilostazol in patients with intermittent claudication: a randomized, double-blind, placebo-controlled study. Vasc Endovascular Surg 2002; 36:83-91.
162. Yao ST. Haemodynamic studies in peripheral arterial disease. Br J Surg 1970;57:761-6.
163. Ouriel K, Zarins CK. Doppler ankle pressure: an evaluation of three methods of expression. Arch Surg 1982;117:1297-1300.
164. Jelnes R, Gaardsting O, Hougaard Jensen K, et al. Fate in intermittent claudication: outcome and risk factors. Br Med J (Clin Res Ed) 1986;293:1137-40.
165. McLafferty RB, Moneta GL, Taylor LM Jr, et al. Ability of ankle-brachial index to detect lower-extremity atherosclerotic disease progression. Arch Surg 1997;132:836-40; discussion 840-1.
166. Resnick HE, Lindsay RS, McDermott MM, et al. Relationship of high and low ankle brachial index to all-cause and cardiovascular disease mortality: the Strong Heart Study. Circulation 2004; 109:733-9.
167. Sikkink CJ, van Asten WN, van't Hof MA, et al. Decreased ankle/brachial indices in relation to morbidity and mortality in patients with peripheral arterial disease. Vasc Med 1997;2:169-73.
168. Mohler ER 3rd, Treat-Jacobson D, Reilly MP, et al. Utility and barriers to performance of the ankle-brachial index in primary care practice. Vasc Med 2004;9:253-60.
169. Orchard TJ, Strandness DE Jr. Assessment of peripheral vascular disease in diabetes: report and recommendations of an international workshop sponsored by the American Diabetes Association and the American Heart Association September 18-20, 1992 New Orleans, Louisiana. Circulation 1993;88:819-28.
170. American Diabetes Association. Peripheral arterial disease in people with diabetes. Diabetes Care 2003;26:3333-41.
171. American Medical Association. Current Procedural Terminology (CPT). Chicago, Ill: American Medical Association; 2001.
172. Heintz SE, Bone GE, Slaymaker EE, et al. Value of arterial pressure measurements in the proximal and distal part of the thigh in arterial occlusive disease. Surg Gynecol Obstet 1978;146:337-43.
173. Rutherford RB, Lowenstein DH, Klein MF. Combining segmental systolic pressures and plethysmography to diagnose arterial occlusive disease of the legs. Am J Surg 1979;138:211-8.
174. Carter SA. Indirect systolic pressures and pulse waves in arterial occlusive diseases of the lower extremities. Circulation 1968; 37:624-37.
175. Carter SA. Clinical measurement of systolic pressures in limbs with arterial occlusive disease. JAMA 1969;207:1869-74.
176. Carter SA, Tate RB. Value of toe pulse waves in addition to systolic pressures in the assessment of the severity of peripheral arterial disease and critical limb ischemia. J Vasc Surg 1996;24:258-65.
177. Carter SA, Tate RB. The value of toe pulse waves in determination of risks for limb amputation and death in patients with peripheral arterial disease and skin ulcers or gangrene. J Vasc Surg 2001;33:708-14.
178. Brooks B, Dean R, Patel S, et al. TBI or not TBI: that is the question. Is it better to measure toe pressure than ankle pressure in diabetic patients? Diabet Med 2001;18:528-32.
179. Ramsey DE, Manke DA, Sumner DS. Toe blood pressure: a valuable adjunct to ankle pressure measurement for assessing peripheral arterial disease. J Cardiovasc Surg (Torino) 1983;24:43-8.
180. Raines JK. The pulse volume recorder in peripheral arterial disease. In: Bernstein EF, ed. Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, Mo: Mosby; 1985:513-44.
181. Jorgensen JJ, Stranden E, Gjolberg T. Measurements of common femoral artery flow velocity in the evaluation of aortoiliac atherosclerosis: comparisons between pulsatility index, pressures measurements and pulse-volume recordings. Acta Chir Scand 1988;154:261-6.
182. Symes JF, Graham AM, Mousseau M. Doppler waveform analysis versus segmental pressure and pulse-volume recording: assessment of occlusive disease in the lower extremity. Can J Surg 1984;27:345-7.
183. Clifford PC, Morgan AP, Thomas WE, et al. Monitoring arterial surgery: a comparison of pulse volume recording and electromagnetic flowmetering in aortofemoral reconstruction. J Cardiovasc Surg (Torino) 1986;27:262-7.
184. Kaufman JL, Fitzgerald KM, Shah DM, et al. The fate of extremities with flat lower calf pulse volume recordings. J Cardiovasc Surg (Torino) 1989;30:216-9.
185. Gale SS, Scissons RP, Salles-Cunha SX, et al. Lower extremity arterial evaluation: are segmental arterial blood pressures worthwhile? J Vasc Surg 1998;27:831-8; discussion 838-9.
186. Gosling RG, Dunbar G, King DH, et al. The quantitative analysis of occlusive peripheral arterial disease by a non-intrusive ultrasonic technique. Angiology 1971;22:52-5.
187. Johnston KW, Taraschuk I. Validation of the role of pulsatility index in quantitation of the severity of peripheral arterial occlusive disease. Am J Surg 1976;131:295-7.
188. Thiele BL, Hutchinson KJ, Greene FM, et al. Pulsed Doppler waveform patters produced by smooth stenosis in the dog thoracic aorta. In: Taylor DM, Stevens AL, eds. Blood Flow, Theory and Practice. San Diego, Calif: Academic Press; 1983:85-104.
189. Bascom PA, Johnston KW, Cobbold RS, et al. Defining the limitations of measurements from Doppler spectral recordings. J Vasc Surg 1996;24:34-44; discussion 44-5.
190. Thiele BL, Bandyk DF, Zierler RE, et al. A systematic approach to the assessment of aortoiliac disease. Arch Surg 1983;118:477-81.
191. Johnston KW, Kassam M, Cobbold RS. Relationship between Doppler pulsatility index and direct femoral pressure measurements in the diagnosis of aortoiliac occlusive disease. Ultrasound Med Biol 1983;9:271-81.
192. Gardner AW, Skinner JS, Cantwell BW, et al. Progressive vs single-stage treadmill tests for evaluation of claudication. Med Sci Sports Exerc 1991;23:402-8.
193. Hiatt WR, Nawaz D, Regensteiner JG, et al. The evaluation of exercise performance in patients with peripheral vascular disease.

J Cardiopulm Rehabil 1988;8:525-32.
194. Nagle FJ, Balke B, Naughton JP. Gradational step tests for assessing work capacity. J Appl Physiol 1965;20:745-8.
195. Sumner DS, Strandness DE Jr. The relationship between calf blood flow and ankle blood pressure in patients with intermittent claudication. Surgery 1969;65:763-71.
196. Raines JK, Darling RC, Buth J, et al. Vascular laboratory criteria for the management of peripheral vascular disease of the lower extremities. Surgery 1976;79:21-9.
197. McPhail IR, Spittell PC, Weston SA, et al. Intermittent claudication: an objective office-based assessment. J Am Coll Cardiol 2001;37:1381-5.
198. Greig C, Butler F, Skelton D, et al. Treadmill walking in old age may not reproduce the real life situation. J Am Geriatr Soc 1993;41:15-8.
199. Gardner AW, Katzel LI, Sorkin JD, et al. Exercise rehabilitation improves functional outcomes and peripheral circulation in patients with intermittent claudication: a randomized controlled trial. J Am Geriatr Soc 2001;49:755-62.
200. Simonsick EM, Gardner AW, Poehlman ET. Assessment of physical function and exercise tolerance in older adults: reproducibility and comparability of five measures. Aging (Milano) 2000;12:274-80.
201. Moneta GL, Yeager RA, Lee RW, et al. Noninvasive localization of arterial occlusive disease: a comparison of segmental Doppler pressures and arterial duplex mapping. J Vasc Surg 1993;17:578-82.
202. Pinto F, Lencioni R, Napoli V, et al. Peripheral ischemic occlusive arterial disease: comparison of color Doppler sonography and angiography. J Ultrasound Med 1996;15:697-704; quiz 705-6.
203. Sacks D, Robinson ML, Marinelli DL, et al. Peripheral arterial Doppler ultrasonography: diagnostic criteria. J Ultrasound Med 1992;11:95-103.
204. de Smet AA, Ermers EJ, Kitslaar PJ. Duplex velocity characteristics of aortoiliac stenoses. J Vasc Surg 1996;23:628-36.
205. Fletcher JP, Kershaw LZ, Chan A, et al. Noninvasive imaging of the superficial femoral artery using ultrasound Duplex scanning. J Cardiovasc Surg (Torino) 1990;31:364-7.
206. Ranke C, Creutzig A, Alexander K. Duplex scanning of the peripheral arteries: correlation of the peak velocity ratio with angiographic diameter reduction. Ultrasound Med Biol 1992; 18:433-40.
207. Whelan JF, Barry MH, Moir JD. Color flow Doppler ultrasonography: comparison with peripheral arteriography for the investigation of peripheral vascular disease. J Clin Ultrasound 1992;20:369-74.
208. Davies AH, Willcox JH, Magee TR, et al. Colour duplex in assessing the infrainguinal arteries in patients with claudication. Cardiovasc Surg 1995;3:211-2.
209. Currie IC, Jones AJ, Wakeley CJ, et al. Non-invasive aortoiliac assessment. Eur J Vasc Endovasc Surg 1995;9:24-8.
210. van der Heijden FH, Legemate DA, van Leeuwen MS, et al. Value of duplex scanning in the selection of patients for percutaneous transluminal angioplasty. Eur J Vasc Surg 1993;7:71-6.
211. de Vries SO, Hunink MG, Polak JF. Summary receiver operating characteristic curves as a technique for meta-analysis of the diagnostic performance of duplex ultrasonography in peripheral arterial disease. Acad Radiol 1996;3:361-9.
212. Allard L, Cloutier G, Durand LG, et al. Limitations of ultrasonic duplex scanning for diagnosing lower limb arterial stenoses in the presence of adjacent segment disease. J Vasc Surg 1994;19:650-7.
213. Edwards JM, Coldwell DM, Goldman ML, et al. The role of duplex scanning in the selection of patients for transluminal angioplasty. J Vasc Surg 1991;13:69-74.
214. Proia RR, Walsh DB, Nelson PR, et al. Early results of infragenicular revascularization based solely on duplex arteriography. J Vasc Surg 2001;33:1165-70.
215. Ligush J Jr, Reavis SW, Preisser JS, et al. Duplex ultrasound scanning defines operative strategies for patients with limb-threatening ischemia. J Vasc Surg 1998;28:482-90; discussion 490-1.
216. Ascher E, Mazzariol F, Hingorani A, et al. The use of duplex ultrasound arterial mapping as an alternative to conventional arteriography for primary and secondary infrapopliteal bypasses. Surg Gynecol Obstet 1999;178:162-5.
217. Wain RA, Berdejo GL, Delvalle WN, et al. Can duplex scan arterial mapping replace contrast arteriography as the test of choice before infrainguinal revascularization? J Vasc Surg 1999;29:100-7; discussion 107-9.
218. Larch E, Minar E, Ahmadi R, et al. Value of color duplex sonography for evaluation of tibioperoneal arteries in patients with femoropopliteal obstruction: a prospective comparison with anterograde intraarterial digital subtraction angiography. J Vasc Surg 1997;25:629-36.
219. Mattos MA, van Bemmelen PS, Hodgson KJ, et al. Does correction of stenoses identified with color duplex scanning improve infrainguinal graft patency? J Vasc Surg 1993;17:54-64; discussion 64-6.
220. Mills JL, Harris EJ, Taylor LM Jr, et al. The importance of routine surveillance of distal bypass grafts with duplex scanning: a study of 379 reversed vein grafts. J Vasc Surg 1990;12:379-86; discussion 387-9.
221. Laborde AL, Synn AY, Worsey MJ, et al. A prospective comparison of ankle/brachial indices and color duplex imaging in surveillance of the in situ saphenous vein bypass. J Cardiovasc Surg (Torino) 1992;33:420-5.
222. Taylor PR, Tyrrell MR, Crofton M, et al. Colour flow imaging in the detection of femoro-distal graft and native artery stenosis: improved criteria. Eur J Vasc Surg 1992;6:232-6.
223. Bandyk DF, Schmitt DD, Seabrook GR, et al. Monitoring functional patency of in situ saphenous vein bypasses: the impact of a surveillance protocol and elective revision. J Vasc Surg 1989;9:286-96.
224. Golledge J, Beattie DK, Greenhalgh RM, et al. Have the results of infrainguinal bypass improved with the widespread utilisation of postoperative surveillance? Eur J Vasc Endovasc Surg 1996;11:388-92.
225. Lundell A, Lindblad B, Bergqvist D, et al. Femoropopliteal-crural graft patency is improved by an intensive surveillance program: a prospective randomized study. J Vasc Surg 1995;21:26-33; discussion 33-4.
226. Ihlberg L, Luther M, Tierala E, et al. The utility of duplex scanning in infrainguinal vein graft surveillance: results from a randomised controlled study. Eur J Vasc Endovasc Surg 1998;16:19-27.
227. Lalak NJ, Hanel KC, Hunt J, et al. Duplex scan surveillance of infrainguinal prosthetic bypass grafts. J Vasc Surg 1994;20:637-41.
228. Dunlop P, Sayers RD, Naylor AR, et al. The effect of a surveillance programme on the patency of synthetic infrainguinal bypass grafts. Eur J Vasc Endovasc Surg 1996;11:441-5.
229. Calligaro KD, Musser DJ, Chen AY, et al. Duplex ultrasonography to diagnose failing arterial prosthetic grafts. Surgery 1996;120:455-9.
230. Woodburn KR, Murtagh A, Breslin P, et al. Insonation and impedance analysis in graft surveillance. Br J Surg 1995;82:1222-5.
231. Sacks D, Robinson ML, Marinelli DL, et al. Evaluation of the peripheral arteries with duplex US after angioplasty. Radiology

1990;176:39-44.
232. Sacks D, Robinson ML, Summers TA, et al. The value of duplex sonography after peripheral artery angioplasty in predicting subacute restenosis. AJR Am J Roentgenol 1994;162:179-83.
233. Spijkerboer AM, Nass PC, de Valois JC, et al. Iliac artery stenoses after percutaneous transluminal angioplasty: follow-up with duplex ultrasonography. J Vasc Surg 1996;23:691-7.
234. Spijkerboer AM, Nass PC, de Valois JC, et al. Evaluation of femoropopliteal arteries with duplex ultrasound after angioplasty. Can we predict results at one year? Eur J Vasc Endovasc Surg 1996;12:418-23.
235. Mewissen MW, Kinney EV, Bandyk DF, et al. The role of duplex scanning versus angiography in predicting outcome after balloon angioplasty in the femoropopliteal artery. J Vasc Surg 1992; 15:860-5; discussion 865-6.
236. Miller BV, Sharp WJ, Shamma AR, et al. Surveillance for recurrent stenosis after endovascular procedures: a prospective study. Arch Surg 1991;126:867-71; discussion 871-2.
237. Vroegindeweij D, Kemper FJ, Tielbeek AV, et al. Recurrence of stenoses following balloon angioplasty and Simpson atherectomy of the femoro-popliteal segment: a randomised comparative 1-year follow-up study using colour flow duplex. Eur J Vasc Surg 1992;6:164-71.
238. Vroegindeweij D, Tielbeek AV, Buth J, et al. Patterns of recurrent disease after recanalization of femoropopliteal artery occlusions. Cardiovasc Intervent Radiol 1997;20:257-62.
239. Rubin GD, Shiau MC, Leung AN, et al. Aorta and iliac arteries: single versus multiple detector-row helical CT angiography. Radiology 2000;215:670-6.
240. Martin ML, Tay KH, Flak B, et al. Multidetector CT angiography of the aortoiliac system and lower extremities: a prospective comparison with digital subtraction angiography. AJR Am J Roentgenol 2003;180:1085-91.
241. Willmann JK, Wildermuth S, Pfammatter T, et al. Aortoiliac and renal arteries: prospective intraindividual comparison of contrast-enhanced three-dimensional MR angiography and multi-detector row CT angiography. Radiology 2003;226:798-811.
242. Willmann JK, Mayer D, Banyai M, et al. Evaluation of peripheral arterial bypass grafts with multi-detector row CT angiography: comparison with duplex US and digital subtraction angiography. Radiology 2003;229:465-74.
243. Ofer A, Nitecki SS, Linn S, et al. Multidetector CT angiography of peripheral vascular disease: a prospective comparison with intraarterial digital subtraction angiography. AJR Am J Roentgenol 2003;180:719-24.
244. Ota H, Takase K, Igarashi K, et al. MDCT compared with digital subtraction angiography for assessment of lower extremity arterial occlusive disease: importance of reviewing cross-sectional images. AJR Am J Roentgenol 2004;182:201-9.
245. Rieker O, Duber C, Schmiedt W, et al. Prospective comparison of CT angiography of the legs with intraarterial digital subtraction angiography. AJR Am J Roentgenol 1996;166:269-76.
246. Tins B, Oxtoby J, Patel S. Comparison of CT angiography with conventional arterial angiography in aortoiliac occlusive disease. Br J Radiol 2001;74:219-25.
247. Rubin GD, Schmidt AJ, Logan LJ, et al. Multi-detector row CT angiography of lower extremity arterial inflow and runoff: initial experience. Radiology 2001;221:146-58.
248. Catalano C, Fraioli F, Laghi A, et al. Infrarenal aortic and lower-extremity arterial disease: diagnostic performance of multi-detector row CT angiography. Radiology 2004;231:555-63.
249. Beregi JP, Djabbari M, Desmoucelle F, et al. Popliteal vascular disease: evaluation with spiral CT angiography. Radiology 1997;203:477-83.
250. Adriaensen ME, Kock MC, Stijnen T, et al. Peripheral arterial disease: therapeutic confidence of CT versus digital subtraction angiography and effects on additional imaging recommendations. Radiology 2004;233:385-91.
251. Rofsky NM, Adelman MA. MR angiography in the evaluation of atherosclerotic peripheral vascular disease. Radiology 2000; 214:325-38.
252. Baum RA, Rutter CM, Sunshine JH, et al. Multicenter trial to evaluate vascular magnetic resonance angiography of the lower extremity. American College of Radiology Rapid Technology Assessment Group. JAMA 1995;274:875-80.
253. Nelemans PJ, Leiner T, de Vet HC, et al. Peripheral arterial disease: meta-analysis of the diagnostic performance of MR angiography. Radiology 2000;217:105-14.
254. Khilnani NM, Winchester PA, Prince MR, et al. Peripheral vascular disease: combined 3D bolus chase and dynamic 2D MR angiography compared with x-ray angiography for treatment planning. Radiology 2002;224:63-74.
255. Visser K, Hunink MG. Peripheral arterial disease: gadolinium-enhanced MR angiography versus color-guided duplex US—a meta-analysis. Radiology 2000;216:67-77.
256. Kreitner KF, Kalden P, Neufang A, et al. Diabetes and peripheral arterial occlusive disease: prospective comparison of contrast-enhanced three-dimensional MR angiography with conventional digital subtraction angiography. AJR Am J Roentgenol 2000;174:171-9.
257. Owen RS, Carpenter JP, Baum RA, et al. Magnetic resonance imaging of angiographically occult runoff vessels in peripheral arterial occlusive disease. N Engl J Med 1992;326:1577-81.
258. Dorweiler B, Neufang A, Kreitner KF, et al. Magnetic resonance angiography unmasks reliable target vessels for pedal bypass grafting in patients with diabetes mellitus. J Vasc Surg 2002;35:766-72.
259. Hartnell G. MR angiography compared with digital subtraction angiography. AJR Am J Roentgenol 2000;175:1188-9.
260. Oser RF, Picus D, Hicks ME, et al. Accuracy of DSA in the evaluation of patency of infrapopliteal vessels. J Vasc Interv Radiol 1995;6:589-94.
261. Leyendecker JR, Elsass KD, Johnson SP, et al. The role of infrapopliteal MR angiography in patients undergoing optimal contrast angiography for chronic limb-threatening ischemia. J Vasc Interv Radiol 1998;9:545-51.
262. Maintz D, Tombach B, Juergens KU, et al. Revealing in-stent stenoses of the iliac arteries: comparison of multidetector CT with MR angiography and digital radiographic angiography in a Phantom model. AJR Am J Roentgenol 2002;179:1319-22.
263. Lee VS, Martin DJ, Krinsky GA, et al. Gadolinium-enhanced MR angiography: artifacts and pitfalls. AJR Am J Roentgenol 2000;175:197-205.
264. Sam AD 2nd, Morasch MD, Collins J, et al. Safety of gadolinium contrast angiography in patients with chronic renal insufficiency. J Vasc Surg 2003;38:313-8.
265. Hilfiker PR, Quick HH, Debatin JF. Plain and covered stent-grafts: in vitro evaluation of characteristics at three-dimensional MR angiography. Radiology 1999;211:693-7.
266. Snidow JJ, Harris VJ, Trerotola SO, et al. Interpretations and treatment decisions based on MR angiography versus conventional arteriography in symptomatic lower extremity ischemia. J Vasc Interv Radiol 1995;6:595-603.
267. Cambria RP, Kaufman JA, L'Italien GJ, et al. Magnetic resonance

angiography in the management of lower extremity arterial occlusive disease: a prospective study. J Vasc Surg 1997;25:380-9.
268. Hoch JR, Tullis MJ, Kennell TW, et al. Use of magnetic resonance angiography for the preoperative evaluation of patients with infrainguinal arterial occlusive disease. J Vasc Surg 1996;23:792-800; discussion 801.
269. Huber TS, Back MR, Ballinger RJ, et al. Utility of magnetic resonance arteriography for distal lower extremity revascularization. J Vasc Surg 1997;26:415-23; discussion 423-4.
270. Loewe C, Cejna M, Lammer J, et al. Contrast-enhanced magnetic resonance angiography in the evaluation of peripheral bypass grafts. Eur Radiol 2000;10:725-32.
271. Dorenbeck U, Seitz J, Volk M, et al. Evaluation of arterial bypass grafts of the pelvic and lower extremities with gadolinium-enhanced magnetic resonance angiography: comparison with digital subtraction angiography. Invest Radiol 2002;37:60-4.
272. Bertschinger K, Cassina PC, Debatin JF, et al. Surveillance of peripheral arterial bypass grafts with three-dimensional MR angiography: comparison with digital subtraction angiography. AJR Am J Roentgenol 2001;176:215-20.
273. Davis CP, Schopke WD, Seifert B, et al. MR angiography of patients with peripheral arterial disease before and after transluminal angioplasty. AJR Am J Roentgenol 1997;168:1027-34.
274. Bettmann MA, Heeren T, Greenfield A, et al. Adverse events with radiographic contrast agents: results of the SCVIR Contrast Agent Registry. Radiology 1997;203:611-20.
275. Waugh JR, Sacharias N. Arteriographic complications in the DSA era. Radiology 1992;182:243-6.
276. Aspelin P, Aubry P, Fransson SG, et al. Nephrotoxic effects in high-risk patients undergoing angiography. N Engl J Med 2003;348:491-9.
277. Baker CS, Wragg A, Kumar S, et al. A rapid protocol for the prevention of contrast-induced renal dysfunction: the RAPPID study. J Am Coll Cardiol 2003;41:2114-8.
278. Kay J, Chow WH, Chan TM, et al. Acetylcysteine for prevention of acute deterioration of renal function following elective coronary angiography and intervention: a randomized controlled trial. JAMA 2003;289:553-8.
279. Isenbarger DW, Kent SM, O'Malley PG. Meta-analysis of randomized clinical trials on the usefulness of acetylcysteine for prevention of contrast nephropathy. Am J Cardiol 2003;92:1454-8.
280. Stone GW, McCullough PA, Tumlin JA, et al. Fenoldopam mesylate for the prevention of contrast-induced nephropathy: a randomized controlled trial. JAMA 2003;290:2284-91.
281. Marenzi G, Marana I, Lauri G, et al. The prevention of radiocontrast-agent–induced nephropathy by hemofiltration. N Engl J Med 2003;349:1333-40.
282. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S) Lancet 1994;344:1383-9.
283. Sacks FM, Pfeffer MA, Moye LA, et al. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. Cholesterol and Recurrent Events Trial investigators. N Engl J Med 1996;335:1001-9.
284. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group. N Engl J Med 1998;339:1349-57.
285. Rubins HB, Robins SJ, Collins D, et al. Gemfibrozil for the secondary prevention of coronary heart disease in men with low levels of high-density lipoprotein cholesterol. Veterans Affairs High-Density Lipoprotein Cholesterol Intervention Trial Study Group. N Engl J Med 1999;341:410-8.
286. Blankenhorn DH, Azen SP, Crawford DW, et al. Effects of colestipol-niacin therapy on human femoral atherosclerosis. Circulation 1991;83:438-47.
287. Duffield RG, Lewis B, Miller NE, et al. Treatment of hyperlipidaemia retards progression of symptomatic femoral atherosclerosis: a randomised controlled trial. Lancet 1983;2:639-42.
288. Buchwald H, Bourdages HR, Campos CT, et al. Impact of cholesterol reduction on peripheral arterial disease in the Program on the Surgical Control of the Hyperlipidemias (POSCH). Surgery 1996;120:672-9.
289. Pedersen TR, Kjekshus J, Pyorala K, et al. Effect of simvastatin on ischemic signs and symptoms in the Scandinavian simvastatin survival study (4S). Am J Cardiol 1998;81:333-5.
290. Mohler ER 3rd, Hiatt WR, Creager MA. Cholesterol reduction with atorvastatin improves walking distance in patients with peripheral arterial disease. Circulation 2003;108:1481-6.
291. Aronow WS, Nayak D, Woodworth S, et al. Effect of simvastatin versus placebo on treadmill exercise time until the onset of intermittent claudication in older patients with peripheral arterial disease at six months and at one year after treatment. Am J Cardiol 2003;92:711-2.
292. Mondillo S, Ballo P, Barbati R, et al. Effects of simvastatin on walking performance and symptoms of intermittent claudication in hypercholesterolemic patients with peripheral vascular disease. Am J Med 2003;114:359-64.
293. Psaty BM, Smith NL, Siscovick DS, et al. Health outcomes associated with antihypertensive therapies used as first-line agents: a systematic review and meta-analysis. JAMA 1997;277:739-45.
294. 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. Erratum in: JAMA 2003;290:197.
295. Hennekens CH, Albert CM, Godfried SL, et al. Adjunctive drug therapy of acute myocardial infarction—evidence from clinical trials. N Engl J Med 1996;335:1660-7.
296. Radack K, Deck C. Beta-adrenergic blocker therapy does not worsen intermittent claudication in subjects with peripheral arterial disease: a meta-analysis of randomized controlled trials. Arch Intern Med 1991;151:1769-76.
297. Pfeffer MA, Braunwald E, Moye LA, et al. Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction. Results of the survival and ventricular enlargement trial. The SAVE Investigators. N Engl J Med 1992;327:669-77.
298. Gustafsson F, Torp-Pedersen C, Kober L, et al. Effect of angiotensin converting enzyme inhibition after acute myocardial infarction in patients with arterial hypertension. TRACE Study Group, Trandolapril Cardiac Event. J Hypertens 1997;15:793-8.
299. Yusuf S, Sleight P, Pogue J, et al. Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. N Engl J Med 2000;342:145-53. Errata in: N Engl J Med 2000;342:1376; N Engl J Med 2000;342:748.
300. Effect of intensive diabetes management on macrovascular events and risk factors in the Diabetes Control and Complications Trial. Am J Cardiol 1995;75:894-903.
301. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective

Diabetes Study (UKPDS) Group. Lancet 1998;352:837-53. Erratum in: Lancet 1999;354:602.
302. American Diabetes Association. Standards of medical care for patients with diabetes mellitus. Diabetes Care 2003;26 suppl 1:S33-50. Erratum in: Diabetes Care 2003;26:972.
303. Donohoe ME, Fletton JA, Hook A, et al. Improving foot care for people with diabetes mellitus–-a randomized controlled trial of an integrated care approach. Diabet Med 2000;17:581-7.
304. Faulkner KW, House AK, Castleden WM. The effect of cessation of smoking on the accumulative survival rates of patients with symptomatic peripheral vascular disease. Med J Aust 1983;1:217-9.
305. Lassila R, Lepantalo M. Cigarette smoking and the outcome after lower limb arterial surgery. Acta Chir Scand 1988;154:635-40.
306. Jonason T, Bergstrom R. Cessation of smoking in patients with intermittent claudication: effects on the risk of peripheral vascular complications, myocardial infarction and mortality. Acta Med Scand 1987;221:253-60.
307. Quick CR, Cotton LT. The measured effect of stopping smoking on intermittent claudication. Br J Surg 1982;69 suppl:S24-6.
308. Gardner AW. The effect of cigarette smoking on exercise capacity in patients with intermittent claudication. Vasc Med 1996; 1:181-6.
309. Law M, Tang JL. An analysis of the effectiveness of interventions intended to help people stop smoking. Arch Intern Med 1995; 155:1933-41.
310. Jorenby DE, Leischow SJ, Nides MA, et al. A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. N Engl J Med 1999;340:685-91.
311. Lowering blood homocysteine with folic acid based supplements: meta-analysis of randomised trials. Homocysteine Lowering Trialists' Collaboration. BMJ 1998;316:894-8.
312. Omenn GS, Beresford SA, Motulsky AG. Preventing coronary heart disease: B vitamins and homocysteine. Circulation 1998;97:421-4.
313. Clarke R, Collins R. Can dietary supplements with folic acid or vitamin B6 reduce cardiovascular risk? Design of clinical trials to test the homocysteine hypothesis of vascular disease. J Cardiovasc Risk 1998;5:249-55.
314. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. BMJ 2002;324:71-86. Erratum in: BMJ 2002;324:141.
315. Roderick PJ, Wilkes HC, Meade TW. The gastrointestinal toxicity of aspirin: an overview of randomised controlled trials. Br J Clin Pharmacol 1993;35:219-26.
316. Yusuf S, Zhao F, Mehta SR, et al. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. N Engl J Med 2001;345:494-502. Errata in: N Engl J Med 2001;345:1716; N Engl J Med 2001;345:1506.
317. Collaborative overview of randomised trials of antiplatelet therapy–-I: prevention of death, myocardial infarction, and stroke by prolonged antiplatelet therapy in various categories of patients. Antiplatelet Trialists' Collaboration. BMJ 1994;308:81-106. Erratum in: BMJ 1994;308:1540.
318. Girolami B, Bernardi E, Prins MH, et al. Antithrombotic drugs in the primary medical management of intermittent claudication: a meta-analysis. Thromb Haemost 1999;81:715-22.
319. Anand SS, Yusuf S. Oral anticoagulant therapy in patients with coronary artery disease: a meta-analysis. JAMA 1999;282:2058-67. Erratum in: JAMA 2000;284:45.
320. Anand SS, Yusuf S. Oral anticoagulants in patients with coronary artery disease. J Am Coll Cardiol 2003;41(4 suppl S):62S-69S. 3
321. Efficacy of oral anticoagulants compared with aspirin after infrainguinal bypass surgery (The Dutch Bypass Oral Anticoagulants or Aspirin Study): a randomised trial. Lancet 2000;355:346-51. Erratum in: Lancet 2000;355:1104.
322. Regensteiner JG. Exercise in the treatment of claudication: assessment and treatment of functional impairment. Vasc Med 1997;2:238-42.
323. Gardner AW, Poehlman ET. Exercise rehabilitation programs for the treatment of claudication pain: a meta-analysis. JAMA 1995;274:975-80.
324. Hiatt WR, Wolfel EE, Meier RH, et al. Superiority of treadmill walking exercise versus strength training for patients with peripheral arterial disease: implications for the mechanism of the training response. Circulation 1994;90:1866-74.
325. Regensteiner JG, Meyer TJ, Krupski WC, et al. Hospital vs home-based exercise rehabilitation for patients with peripheral arterial occlusive disease. Angiology 1997;48:291-300.
326. Hiatt WR, Regensteiner JG, Hargarten ME, et al. Benefit of exercise conditioning for patients with peripheral arterial disease. Circulation 1990;81:602-9.
327. Lundgren F, Dahllof AG, Schersten T, et al. Muscle enzyme adaptation in patients with peripheral arterial insufficiency: spontaneous adaptation, effect of different treatments and consequences on walking performance. Clin Sci (Lond) 1989;77:485-93.
328. Hirsch AT, Ekers MA. A comprehensive vascular medical therapeutic approach to peripheral arterial disease: the foundation of effective vascular rehabilitation. In: Fahey VA, ed. Vascular Nursing. 3rd ed. Philadelphia, Pa: WB Saunders; 1999:188-211.
329. Leng GC, Fowler B, Ernst E. Exercise for intermittent claudication. Cochrane Database Syst Rev 2000;CD000990.
330. Larsen OA, Lassen NA. Effect of daily muscular exercise in patients with intermittent claudication. Lancet 1966;2:1093-6.
331. Holm J, Dahllof AG, Bjorntorp P, et al. Enzyme studies in muscles of patients with intermittent claudication: effect of training. Scand J Clin Lab Invest Suppl 1973;128:201-5.
332. Dahllof AG, Bjorntorp P, Holm J, et al. Metabolic activity of skeletal muscle in patients with peripheral arterial insufficiency. Eur J Clin Invest 1974;4:9-15.
333. Dahllof AG, Holm J, Schersten T, et al. Peripheral arterial insufficiency, effect of physical training on walking tolerance, calf blood flow, and blood flow resistance. Scand J Rehabil Med 1976;8:19-26.
334. Creasy TS, McMillan PJ, Fletcher EW, et al. Is percutaneous transluminal angioplasty better than exercise for claudication? Preliminary results from a prospective randomised trial. Eur J Vasc Surg 1990;4:135-40.
335. Mannarino E, Pasqualini L, Innocente S, et al. Physical training and antiplatelet treatment in stage II peripheral arterial occlusive disease: alone or combined? Angiology 1991;42:513-21.
336. Patterson RB, Pinto B, Marcus B, et al. Value of a supervised exercise program for the therapy of arterial claudication. J Vasc Surg 1997;25:312-8; discussion 318-9.
337. Dawson DL, Cutler BS, Meissner MH, et al. Cilostazol has beneficial effects in treatment of intermittent claudication: results from a multicenter, randomized, prospective, double-blind trial. Circulation 1998;98:678-86.
338. Money SR, Herd JA, Isaacsohn JL, et al. Effect of cilostazol on walking distances in patients with intermittent claudication caused by peripheral vascular disease. J Vasc Surg 1998;27:267-74; discussion 274-5.
339. Clifford PC, Davies PW, Hayne JA, et al. Intermittent claudica-

tion: is a supervised exercise class worth while? Br Med J 1980; 280:1503-5.
340. Regensteiner JG, Steiner JF, Hiatt WR. Exercise training improves functional status in patients with peripheral arterial disease. J Vasc Surg 1996;23:104-15.
341. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001;285:2486-97.
342. The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Arch Intern Med 1997;157:2413-46. Erratum in Arch Intern Med 1998;158:573.
343. Gibellini R, Fanello M, Bardile AF, et al. Exercise training in intermittent claudication. Int Angiol 2000;19:8-13.
344. Gardner AW, Katzel LI, Sorkin JD, et al. Effects of long-term exercise rehabilitation on claudication distances in patients with peripheral arterial disease: a randomized controlled trial. J Cardiopulm Rehabil 2002;22:192-8.
345. ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription. 4th ed. Roitman JL, ed. Baltimore, Md: Lippincott, Williams, and Wilkins; 2001.
346. Ryan AS, Katzel LI, Gardner AW. Determinants of peak V(O2) in peripheral arterial occlusive disease patients. J Gerontol A Biol Sci Med Sci 2000;55:B302-6.
347. Pollock ML, Franklin BA, Balady GJ, et al. AHA Science Advisory. Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescription: an advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association; Position paper endorsed by the American College of Sports Medicine. Circulation 2000;101:828-33.
348. Coffman JD. Intermittent claudication—be conservative. N Engl J Med 1991;325:577-8.
349. Radack K, Wyderski RJ. Conservative management of intermittent claudication. Ann Intern Med 1990;113:135-46.
350. Savage P, Ricci MA, Lynn M, et al. Effects of home versus supervised exercise for patients with intermittent claudication. J Cardiopulm Rehabil 2001;21:152-7.
351. Degischer S, Labs KH, Hochstrasser J, et al. Physical training for intermittent claudication: a comparison of structured rehabilitation versus home-based training. Vasc Med 2002;7:109-15.
352. Perkins JM, Collin J, Creasy TS, et al. Exercise training versus angioplasty for stable claudication: long and medium term results of a prospective, randomised trial. Eur J Vasc Endovasc Surg 1996;11:409-13.
353. Chong PF, Golledge J, Greenhalgh RM, et al. Exercise therapy or angioplasty? A summation analysis. Eur J Vasc Endovasc Surg 2000;20:4-12.
354. Gelin J, Jivegard L, Taft C, et al. Treatment efficacy of intermittent claudication by surgical intervention, supervised physical exercise training compared to no treatment in unselected randomised patients I: one year results of functional and physiological improvements. Eur J Vasc Endovasc Surg 2001;22:107-13.
355. Lundgren F, Dahllof AG, Lundholm K, et al. Intermittent claudication–-surgical reconstruction or physical training? A prospective randomized trial of treatment efficiency. Ann Surg 1989;209:346-55.
356. Priebe M, Davidoff G, Lampman RM. Exercise testing and training in patients with peripheral vascular disease and lower extremity amputation. West J Med 1991;154:598-601.
357. Walker RD, Nawaz S, Wilkinson CH, et al. Influence of upper- and lower-limb exercise training on cardiovascular function and walking distances in patients with intermittent claudication. J Vasc Surg 2000;31:662-9.
358. Belcaro G, Nicolaides AN, Agus G, et al. PGE(1) treatment of severe intermittent claudication (short-term versus long-term, associated with exercise)–-efficacy and costs in a 20-week, randomized trial. Angiology 2000;51(8 pt 2):S15-26.
359. Diehm C, Kuhn A, Strauss R, et al. Effects of regular physical training in a supervised class and additional intravenous prostaglandin E1 and naftidrofuryl infusion therapy in patients with intermittent claudication–-a controlled study. Vasa Suppl 1989;28:26-30.
360. Girolami B, Bernardi E, Prins MH, et al. Treatment of intermittent claudication with physical training, smoking cessation, pentoxifylline, or nafronyl: a meta-analysis. Arch Intern Med 1999;159: 337-45.
361. Hall JA, Barnard J. The effects of an intensive 26-day program of diet and exercise on patients with peripheral vascular disease. J Cardiac Rehabil 1982;2:569-74.
362. Rosfors S, Bygdeman S, Arnetz BB, et al. Longterm neuroendocrine and metabolic effects of physical training in intermittent claudication. Scand J Rehabil Med 1989;21:7-11.
362a. Ruderman N, Devlin JT, Schneider S, Kriska A. Handbook of Exercise in Diabetes. Alexandria, VA: American Diabetes Association, 2002.
362b. ACSM's Guidelines for Exercise Testing and Prescription. In: Franklin BA, ed. Baltimore, MD: Lippincott, Williams, & Wilkins, 2000.
362c. Guidelines for Cardiac Rehabilitation and Secondary Prevention/American Association of Cardiovascular and Pulmonary Rehabilitation. Champaign, IL: Human Kinetics, 1999.
362d. Stewart KJ, Hiatt WR, Regensteiner JG, Hirsch AT. Medical progress: exercise training for claudication. N Engl J Med 2002;347:1941-51.
363. Igawa T, Tani T, Chijiwa T, et al. Potentiation of anti-platelet aggregating activity of cilostazol with vascular endothelial cells. Thromb Res 1990;57:617-23.
364. Woo SK, Kang WK, Kwon KI. Pharmacokinetic and pharmacodynamic modeling of the antiplatelet and cardiovascular effects of cilostazol in healthy humans. Clin Pharmacol Ther 2002;71:246-52.
365. Elam MB, Heckman J, Crouse JR, et al. Effect of the novel antiplatelet agent cilostazol on plasma lipoproteins in patients with intermittent claudication. Arterioscler Thromb Vasc Biol 1998;18:1942-7.
366. Otsuki M, Saito H, Xu X, et al. Cilostazol represses vascular cell adhesion molecule-1 gene transcription via inhibiting NF-kappaB binding to its recognition sequence. Atherosclerosis 2001; 158:121-8.
367. Tsuchikane E, Fukuhara A, Kobayashi T, et al. Impact of cilostazol on restenosis after percutaneous coronary balloon angioplasty. Circulation 1999;100:21-6.
368. Takahashi S, Oida K, Fujiwara R, et al. Effect of cilostazol, a cyclic AMP phosphodiesterase inhibitor, on the proliferation of rat aortic smooth muscle cells in culture. J Cardiovasc Pharmacol1992;20:900-6.
369. Beebe HG, Dawson DL, Cutler BS, et al. A new pharmacological treatment for intermittent claudication: results of a randomized, multicenter trial. Arch Intern Med 1999;159:2041-50.
370. Dawson DL, Cutler BS, Hiatt WR, et al. A comparison of cilosta-

zol and pentoxifylline for treating intermittent claudication. Am J Med 2000;109:523-30.
371. Mohler ER 3rd, Beebe HG, Salles-Cuhna S, et al. Effects of cilostazol on resting ankle pressures and exercise-induced ischemia in patients with intermittent claudication. Vasc Med 2001;6:151-6.
372. Regensteiner JG, Ware JE Jr, McCarthy WJ, et al. Effect of cilostazol on treadmill walking, community-based walking ability, and health-related quality of life in patients with intermittent claudication due to peripheral arterial disease: meta-analysis of six randomized controlled trials. J Am Geriatr Soc 2002;50:1939-46.
373. Packer M, Carver JR, Rodeheffer RJ, et al. Effect of oral milrinone on mortality in severe chronic heart failure. The PROMISE Study Research Group. N Engl J Med 1991;325:1468-75.
374. Cohn JN, Goldstein SO, Greenberg BH, et al. A dose-dependent increase in mortality with vesnarinone among patients with severe heart failure. Vesnarinone Trial Investigators. N Engl J Med 1998; 339:1810-6.
375. Strano A, Davi G, Avellone G, et al. Double-blind, crossover study of the clinical efficacy and the hemorheological effects of pentoxifylline in patients with occlusive arterial disease of the lower limbs. Angiology 1984;35:459-66.
376. Rao KM, Simel DL, Cohen HJ, et al. Effects of pentoxifylline administration on blood viscosity and leukocyte cytoskeletal function in patients with intermittent claudication. J Lab Clin Med 1990;115:738-44.
377. Schratzberger P, Dunzendorfer S, Reinisch N, et al. Mediator-dependent effects of pentoxifylline on endothelium for transmigration of neutrophils. Immunopharmacology 1999;41:65-75.
378. Dawson DL, Zheng Q, Worthy SA, et al. Failure of pentoxifylline or cilostazol to improve blood and plasma viscosity, fibrinogen, and erythrocyte deformability in claudication. Angiology 2002;53:509-20.
379. Franzini E, Sellak H, Babin-Chevaye C, et al. Effects of pentoxifylline on the adherence of polymorphonuclear neutrophils to oxidant-stimulated human endothelial cells: involvement of cyclic AMP. J Cardiovasc Pharmacol 1995;25 suppl 2:S92-5.
380. Hood SC, Moher D, Barber GG. Management of intermittent claudication with pentoxifylline: meta-analysis of randomized controlled trials. CMAJ 1996;155:1053-9.
381. Lindgarde F, Jelnes R, Bjorkman H, et al. Conservative drug treatment in patients with moderately severe chronic occlusive peripheral arterial disease. Scandinavian Study Group. Circulation 1989; 80:1549-56.
382. Porter JM, Cutler BS, Lee BY, et al. Pentoxifylline efficacy in the treatment of intermittent claudication: multicenter controlled double-blind trial with objective assessment of chronic occlusive arterial disease patients. Am Heart J 1982;104:66-72.
383. Lindgarde F, Labs KH, Rossner M. The pentoxifylline experience: exercise testing reconsidered. Vasc Med 1996;1:145-54.
384. Belch JJ, Bell PR, Creissen D, et al. Randomized, double-blind, placebo-controlled study evaluating the efficacy and safety of AS-013, a prostaglandin E1 prodrug, in patients with intermittent claudication. Circulation 1997;95:2298-302.
385. Diehm C, Balzer K, Bisler H, et al. Efficacy of a new prostaglandin E1 regimen in outpatients with severe intermittent claudication: results of a multicenter placebo-controlled double-blind trial. J Vasc Surg 1997;25:537-44.
386. Boger RH, Bode-Boger SM, Thiele W, et al. Restoring vascular nitric oxide formation by L-arginine improves the symptoms of intermittent claudication in patients with peripheral arterial occlusive disease. J Am Coll Cardiol 1998;32:1336-44.
387. Mangiafico RA, Messina R, Attina T, et al. Impact of a 4-week treatment with prostaglandin E1 on health-related quality of life of patients with intermittent claudication. Angiology 2000; 51:441-9.
388. Lievre M, Morand S, Besse B, et al. Oral Beraprost sodium, a prostaglandin I(2) analogue, for intermittent claudication: a double-blind, randomized, multicenter controlled trial. Beraprost et Claudication Intermittente (BERCI) Research Group. Circulation 2000;102:426-31.
389. Mohler ER 3rd, Hiatt WR, Olin JW, et al. Treatment of intermittent claudication with beraprost sodium, an orally active prostaglandin I2 analogue: a double-blinded, randomized, controlled trial. J Am Coll Cardiol 2003;41:1679-86.
390. Yang HT, Deschenes MR, Ogilvie RW, et al. Basic fibroblast growth factor increases collateral blood flow in rats with femoral arterial ligation. Circ Res 1996;79:62-9.
391. Takeshita S, Zheng LP, Brogi E, et al. Therapeutic angiogenesis: a single intraarterial bolus of vascular endothelial growth factor augments revascularization in a rabbit ischemic hind limb model. J Clin Invest 1994;93:662-70.
392. Tsurumi Y, Takeshita S, Chen D, et al. Direct intramuscular gene transfer of naked DNA encoding vascular endothelial growth factor augments collateral development and tissue perfusion. Circulation 1996;94:3281-90.
393. Ohara N, Koyama H, Miyata T, et al. Adenovirus-mediated ex vivo gene transfer of basic fibroblast growth factor promotes collateral development in a rabbit model of hind limb ischemia. Gene Ther 2001;8:837-45.
394. Lazarous DF, Unger EF, Epstein SE, et al. Basic fibroblast growth factor in patients with intermittent claudication: results of a phase I trial. J Am Coll Cardiol 2000;36:1239-44.
395. Lederman RJ, Mendelsohn FO, Anderson RD, et al. Therapeutic angiogenesis with recombinant fibroblast growth factor-2 for intermittent claudication (the TRAFFIC study): a randomised trial. Lancet 2002;359:2053-8.
396. Cooper LT Jr, Hiatt WR, Creager MA, et al. Proteinuria in a placebo-controlled study of basic fibroblast growth factor for intermittent claudication. Vasc Med 2001;6:235-9.
397. Rajagopalan S, Trachtenberg J, Mohler E, et al. Phase I study of direct administration of a replication deficient adenovirus vector containing the vascular endothelial growth factor cDNA (CI-1023) to patients with claudication. Am J Cardiol 2002;90:512-6.
398. Rajagopalan S, Mohler ER 3rd, Lederman RJ, et al. Regional angiogenesis with vascular endothelial growth factor in peripheral arterial disease: a phase II randomized, double-blind, controlled study of adenoviral delivery of vascular endothelial growth factor 121 in patients with disabling intermittent claudication. Circulation 2003;108:1933-8.
399. Cooke JP, Creager MA. Hypercholesterolemia, atherosclerosis, and the NO synthase pathway. In: Vallance PJ, Webb DJ, eds. Vascular Endothelium in Human Physiology and Pathophysiology. Amsterdam, the Netherlands: Harwood Academic Publishers, 2000:147-70.
400. Creager MA, Gallagher SJ, Girerd XJ, et al. L-arginine improves endothelium-dependent vasodilation in hypercholesterolemic humans. J Clin Invest 1992;90:1248-53.
401. Maxwell AJ, Anderson BE, Cooke JP. Nutritional therapy for peripheral arterial disease: a double-blind, placebo-controlled, randomized trial of HeartBar. Vasc Med 2000;5:11-9.
402. Brevetti G, Chiariello M, Ferulano G, et al. Increases in walking distance in patients with peripheral vascular disease treated with

L-carnitine: a double-blind, cross-over study. Circulation 1988; 77:767-73.
403. Brevetti G, Diehm C, Lambert D. European multicenter study on propionyl-L-carnitine in intermittent claudication. J Am Coll Cardiol 1999;34:1618-24.
404. Brevetti G, Perna S, Sabba C, et al. Propionyl-L-carnitine in intermittent claudication: double-blind, placebo-controlled, dose titration, multicenter study. J Am Coll Cardiol 1995;26:1411-6.
405. Hiatt WR, Regensteiner JG, Creager MA, et al. Propionyl-L-carnitine improves exercise performance and functional status in patients with claudication. Am J Med 2001;110:616-22.
406. Pittler MH, Ernst E. Ginkgo biloba extract for the treatment of intermittent claudication: a meta-analysis of randomized trials. Am J Med 2000;108:276-81.
407. Kleijnen J, Mackerras D. Vitamin E for intermittent claudication. Cochrane Database Syst Rev 2000;(2):CD000987.
408. Tornwall ME, Virtamo J, Haukka JK, et al. The effect of alpha-tocopherol and beta-carotene supplementation on symptoms and progression of intermittent claudication in a controlled trial. Atherosclerosis 1999;147:193-7.
409. Ernst E. Chelation therapy for peripheral arterial occlusive disease: a systematic review. Circulation 1997;96:1031-3.
410. Villarruz MV, Dans A, Tan F. Chelation therapy for atherosclerotic cardiovascular disease. Cochrane Database Syst Rev 2002; CD002785.
411. Olszewer E, Sabbag FC, Carter JP. A pilot double-blind study of sodium-magnesium EDTA in peripheral vascular disease. J Natl Med Assoc 1990;82:173-7.
412. Sloth-Nielsen J, Guldager B, Mouritzen C, et al. Arteriographic findings in EDTA chelation therapy on peripheral arteriosclerosis. Surg Gynecol Obstet 1991;162:122-5.
413. van Rij AM, Solomon C, Packer SG, et al. Chelation therapy for intermittent claudication: a double-blind, randomized, controlled trial. Circulation 1994;90:1194-9.
414. Guldager B, Jelnes R, Jorgensen SJ, et al. EDTA treatment of intermittent claudication—a double-blind, placebo-controlled study. J Intern Med 1992;231:261-7.
415. Johnston KW, Rae M, Hogg-Johnston SA, et al. 5-year results of a prospective study of percutaneous transluminal angioplasty. Ann Surg 1987;206:403-13.
416. Lofberg AM, Karacagil S, Ljungman C, et al. Percutaneous transluminal angioplasty of the femoropopliteal arteries in limbs with chronic critical lower limb ischemia. J Vasc Surg 2001;34:114-21.
417. Jamsen T, Manninen H, Tulla H, et al. The final outcome of primary infrainguinal percutaneous transluminal angioplasty in 100 consecutive patients with chronic critical limb ischemia. J Vasc Interv Radiol 2002;13:455-63.
418. Powell RJ, Fillinger M, Walsh DB, et al. Predicting outcome of angioplasty and selective stenting of multisegment iliac artery occlusive disease. J Vasc Surg 2000;32:564-9.
419. Laborde JC, Palmaz JC, Rivera FJ, et al. Influence of anatomic distribution of atherosclerosis on the outcome of revascularization with iliac stent placement. J Vasc Interv Radiol 1995;6:513-21.
420. Capek P, McLean GK, Berkowitz HD. Femoropopliteal angioplasty: factors influencing long-term success. Circulation 1991; 83(2 suppl):I70-80.
421. Stokes KR, Strunk HM, Campbell DR, et al. Five-year results of iliac and femoropopliteal angioplasty in diabetic patients. Radiology 1990;174(3 pt 2):977-82.
422. Johnston KW. Iliac arteries: reanalysis of results of balloon angioplasty. Radiology 1993;186:207-12.
423. Clark TW, Groffsky JL, Soulen MC. Predictors of long-term patency after femoropopliteal angioplasty: results from the STAR registry. J Vasc Interv Radiol 2001;12:923-33.
424. Beck AH, Muhe A, Ostheim W, et al. Long-term results of percutaneous transluminal angioplasty: a study of 4750 dilatations and local lyses. Eur J Vasc Surg 1989;3:245-52.
425. Palmaz JC, Laborde JC, Rivera FJ, et al. Stenting of the iliac arteries with the Palmaz stent: experience from a multicenter trial. Cardiovasc Intervent Radiol 1992;15:291-7.
426. Soder HK, Manninen HI, Jaakkola P, et al. Prospective trial of infrapopliteal artery balloon angioplasty for critical limb ischemia: angiographic and clinical results. J Vasc Interv Radiol 2000;11:1021-31.
427. Sapoval MR, Chatellier G, Long AL, et al. Self-expandable stents for the treatment of iliac artery obstructive lesions: long-term success and prognostic factors. AJR Am J Roentgenol 1996;166:1173-9.
428. Bakal CW, Sprayregen S, Scheinbaum K, et al. Percutaneous transluminal angioplasty of the infrapopliteal arteries: results in 53 patients. AJR Am J Roentgenol 1990;154:171-4.
429. Brown KT, Moore ED, Getrajdman GI, et al. Infrapopliteal angioplasty: long-term follow-up. J Vasc Interv Radiol 1993;4:139-44.
430. Bull PG, Mendel H, Hold M, et al. Distal popliteal and tibioperoneal transluminal angioplasty: long-term follow-up. J Vasc Interv Radiol 1992;3:45-53.
431. Timaran CH, Stevens SL, Freeman MB, et al. External iliac and common iliac artery angioplasty and stenting in men and women. J Vasc Surg 2001;34:440-6.
432. Timaran CH, Stevens SL, Grandas OH, et al. Influence of hormone replacement therapy on the outcome of iliac angioplasty and stenting. J Vasc Surg 2001;33(2 suppl):S85-92.
433. Avino AJ, Bandyk DF, Gonsalves AJ, et al. Surgical and endovascular intervention for infrainguinal vein graft stenosis. J Vasc Surg 1999;29:60-70; discussion 70-1.
434. Whittemore AD, Donaldson MC, Polak JF, et al. Limitations of balloon angioplasty for vein graft stenosis. J Vasc Surg 1991; 14:340-5.
435. Goh RH, Sniderman KW, Kalman PG. Long-term follow-up of management of failing in situ saphenous vein bypass grafts using endovascular intervention techniques. J Vasc Interv Radiol 2000;11:705-12.
436. Bosch JL, Hunink MG. Meta-analysis of the results of percutaneous transluminal angioplasty and stent placement for aortoiliac occlusive disease. Radiology 1997;204:87-96. Erratum in: Radiology 1997;205:584.
437. Hunink MG, Wong JB, Donaldson MC, et al. Patency results of percutaneous and surgical revascularization for femoropopliteal arterial disease. Med Decis Making 1994;14:71-81.
438. Hunink MG, Wong JB, Donaldson MC, et al. Revascularization for femoropopliteal disease: a decision and cost-effectiveness analysis. JAMA 1995;274:165-71.
438a. Kandarpa K, Becker, BJ, Hunink, M, et al. J Vasc Interv Radiol 2001;12:683-95.
439. Udoff EJ, Barth KH, Harrington DP, et al. Hemodynamic significance of iliac artery stenosis: pressure measurements during angiography. Radiology 1979;132:289-93.
440. Tetteroo E, van Engelen AD, Spithoven JH, et al. Stent placement after iliac angioplasty: comparison of hemodynamic and angiographic criteria. Dutch Iliac Stent Trial Study Group. Radiology 1996;201:155-9.
441. Kinney TB, Rose SC. Intraarterial pressure measurements during angiographic evaluation of peripheral vascular disease: techniques, interpretation, applications, and limitations. AJR Am J

Roentgenol 1996;166:277-84.
442. Bonn J. Percutaneous vascular intervention: value of hemodynamic measurements. Radiology 1996;201:18-20.
443. Whyman MR, Fowkes FG, Kerracher EM, et al. Randomised controlled trial of percutaneous transluminal angioplasty for intermittent claudication. Eur J Vasc Endovasc Surg 1996;12:167-72.
444. Whyman MR, Fowkes FG, Kerracher EM, et al. Is intermittent claudication improved by percutaneous transluminal angioplasty? A randomized controlled trial. J Vasc Surg 1997;26:551-7.
445. Feinglass J, McCarthy WJ, Slavensky R, et al. Functional status and walking ability after lower extremity bypass grafting or angioplasty for intermittent claudication: results from a prospective outcomes study. J Vasc Surg 2000;31(1 pt 1):93-103.
446. Holm J, Arfvidsson B, Jivegard L, et al. Chronic lower limb ischaemia. A prospective randomised controlled study comparing the 1-year results of vascular surgery and percutaneous transluminal angioplasty (PTA). Eur J Vasc Surg 1991;5:517-22.
447. Wolf GL, Wilson SE, Cross AP, et al. Surgery or balloon angioplasty for peripheral vascular disease: a randomized clinical trial. Principal investigators and their Associates of Veterans Administration Cooperative Study Number 199. J Vasc Interv Radiol 1993;4:639-48.
448. Wilson SE, Wolf GL, Cross AP. Percutaneous transluminal angioplasty versus operation for peripheral arteriosclerosis: report of a prospective randomized trial in a selected group of patients. J Vasc Surg 1989;9:1-9.
449. Gray BH, Sullivan TM, Childs MB, et al. High incidence of restenosis/reocclusion of stents in the percutaneous treatment of long-segment superficial femoral artery disease after suboptimal angioplasty. J Vasc Surg 1997;25:74-83.
450. de Vries SO, Visser K, de Vries JA, et al. Intermittent claudication: cost-effectiveness of revascularization versus exercise therapy. Radiology 2002;222:25-36.
451. Treesak C, Kasemsup V, Treat-Jacobson D, et al. Cost-effectiveness of exercise training to improve claudication symptoms in patients with peripheral arterial disease. Vasc Med 2004;9:279-85.
452. Tetteroo E, van der Graaf Y, Bosch JL, et al. Randomised comparison of primary stent placement versus primary angioplasty followed by selective stent placement in patients with iliac-artery occlusive disease. Dutch Iliac Stent Trial Study Group. Lancet 1998;351:1153-9.
453. Richter GM, Roeren T, Noeldge G, et al. [Initial long-term results of a randomized 5-year study: iliac stent implantation versus PTA] Vasa Suppl 1992;35:192-3.
454. Bosch JL, Tetteroo E, Mali WP, et al. Iliac arterial occlusive disease: cost-effectiveness analysis of stent placement versus percutaneous transluminal angioplasty. Dutch Iliac Stent Trial Study Group. Radiology 1998;208:641-8.
455. Bosch JL, Haaring C, Meyerovitz MF, et al. Cost-effectiveness of percutaneous treatment of iliac artery occlusive disease in the United States. AJR Am J Roentgenol 2000;175:517-21.
456. Muradin GS, Bosch JL, Stijnen T, et al. Balloon dilation and stent implantation for treatment of femoropopliteal arterial disease: meta-analysis. Radiology 2001;221:137-45.
457. Cejna M, Thurnher S, Illiasch H, et al. PTA versus Palmaz stent placement in femoropopliteal artery obstructions: a multicenter prospective randomized study. J Vasc Interv Radiol 2001;12:23-31.
458. Grimm J, Muller-Hulsbeck S, Jahnke T, et al. Randomized study to compare PTA alone versus PTA with Palmaz stent placement for femoropopliteal lesions. J Vasc Interv Radiol 2001;12:935-42.
459. Vroegindeweij D, Vos LD, Tielbeek AV, et al. Balloon angioplasty combined with primary stenting versus balloon angioplasty alone in femoropopliteal obstructions: a comparative randomized study. Cardiovasc Intervent Radiol 1997;20:420-5.
460. Zdanowski Z, Albrechtsson U, Lundin A, et al. Percutaneous transluminal angioplasty with or without stenting for femoropopliteal occlusions? A randomized controlled study. Int Angiol 1999;18:251-5.
461. Schillinger M, Mlekusch W, Haumer M, et al. Angioplasty and elective stenting of de novo versus recurrent femoropopliteal lesions: 1-year follow-up. J Endovasc Ther 2003;10:288-97.
462. Vroegindeweij D, Tielbeek AV, Buth J, et al. Directional atherectomy versus balloon angioplasty in segmental femoropopliteal artery disease: two-year follow-up with color-flow duplex scanning. J Vasc Surg 1995;21:255-68; discussion 268-9.
463. Nakamura S, Conroy RM, Gordon IL, et al. A randomized trial of transcutaneous extraction atherectomy in femoral arteries: intravascular ultrasound observations. J Clin Ultrasound 1995;23:461-71.
464. Jahnke T, Link J, Muller-Hulsbeck S, et al. Treatment of infrapopliteal occlusive disease by high-speed rotational atherectomy: initial and mid-term results. J Vasc Interv Radiol 2001;12:221-6.
465. Belli AM, Cumberland DC, Procter AE, et al. Follow-up of conventional angioplasty versus laser thermal angioplasty for total femoropopliteal artery occlusions: results of a randomized trial. J Vasc Interv Radiol 1991;2:485-8.
466. Fisher CM, Fletcher JP, May J, et al. No additional benefit from laser in balloon angioplasty of the superficial femoral artery. Eur J Vasc Endovasc Surg 1996;11:349-52.
467. Jeans WD, Murphy P, Hughes AO, et al. Randomized trial of laser-assisted passage through occluded femoro-popliteal arteries. Br J Radiol 1990;63:19-21.
468. Lammer J, Pilger E, Decrinis M, et al. Pulsed excimer laser versus continuous-wave Nd:YAG laser versus conventional angioplasty of peripheral arterial occlusions: prospective, controlled, randomised trial. Lancet 1992;340:1183-8.
469. Minar E, Pokrajac B, Maca T, et al. Endovascular brachytherapy for prophylaxis of restenosis after femoropopliteal angioplasty: results of a prospective randomized study. Circulation 2000;102:2694-9.
470. Waksman R, Laird JR, Jurkovitz CT, et al. Intravascular radiation therapy after balloon angioplasty of narrowed femoropopliteal arteries to prevent restenosis: results of the PARIS feasibility clinical trial. J Vasc Interv Radiol 2001;12:915-21.
471. Sidawy AN, Weiswasser JM, Waksman R. Peripheral vascular brachytherapy. J Vasc Surg 2002;35:1041-7.
472. Zehnder T, von Briel C, Baumgartner I, et al. Endovascular brachytherapy after percutaneous transluminal angioplasty of recurrent femoropopliteal obstructions. J Endovasc Ther 2003; 10:304-11.
473. Krueger K, Landwehr P, Bendel M, et al. Endovascular gamma irradiation of femoropopliteal de novo stenoses immediately after PTA: interim results of prospective randomized controlled trial. Radiology 2002;224:519-28.
474. Jahnke T, Andresen R, Muller-Hulsbeck S, et al. Hemobahn stentgrafts for treatment of femoropopliteal arterial obstructions: midterm results of a prospective trial. J Vasc Interv Radiol 2003; 14:41-51.
475. Saxon RR, Coffman JM, Gooding JM, et al. Long-term results of ePTFE stent-graft versus angioplasty in the femoropopliteal artery: single center experience from a prospective, randomized trial. J Vasc Interv Radiol 2003;14:303-11.
476. Duda SH, Poerner TC, Wiesinger B, et al. Drug-eluting stents:

potential applications for peripheral arterial occlusive disease. J Vasc Interv Radiol 2003;14:291-301.
477. Duda SH, Pusich B, Richter G, et al. Sirolimus-eluting stents for the treatment of obstructive superficial femoral artery disease: six-month results. Circulation 2002;106:1505-9.
478. Girolami B, Bernardi E, Prins MH, et al. Antiplatelet therapy and other interventions after revascularisation procedures in patients with peripheral arterial disease: a meta-analysis. Eur J Vasc Endovasc Surg 2000;19:370-80.
479. Watson HR, Bergqvist D. Antithrombotic agents after peripheral transluminal angioplasty: a review of the studies, methods and evidence for their use. Eur J Vasc Endovasc Surg 2000;19:445-50.
480. Timaran CH, Prault TL, Stevens SL, et al. Iliac artery stenting versus surgical reconstruction for TASC (TransAtlantic Inter-Society Consensus) type B and type C iliac lesions. J Vasc Surg 2003;38:272-8.
481. Reed AB, Conte MS, Donaldson MC, et al. The impact of patient age and aortic size on the results of aortobifemoral bypass grafting. J Vasc Surg 2003;37:1219-25.
482. Olsen PS, Gustafsen J, Rasmussen L, et al. Long-term results after arterial surgery for arteriosclerosis of the lower limbs in young adults. Eur J Vasc Surg 1988;2:15-8.
483. Green RM, Abbott WM, Matsumoto T, et al. Prosthetic above-knee femoropopliteal bypass grafting: five-year results of a randomized trial. J Vasc Surg 2000;31:417-25.
484. Eagle KA, Berger PB, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). Circulation 2002;105:1257-67.
485. de Vries SO, Hunink MG. Results of aortic bifurcation grafts for aortoiliac occlusive disease: a meta-analysis. J Vasc Surg 1997; 26:558-69.
486. van der Vliet JA, Scharn DM, de Waard JW, et al. Unilateral vascular reconstruction for iliac obstructive disease. J Vasc Surg 1994;19:610-4.
487. Ricco JB. Unilateral iliac artery occlusive disease: a randomized multicenter trial examining direct revascularization versus crossover bypass. Association Universitaire de Recherche en Chirurgie. Ann Vasc Surg 1992;6:209-19.
488. Raptis S, Faris I, Miller J, et al. The fate of the aortofemoral graft. Eur J Vasc Endovasc Surg 1995;9:97-102.
489. Oskam J, van den Dungen JJ, Boontje AH. Thrombo-endarterectomy for obstructive disease of the common iliac artery. Cardiovasc Surg 1996;4:356-9.
490. Pretre R, Katchatourian G, Bednarkiewicz M, et al. Aortoiliac endarterectomy: a 9-year experience. Thorac Cardiovasc Surg 1992;40:152-4.
491. Radoux JM, Maiza D, Coffin O. Long-term outcome of 121 iliofemoral endarterectomy procedures. Ann Vasc Surg 2001; 15:163-70.
492. Mingoli A, Sapienza P, Feldhaus RJ, et al. Comparison of femorofemoral and aortofemoral bypass for aortoiliac occlusive disease. J Cardiovasc Surg (Torino) 2001;42:381-7.
493. Mohan CR, Sharp WJ, Hoballah JJ, et al. A comparative evaluation of externally supported polytetrafluoroethylene axillobifemoral and axillounifemoral bypass grafts. J Vasc Surg 1995; 21:801-8; discussion 808-9.
494. Harrington ME, Harrington EB, Haimov M, et al. Axillofemoral bypass: compromised bypass for compromised patients. J Vasc Surg 1994;20:195-201.
495. Onohara T, Komori K, Kume M, et al. Multivariate analysis of long-term results after an axillobifemoral and aortobifemoral bypass in patients with aortoiliac occlusive disease. J Cardiovasc Surg (Torino) 2000;41:905-10.
496. Martin D, Katz SG. Axillofemoral bypass for aortoiliac occlusive disease. Surg Gynecol Obstet 2000;180:100-3.
497. Faries PL, LoGerfo FW, Hook SC, et al. The impact of diabetes on arterial reconstructions for multilevel arterial occlusive disease. Surg Gynecol Obstet 2001;181:251-5.
498. Archie JP Jr. Femoropopliteal bypass with either adequate ipsilateral reversed saphenous vein or obligatory polytetrafluoroethylene. Ann Vasc Surg 1994;8:475-84.
499. Nicoloff AD, Taylor LM Jr, McLafferty RB, et al. Patient recovery after infrainguinal bypass grafting for limb salvage. J Vasc Surg 1998;27:256-63; discussion 264-6.
500. Gentile AT, Lee RW, Moneta GL, et al. Results of bypass to the popliteal and tibial arteries with alternative sources of autogenous vein. J Vasc Surg 1996;23:272-9; discussion 279-80.
500a. Allen BT, Reilly JM, Rubin BG, et al. Femoropopliteal bypass for claudication: vein vs. PTFE. Ann Vasc Surg 1996;10:178-85.
501. Taylor LM Jr, Edwards JM, Porter JM. Present status of reversed vein bypass grafting: five-year results of a modern series. J Vasc Surg 1990;11:193-205; discussion 205-6.
502. Schweiger H, Klein P, Lang W. Tibial bypass grafting for limb salvage with ringed polytetrafluoroethylene prostheses: results of primary and secondary procedures. J Vasc Surg 1993;18:867-74.
503. Londrey GL, Ramsey DE, Hodgson KJ, et al. Infrapopliteal bypass for severe ischemia: comparison of autogenous vein, composite, and prosthetic grafts. J Vasc Surg 1991;13:631-6.
504. McCarthy WJ, Pearce WH, Flinn WR, et al. Long-term evaluation of composite sequential bypass for limb-threatening ischemia. J Vasc Surg 1992;15:761-9; discussion 769-70.
505. Desai TR, Meyerson SL, Skelly CL, et al. Patency and limb salvage after infrainguinal bypass with severely compromised ("blind") outflow. Arch Surg 2001;136:635-42.
506. Towne JB, Bernhard VM, Rollins DL, et al. Profundaplasty in perspective: limitations in the long-term management of limb ischemia. Surgery 1981;90:1037-46.
507. Kalman PG, Johnston KW, Walker PM. The current role of isolated profundaplasty. J Cardiovasc Surg (Torino) 1990;31:107-11.
508. AbuRahma AF, Robinson PA, Holt SM. Prospective controlled study of polytetrafluoroethylene versus saphenous vein in claudicant patients with bilateral above knee femoropopliteal bypasses. Surgery 1999;126:594-601; discussion 601-2.
509. Johnson WC, Lee KK. A comparative evaluation of polytetrafluoroethylene, umbilical vein, and saphenous vein bypass grafts for femoral-popliteal above-knee revascularization: a prospective randomized Department of Veterans Affairs cooperative study. J Vasc Surg 2000;32:268-77.
510. Klinkert P, Schepers A, Burger DH, et al. Vein versus polytetrafluoroethylene in above-knee femoropopliteal bypass grafting: five-year results of a randomized controlled trial. J Vasc Surg 2003;37:149-55.
511. Baldwin ZK, Pearce BJ, Curi MA, et al. Limb salvage after infrainguinal bypass graft failure. J Vasc Surg 2004;39:951-7.
512. Veith FJ, Gupta SK, Ascer E, et al. Six-year prospective multicenter randomized comparison of autologous saphenous vein and expanded polytetrafluoroethylene grafts in infrainguinal arterial reconstructions. J Vasc Surg 1986;3:104-14.
513. Brothers TE, Greenfield LJ. Long-term results of aortoiliac reconstruction. J Vasc Interv Radiol 1990;1:49-55.

514. Criado E, Burnham SJ, Tinsley EA Jr, et al. Femorofemoral bypass graft: analysis of patency and factors influencing long-term outcome. J Vasc Surg 1993;18:495-504; discussion 504-5.
515. Perler BA, Williams GM. Does donor iliac artery percutaneous transluminal angioplasty or stent placement influence the results of femorofemoral bypass? Analysis of 70 consecutive cases with long-term follow-up. J Vasc Surg 1996;24:363-9; discussion 369-70.
516. Szilagyi DE, Elliott JP Jr, Smith RF, et al. A thirty-year survey of the reconstructive surgical treatment of aortoiliac occlusive disease. J Vasc Surg 1986;3:421-36.
517. Johnson WC, Lee KK. Comparative evaluation of externally supported Dacron and polytetrafluoroethylene prosthetic bypasses for femorofemoral and axillofemoral arterial reconstructions. Veterans Affairs Cooperative Study #141. J Vasc Surg 1999;30:1077-83.
518. Rutherford RB, Baker JD, Ernst C, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg 1997;26:517-38. Erratum in: J Vasc Surg 2001;33:805.
519. Intravenous pentoxifylline for the treatment of chronic critical limb ischaemia. The European Study Group. Eur J Vasc Endovasc Surg 1995;9:426-36.
520. Efficacy and clinical tolerance of parenteral pentoxifylline in the treatment of critical lower limb ischemia: a placebo controlled multicenter study. Norwegian Pentoxifylline Multicenter Trial Group. Int Angiol 1996;15:75-80.
521. Nizankowski R, Krolikowski W, Bielatowicz J, et al. Prostacyclin for ischemic ulcers in peripheral arterial disease: a random assignment, placebo controlled study. Thromb Res 1985;37:21-8.
522. Negus D, Irving JD, Friedgood A. Intra-arterial prostacyclin compared to Praxilene in the management of severe lower limb ischaemia: a double blind trial. J Cardiovasc Surg (Torino) 1987;28:196-9.
523. Eklund AE, Eriksson G, Olsson AG. A controlled study showing significant short term effect of prostaglandin E1 in healing of ischaemic ulcers of the lower limb in man. Prostaglandins Leukot Med 1982;8:265-71.
524. Schuler JJ, Flanigan DP, Holcroft JW, et al. Efficacy of prostaglandin E1 in the treatment of lower extremity ischemic ulcers secondary to peripheral vascular occlusive disease: results of a prospective randomized, double-blind, multicenter clinical trial. J Vasc Surg 1984;1:160-70.
525. Telles GS, Campbell WB, Wood RF, et al. Prostaglandin E1 in severe lower limb ischaemia: a double-blind controlled trial. Br J Surg 1984;71:506-8.
526. Belch JJ, McKay A, McArdle B, et al. Epoprostenol (prostacyclin) and severe arterial disease: a double-blind trial. Lancet 1983;1:315-7.
527. Cronenwett JL, Zelenock GB, Whitehouse WM Jr, et al. Prostacyclin treatment of ischemic ulcers and rest pain in unreconstructible peripheral arterial occlusive disease. Surgery 1986;100:369-75.
528. Trubestein G, Diehm C, Gruss JD, et al. Prostaglandin E1 in chronic arterial disease—a multicenter study. Vasa Suppl 1987;17:39-43.
529. The effect of ciprostene in patients with peripheral vascular disease (PVD) characterized by ischemic ulcers. The Ciprostene Study Group. J Clin Pharmacol 1991;31:81-7.
530. Prostanoids for chronic critical leg ischemia. A randomized, controlled, open-label trial with prostaglandin E1. The ICAI Study Group. Ischemia Cronica degli Arti Inferiori. Ann Intern Med 1999;130:412-21.
531. Trubestein G, von Bary S, Breddin K, et al. Intravenous prostaglandin E1 versus pentoxifylline therapy in chronic arterial occlusive disease—a controlled randomised multicenter study. Vasa Suppl 1989;28:44-9.
532. Balzer K, Bechara G, Bisler H, et al. Reduction of ischaemic rest pain in advanced peripheral arterial occlusive disease: a double blind placebo controlled trial with iloprost. Int Angiol 1991; 10:229-32.
533. Diehm C, Abri O, Baitsch G, et al. [Iloprost, a stable prostacyclin derivative, in stage 4 arterial occlusive disease: a placebo-controlled multicenter study] Dtsch Med Wochenschr 1989;114:783-8.
534. Norgren L, Alwmark A, Angqvist KA, et al. A stable prostacyclin analogue (iloprost) in the treatment of ischaemic ulcers of the lower limb: a Scandinavian-Polish placebo controlled, randomised multicenter study. Eur J Vasc Surg 1990;4:463-7.
535. Brock FE, Abri O, Baitsch G, et al. [Iloprost in the treatment of ischemic tissue lesions in diabetics: results of a placebo-controlled multicenter study with a stable prostacyclin derivative] Schweiz Med Wochenschr 1990;120:1477-82.
536. Treatment of limb threatening ischaemia with intravenous iloprost: a randomised double-blind placebo controlled study. U.K. Severe Limb Ischaemia Study Group. Eur J Vasc Surg 1991; 5:511-6.
537. Two randomised and placebo-controlled studies of an oral prostacyclin analogue (Iloprost) in severe leg ischaemia. The Oral Iloprost in severe Leg Ischaemia Study Group. Eur J Vasc Endovasc Surg 2000;20:358-62.
538. Isner JM, Walsh K, Symes J, et al. Arterial gene therapy for therapeutic angiogenesis in patients with peripheral artery disease. Circulation 1995;91:2687-92.
539. Isner JM, Pieczek A, Schainfeld R, et al. Clinical evidence of angiogenesis after arterial gene transfer of phVEGF165 in patient with ischaemic limb. Lancet 1996;348:370-4.
540. Isner JM. Arterial gene transfer of naked DNA for therapeutic angiogenesis: early clinical results. Adv Drug Deliv Rev 1998;30(1-3):185-97.
541. Baumgartner I, Pieczek A, Manor O, et al. Constitutive expression of phVEGF165 after intramuscular gene transfer promotes collateral vessel development in patients with critical limb ischemia. Circulation 1998;97:1114-23.
542. Nasr MK, McCarthy RJ, Hardman J, et al. The increasing role of percutaneous transluminal angioplasty in the primary management of critical limb ischaemia. Eur J Vasc Endovasc Surg 2002;23:398-403.
543. Isner JM, Rosenfield K. Redefining the treatment of peripheral artery disease: role of percutaneous revascularization. Circulation 1993;88(4 pt 1):1534-57.
544. Durham JR, Horowitz JD, Wright JG, et al. Percutaneous transluminal angioplasty of tibial arteries for limb salvage in the high-risk diabetic patient. Ann Vasc Surg 1994;8:48-53.
545. Isner JM, Pieczek A, Rosenfield K. Images in cardiovascular medicine: untreated gangrene in patients with peripheral artery disease. Circulation 1994;89:482-3.
546. Gray BH, Laird JR, Ansel GM, et al. Complex endovascular treatment for critical limb ischemia in poor surgical candidates: a pilot study. J Endovasc Ther 2002;9:599-604.
547. Faglia E, Mantero M, Caminiti M, et al. Extensive use of peripheral angioplasty, particularly infrapopliteal, in the treatment of ischaemic diabetic foot ulcers: clinical results of a multicentric study of 221 consecutive diabetic subjects. J Intern Med 2002;252:225-32.

548. Ingle H, Nasim A, Bolia A, et al. Subintimal angioplasty of isolated infragenicular vessels in lower limb ischemia: long-term results. J Endovasc Ther 2002;9:411-6. Erratum in: J Endovasc Ther 2002;9:A-6.
549. Gordon IL, Conroy RM, Arefi M, et al. Three-year outcome of endovascular treatment of superficial femoral artery occlusion. Arch Surg 2001;136:221-8.
550. Spence LD, Hartnell GG, Reinking G, et al. Diabetic versus nondiabetic limb-threatening ischemia: outcome of percutaneous iliac intervention. AJR Am J Roentgenol 1999;172:1335-41.
551. Marzelle J, Fichelle JM, Cormier F, et al. Outcome of infrainguinal endovascular revascularization procedures for limb-threatening ischemia. Ann Vasc Surg 1995;9 suppl:S24-31.
552. Bernstein EF, Rhodes GA, Stuart SH, et al. Toe pulse reappearance time in prediction of aortofemoral bypass success. Ann Surg 1981;193:201-5.
553. Bakal CW, Cynamon J, Sprayregen S. Infrapopliteal percutaneous transluminal angioplasty: what we know. Radiology 1996;200:36-43.
554. Berridge DC, Gregson RH, Hopkinson BR, et al. Randomized trial of intra-arterial recombinant tissue plasminogen activator, intravenous recombinant tissue plasminogen activator and intra-arterial streptokinase in peripheral arterial thrombolysis. Br J Surg 1991;78:988-95.
555. Graor RA, Risius B, Young JR, et al. Thrombolysis of peripheral arterial bypass grafts: surgical thrombectomy compared with thrombolysis: a preliminary report. J Vasc Surg 1988;7:347-55.
556. Ouriel K, Kandarpa K, Schuerr DM, et al. Prourokinase versus urokinase for recanalization of peripheral occlusions, safety and efficacy: the PURPOSE trial. J Vasc Interv Radiol 1999;10:1083-91.
557. Mahler F, Schneider E, Hess H; Steering Committee, Study on Local Thrombolysis. Recombinant tissue plasminogen activator versus urokinase for local thrombolysis of femoropopliteal occlusions: a prospective, randomized multicenter trial. J Endovasc Ther 2001;8:638-47.
558. Khosla S, Jain P, Manda R, et al. Acute and long-term results after intra-arterial thrombolysis of occluded lower extremity bypass grafts using recombinant tissue plasminogen activator for acute limb-threatening ischemia. Am J Ther 2003;10:3-6.
559. Ouriel K, Shortell CK, DeWeese JA, et al. A comparison of thrombolytic therapy with operative revascularization in the initial treatment of acute peripheral arterial ischemia. J Vasc Surg 1994;19:1021-30.
560. Results of a prospective randomized trial evaluating surgery versus thrombolysis for ischemia of the lower extremity. The STILE trial. Ann Surg 1994;220:251-66; discussion 266-8.
561. Weaver FA, Comerota AJ, Youngblood M, et al. Surgical revascularization versus thrombolysis for nonembolic lower extremity native artery occlusions: results of a prospective randomized trial. The STILE Investigators. Surgery versus Thrombolysis for Ischemia of the Lower Extremity. J Vasc Surg 1996;24:513-21; discussion 521-3.
562. Ouriel K, Veith FJ, Sasahara AA. A comparison of recombinant urokinase with vascular surgery as initial treatment for acute arterial occlusion of the legs. Thrombolysis or Peripheral Arterial Surgery (TOPAS) Investigators. N Engl J Med 1998;338:1105-11.
563. Diffin DC, Kandarpa K. Assessment of peripheral intraarterial thrombolysis versus surgical revascularization in acute lower-limb ischemia: a review of limb-salvage and mortality statistics. J Vasc Interv Radiol 1996;7:57-63.
564. van Breda A, Katzen BT, Deutsch AS. Urokinase versus streptokinase in local thrombolysis. Radiology 1987;165:109-11.
565. Traughber PD, Cook PS, Micklos TJ, et al. Intraarterial fibrinolytic therapy for popliteal and tibial artery obstruction: comparison of streptokinase and urokinase. AJR Am J Roentgenol 1987;149:453-6.
566. Arepally A, Hofmann LV, Kim HS, et al. Weight-based rt-PA thrombolysis protocol for acute native arterial and bypass graft occlusions. J Vasc Interv Radiol 2002;13:45-50.
567. Swischuk JL, Fox PF, Young K, et al. Transcatheter intraarterial infusion of rt-PA for acute lower limb ischemia: results and complications. J Vasc Interv Radiol 2001;12:423-30.
568. Semba CP, Murphy TP, Bakal CW, et al. Thrombolytic therapy with use of alteplase (rt-PA) in peripheral arterial occlusive disease: review of the clinical literature. The Advisory Panel. J Vasc Interv Radiol 2000;11(2 pt 1):149-61.
569. Shortell CK, Queiroz R, Johansson M, et al. Safety and efficacy of limited-dose tissue plasminogen activator in acute vascular occlusion. J Vasc Surg 2001;34:854-9.
570. Braithwaite BD, Buckenham TM, Galland RB, et al. Prospective randomized trial of high-dose bolus versus low-dose tissue plasminogen activator infusion in the management of acute limb ischaemia. Thrombolysis Study Group. Br J Surg 1997;84:646-50.
571. Ouriel K, Gray B, Clair DG, et al. Complications associated with the use of urokinase and recombinant tissue plasminogen activator for catheter-directed peripheral arterial and venous thrombolysis. J Vasc Interv Radiol 2000;11:295-8.
572. Schweizer J, Altmann E, Stosslein F, et al. Comparison of tissue plasminogen activator and urokinase in the local infiltration thrombolysis of peripheral arterial occlusions. Eur J Radiol 1996;22:129-32.
573. Ouriel K, Katzen B, Mewissen M, et al. Reteplase in the treatment of peripheral arterial and venous occlusions: a pilot study. J Vasc Interv Radiol 2000;11:849-54.
574. Davidian MM, Powell A, Benenati JF, et al. Initial results of reteplase in the treatment of acute lower extremity arterial occlusions. J Vasc Interv Radiol 2000;11:289-94.
575. Castaneda F, Swischuk JL, Li R, et al. Declining-dose study of reteplase treatment for lower extremity arterial occlusions. J Vasc Interv Radiol 2002;13:1093-8.
576. Burkart DJ, Borsa JJ, Anthony JP, et al. Thrombolysis of occluded peripheral arteries and veins with tenecteplase: a pilot study. J Vasc Interv Radiol 2002;13:1099-102.
577. Meyerovitz MF, Goldhaber SZ, Reagan K, et al. Recombinant tissue-type plasminogen activator versus urokinase in peripheral arterial and graft occlusions: a randomized trial. Radiology 1990;175:75-8.
578. Cina CS, Goh RH, Chan J, et al. Intraarterial catheter-directed thrombolysis: urokinase versus tissue plasminogen activator. Ann Vasc Surg 1999;13:571-5.
579. Sugimoto K, Hofmann LV, Razavi MK, et al. The safety, efficacy, and pharmacoeconomics of low-dose alteplase compared with urokinase for catheter-directed thrombolysis of arterial and venous occlusions. J Vasc Surg 2003;37:512-7.
580. Drescher P, McGuckin J, Rilling WS, et al. Catheter-directed thrombolytic therapy in peripheral artery occlusions: combining reteplase and abciximab. AJR Am J Roentgenol 2003;180:1385-91.
581. Burkart DJ, Borsa JJ, Anthony JP, et al. Thrombolysis of acute peripheral arterial and venous occlusions with tenecteplase and eptifibatide: a pilot study. J Vasc Interv Radiol 2003;14:729-33.
582. Duda SH, Tepe G, Luz O, et al. Peripheral artery occlusion: treatment with abciximab plus urokinase versus with urokinase alone—a randomized pilot trial (the PROMPT Study). Platelet Receptor Antibodies in Order to Manage Peripheral Artery

Thrombosis. Radiology 2001;221:689-96.
583. Yoon HC, Miller FJ Jr. Using a peptide inhibitor of the glycoprotein IIb/IIIa platelet receptor: initial experience in patients with acute peripheral arterial occlusions. AJR Am J Roentgenol 2002;178:617-22.
584. Silva JA, Ramee SR, Collins TJ, et al. Rheolytic thrombectomy in the treatment of acute limb-threatening ischemia: immediate results and six-month follow-up of the multicenter AngioJet registry. Possis Peripheral AngioJet Study AngioJet Investigators. Cathet Cardiovasc Diagn 1998;45:386-93.
585. Kasirajan K, Haskal ZJ, Ouriel K. The use of mechanical thrombectomy devices in the management of acute peripheral arterial occlusive disease. J Vasc Interv Radiol 2001;12:405-11.
586. Hopfner W, Vicol C, Bohndorf K, et al. Shredding embolectomy thrombectomy catheter for treatment of acute lower-limb ischemia. Ann Vasc Surg 1999;13:426-35.
587. Muller-Hulsbeck S, Kalinowski M, Heller M, et al. Rheolytic hydrodynamic thrombectomy for percutaneous treatment of acutely occluded infra-aortic native arteries and bypass grafts: midterm follow-up results. Invest Radiol 2000;35:131-40.
588. Kasirajan K, Gray B, Beavers FP, et al. Rheolytic thrombectomy in the management of acute and subacute limb-threatening ischemia. J Vasc Interv Radiol 2001;12:413-21.
589. Wagner HJ, Muller-Hulsbeck S, Pitton MB, et al. Rapid thrombectomy with a hydrodynamic catheter: results from a prospective, multicenter trial. Radiology 1997;205:675-81.
590. Reekers JA, Kromhout JG, Spithoven HG, et al. Arterial thrombosis below the inguinal ligament: percutaneous treatment with a thrombosuction catheter. Radiology 1996;198:49-53.
591. Henry M, Amor M, Henry I, et al. The Hydrolyser thrombectomy catheter: a single-center experience. J Endovasc Surg 1998;5:24-31.
592. Rilinger N, Gorich J, Scharrer-Pamler R, et al. Short-term results with use of the Amplatz thrombectomy device in the treatment of acute lower limb occlusions. J Vasc Interv Radiol 1997;8:343-8.
593. Tadavarthy SM, Murray PD, Inampudi S, et al. Mechanical thrombectomy with the Amplatz device: human experience. J Vasc Interv Radiol 1994;5:715-24.
594. Gorich J, Rilinger N, Sokiranski R, et al. Mechanical thrombolysis of acute occlusion of both the superficial and the deep femoral arteries using a thrombectomy device. AJR Am J Roentgenol 1998;170:1177-80.
594a. Haskal ZJ. Mechanical thrombectomy devices for the treatment of peripheral arterial occlusions. Rev Cardiovasc Med 2002;3 Suppl 2:S45-S52.
595. Schneider JR, Besso SR, Walsh DB, et al. Femorofemoral versus aortobifemoral bypass: outcome and hemodynamic results. J Vasc Surg 1994;19:43-55; discussion 55-7.
596. Hobson RW 2nd, Lynch TG, Jamil Z, et al. Results of revascularization and amputation in severe lower extremity ischemia: a five-year clinical experience. J Vasc Surg 1985;2:174-85.
597. Schina MJ Jr, Atnip RG, Healy DA, et al. Relative risks of limb revascularization and amputation in the modern era. Cardiovasc Surg 1994;2:754-9.
598. Dawson I, Keller BP, Brand R, et al. Late outcomes of limb loss after failed infrainguinal bypass. J Vasc Surg 1995;21:613-22.
599. Moller BN, Solund K, Hansen SL. Wound infection after lower extremity amputation because of ischemia. Arch Orthop Trauma Surg 1985;104:262-4.
600. Cutson TM, Bongiorni DR. Rehabilitation of the older lower limb amputee: a brief review. J Am Geriatr Soc 1996;44:1388-93.
601. Propanolol Aneurysm Trial Investigators. Propranolol for small abdominal aortic aneurysms: results of a randomized trial. J Vasc Surg 2002;35:72-9.
602. Kalman PG, Hosang M, Johnston KW, et al. Unilateral iliac disease: the role of iliofemoral bypass. J Vasc Surg 1987;6:139-43.
603. Ng RL, Gillies TE, Davies AH, et al. Iliofemoral versus femorofemoral bypass: a 6-year audit. Br J Surg 1992;79:1011-3.
604. Naylor AR, Ah-See AK, Engeset J. Axillofemoral bypass as a limb salvage procedure in high risk patients with aortoiliac disease. Br J Surg 1990;77:659-61.
605. Ascer E, Veith FJ, Gupta SK, et al. Comparison of axillounifemoral and axillobifemoral bypass operations. Surgery 1985;97:169-75.
606. Shah DM, Darling RC 3rd, Chang BB, et al. Is long vein bypass from groin to ankle a durable procedure? An analysis of a ten-year experience. J Vasc Surg 1992;15:402-7; discussion 407-8.
607. Pomposelli FB Jr, Marcaccio EJ, Gibbons GW, et al. Dorsalis pedis arterial bypass: durable limb salvage for foot ischemia in patients with diabetes mellitus. J Vasc Surg 1995;21:375-84.
608. Roddy SP, Darling RC 3rd, Ozsvath KJ, et al. Composite sequential arterial reconstruction for limb salvage. J Vasc Surg 2002;36:325-9.
609. Johnson WC, Williford WO; Department of Veterans Affairs Cooperative Study #362. Benefits, morbidity, and mortality associated with long-term administration of oral anticoagulant therapy to patients with peripheral arterial bypass procedures: a prospective randomized study. J Vasc Surg 2002;35:413-21.
610. Hamdan AD, Rayan SS, Hook SC, et al. Bypasses to tibial vessels using polytetrafluoroethylene as the solo conduit in a predominantly diabetic population. Vasc Endovascular Surg 2002;36:59-63.
611. Henke PK, Blackburn S, Proctor MC, et al. Patients undergoing infrainguinal bypass to treat atherosclerotic vascular disease are underprescribed cardioprotective medications: effect on graft patency, limb salvage, and mortality. J Vasc Surg 2004;39:357-65.
612. Holley KE, Hunt JC, Brown AL Jr, et al. Renal artery stenosis: a clinical-pathologic study in normotensive and hypertensive patients. Am J Med 1964;37:14-22.
613. Dustan HP, Humphries AW, Dewolfe VG, et al. Normal arterial pressure in patients with renal arterial stenosis. JAMA 1964;187:1028-9.
614. Scoble JE. The epidemiology and clinical manifestations of atherosclerotic renal disease. In: Novick AC, Scoble JE, Hamilton G, eds. Renal Vascular Disease. London, UK: WB Saunders Co, Ltd; 1996:303-14.
615. Uzu T, Inoue T, Fujii T, et al. Prevalence and predictors of renal artery stenosis in patients with myocardial infarction. Am J Kidney Dis 1997;29:733-8.
616. Wilms G, Marchal G, Peene P, et al. The angiographic incidence of renal artery stenosis in the arteriosclerotic population. Eur J Radiol 1990;10:195-7.
617. Choudhri AH, Cleland JG, Rowlands PC, et al. Unsuspected renal artery stenosis in peripheral vascular disease. BMJ 1990; 301:1197-8.
618. Swartbol P, Thorvinger BO, Parsson H, et al. Renal artery stenosis in patients with peripheral vascular disease and its correlation to hypertension: a retrospective study. Int Angiol 1992;11:195-9.
619. Missouris CG, Papavassiliou MB, Khaw K, et al. High prevalence of carotid artery disease in patients with atheromatous renal artery stenosis. Nephrol Dial Transplant 1998;13:945-8.
620. Hansen KJ, Edwards MS, Craven TE, et al. Prevalence of renovascular disease in the elderly: a population-based study. J Vasc Surg 2002;36:443-51.
621. Harding MB, Smith LR, Himmelstein SI, et al. Renal artery stenosis: prevalence and associated risk factors in patients undergoing

routine cardiac catheterization. J Am Soc Nephrol 1992;2:1608-16.
622. Weber-Mzell D, Kotanko P, Schumacher M, et al. Coronary anatomy predicts presence or absence of renal artery stenosis: a prospective study in patients undergoing cardiac catheterization for suspected coronary artery disease. Eur Heart J 2002;23:1684-91.
623. Jean WJ, al-Bitar I, Zwicke DL, et al. High incidence of renal artery stenosis in patients with coronary artery disease. Cathet Cardiovasc Diagn 1994;32:8-10.
624. Missouris CG, Buckenham T, Cappuccio FP, et al. Renal artery stenosis: a common and important problem in patients with peripheral vascular disease. Am J Med 1994;96:10-4.
625. Olin JW, Melia M, Young JR, et al. Prevalence of atherosclerotic renal artery stenosis in patients with atherosclerosis elsewhere. Am J Med 1990;88(1N):46N-51N.
626. Louie J, Isaacson JA, Zierler RE, et al. Prevalence of carotid and lower extremity arterial disease in patients with renal artery stenosis. Am J Hypertens 1994;7:436-9.
627. Zierler RE, Bergelin RO, Polissar NL, et al. Carotid and lower extremity arterial disease in patients with renal artery atherosclerosis. Arch Intern Med 1998;158:761-7.
628. Rossi GP, Rossi A, Zanin L, et al. Excess prevalence of extracranial carotid artery lesions in renovascular hypertension. Am J Hypertens 1992;5:8-15.
629. Metcalfe W, Reid AW, Geddes CC. Prevalence of angiographic atherosclerotic renal artery disease and its relationship to the anatomical extent of peripheral vascular atherosclerosis. Nephrol Dial Transplant 1999;14:105-8.
630. Valentine RJ, Clagett GP, Miller GL, et al. The coronary risk of unsuspected renal artery stenosis. J Vasc Surg 1993;18:433-9; discussion 439-40.
631. Schwartz CJ, White TA. Stenosis of renal artery: an unselected necropsy study. Br Med J 1964;5422:1415-21.
632. Rimmer JM, Gennari FJ. Atherosclerotic renovascular disease and progressive renal failure. Ann Intern Med 1993;118:712-9.
633. Wollenweber J, Sheps SG, Davis GD. Clinical course of atherosclerotic renovascular disease. Am J Cardiol 1968;21:60-71.
634. Meaney TF, Dustan HP, McCormack LJ. Natural history of renal arterial disease. Radiology 1968;91:881-7.
635. Schreiber MJ, Pohl MA, Novick AC. The natural history of atherosclerotic and fibrous renal artery disease. Urol Clin North Am 1984;11:383-92.
636. Tollefson DF, Ernst CB. Natural history of atherosclerotic renal artery stenosis associated with aortic disease. J Vasc Surg 1991; 14:327-31.
637. Dean RH, Kieffer RW, Smith BM, et al. Renovascular hypertension: anatomic and renal function changes during drug therapy. Arch Surg 1981;116:1408-15.
638. Zierler RE, Bergelin RO, Davidson RC, et al. A prospective study of disease progression in patients with atherosclerotic renal artery stenosis. Am J Hypertens 1996;9:1055-61.
639. Caps MT, Perissinotto C, Zierler RE, et al. Prospective study of atherosclerotic disease progression in the renal artery. Circulation 1998;98:2866-72.
640. Mailloux LU, Napolitano B, Bellucci AG, et al. Renal vascular disease causing end-stage renal disease, incidence, clinical correlates, and outcomes: a 20-year clinical experience. Am J Kidney Dis 1994;24:622-9.
641. Guzman RP, Zierler RE, Isaacson JA, et al. Renal atrophy and arterial stenosis. A prospective study with duplex ultrasound. Hypertension 1994;23:346-50.
642. Caps MT, Zierler RE, Polissar NL, et al. Risk of atrophy in kidneys with atherosclerotic renal artery stenosis. Kidney Int 1998; 53:735-42.
643. Crowley JJ, Santos RM, Peter RH, et al. Progression of renal artery stenosis in patients undergoing cardiac catheterization. Am Heart J 1998;136:913-8.
644. Eggers PW, Connerton R, McMullan M. The Medicare experience with end-stage renal disease: trends in incidence, prevalence, and survival. Health Care Financ Rev 1984;5:69-88.
645. Mailloux LU, Bellucci AG, Mossey RT, et al. Predictors of survival in patients undergoing dialysis. Am J Med 1988;84:855-62.
646. Dorros G, Jaff M, Mathiak L, et al. Four-year follow-up of Palmaz-Schatz stent revascularization as treatment for atherosclerotic renal artery stenosis. Circulation 1998;98:642-7.
647. Wright JR, Shurrab AE, Cheung C, et al. A prospective study of the determinants of renal functional outcome and mortality in atherosclerotic renovascular disease. Am J Kidney Dis 2002; 39:1153-61.
648. Gifford RW Jr, McCormack LJ, Poutasse EF. The atrophic kidney: its role in hypertension. Mayo Clin Proc 1965;40:834-52.
649. Packer M, Lee WH, Medina N, et al. Functional renal insufficiency during long-term therapy with captopril and enalapril in severe chronic heart failure. Ann Intern Med 1987;106:346-54.
650. Textor SC. Renal failure related to angiotensin-converting enzyme inhibitors. Semin Nephrol 1997;17:67-76.
651. Watson ML, Bell GM, Muir AL, et al. Captopril/diuretic combinations in severe renovascular disease: a cautionary note. Lancet 1983;2:404-5.
652. Hricik DE, Browning PJ, Kopelman R, et al. Captopril-induced functional renal insufficiency in patients with bilateral renal-artery stenoses or renal-artery stenosis in a solitary kidney. N Engl J Med 1983;308:373-6.
652a. Bakris GL, Weir MR. Angiotensin converting enzyme inhibitor-associated elevations in serum creatinine. Arch Int Med 2000; 160:685-93.
653. Safian RD, Textor SC. Renal-artery stenosis. N Engl J Med 2001;344:431-42.
654. Luscher TF, Keller HM, Imhof HG, et al. Fibromuscular hyperplasia: extension of the disease and therapeutic outcome: results of the University Hospital Zurich Cooperative Study on Fibromuscular Hyperplasia. Nephron 1986;44 suppl 1:109-14.
655. Archibald GR, Beckmann CF, Libertino JA. Focal renal artery stenosis caused by fibromuscular dysplasia: treatment by percutaneous transluminal angioplasty. AJR Am J Roentgenol 1988;151:593-6.
656. Cluzel P, Raynaud A, Beyssen B, et al. Stenoses of renal branch arteries in fibromuscular dysplasia: results of percutaneous transluminal angioplasty. Radiology 1994;193:227-32.
657. Mounier-Vehier C, Haulon S, Devos P, et al. Renal atrophy outcome after revascularization in fibromuscular dysplasia disease. J Endovasc Ther 2002;9:605-13.
658. Stanley JC, Gewertz BL, Bove EL, et al. Arterial fibrodysplasia: histopathologic character and current etiologic concepts. Arch Surg 1975;110:561-6.
659. Stanley JC, Wakefield TW. Arterial fibrodysplasia. In: Rutherford RB, ed. Vascular Surgery 6th ed. Philadelphia, Pa: Saunders; 2004:387-408.
660. Messina LM, Stanley JC. Renal artery fibrodysplasia and renovascular hypertension. In: Rutherford RB, ed. Vascular Surgery 6th ed. Philadelphia, Pa: Saunders; 2004:1650-64.
661. Mettinger KL. Fibromuscular dysplasia and the brain. II. Current concept of the disease. Stroke 1982;13:53-8.
662. Cloft HJ, Kallmes DF, Kallmes MH, et al. Prevalence of cerebral aneurysms in patients with fibromuscular dysplasia: a reassess-

ment. J Neurosurg 1998;88:436-40.
663. Abud O, Chechile GE, Sole-Balcells F. Aneurysm and arteriovenous malformation. In: Novick AC, Scoble JE, Hamilton G, eds. Renal Vascular Disease. London, UK: WB Saunders Co, Ltd; 1996:35-46.
664. Novick AC. Renal artery aneurysms and arteriovenous malformation. In: Novick AC, Straffon RA, eds. Vascular Problems in Urologic Surgery Philadelphia, Pa: WB Saunders; 1982:189-204.
665. Poutasse EF. Renal artery aneurysms. J Urol 1975;113:443-9.
666. McCready RA, Hyde GL, Bivins BA, et al. Radiation-induced arterial injuries. Surgery 1983;93:306-12.
667. Guthaner DF, Schmitz L. Percutaneous transluminal angioplasty of radiation-induced arterial stenoses. Radiology 1982;144:77-8.
668. Andros G, Schneider PA, Harris RW, et al. Management of arterial occlusive disease following radiation therapy. Cardiovasc Surg 1996;4:135-42.
669. Ingelfinger JR, Newburger JW. Spectrum of renal anomalies in patients with Williams syndrome. J Pediatr 1991;119:771-3.
670. Booth C, Preston R, Clark G, et al. Management of renal vascular disease in neurofibromatosis type 1 and the role of percutaneous transluminal angioplasty. Nephrol Dial Transplant 2002;17:1235-40.
671. Courtel JV, Soto B, Niaudet P, et al. Percutaneous transluminal angioplasty of renal artery stenosis in children. Pediatr Radiol 1998;28:59-63.
672. Tyagi S, Singh B, Kaul UA, et al. Balloon angioplasty for renovascular hypertension in Takayasu's arteritis. Am Heart J 1993; 125(5 pt 1):1386-93.
673. Teoh MK. Takayasu's arteritis with renovascular hypertension: results of surgical treatment. Cardiovasc Surg 1999;7:626-32.
674. Prigent A, Cosgriff P, Gates GF, et al. Consensus report on quality control of quantitative measurements of renal function obtained from the renogram: International Consensus Committee from the Scientific Committee of Radionuclides in Nephrourology. Semin Nucl Med 1999;29:146-59.
675. Nally JV Jr, Clarke HS Jr, Grecos GP, et al. Effect of captopril on 99mTc-diethylenetriaminepentaacetic acid renograms in two-kidney, one clip hypertension. Hypertension 1986;8:685-93.
676. Setaro JF, Saddler MC, Chen CC, et al. Simplified captopril renography in diagnosis and treatment of renal artery stenosis. Hypertension 1991;18:289-98.
677. Dondi M. Captopril renal scintigraphy with 99mTc-mercaptoacetyltriglycine (99mTc-MAG3) for detecting renal artery stenosis. Am J Hypertens 1991;4(12 pt 2):737S-740S.
678. Fommei E, Ghione S, Hilson AJ, et al. Captopril radionuclide test in renovascular hypertension: a European multicentre study. European Multicentre Study Group. Eur J Nucl Med 1993;20: 617-23.
679. Geyskes GG, Oei HY, Puylaert CB, et al. Renography with captopril. Changes in a patient with hypertension and unilateral renal artery stenosis. Arch Intern Med 1986;146:1705-8.
680. Sfakianakis GN, Bourgoignie JJ, Jaffe D, et al. Single-dose captopril scintigraphy in the diagnosis of renovascular hypertension. J Nucl Med 1987;28:1383-92.
681. Erbsloh-Moller B, Dumas A, Roth D, et al. Furosemide-131I-hippuran renography after angiotensin-converting enzyme inhibition for the diagnosis of renovascular hypertension. Am J Med 1991;90:23-9.
682. Mann SJ, Pickering TG, Sos TA, et al. Captopril renography in the diagnosis of renal artery stenosis: accuracy and limitations. Am J Med 1991;90:30-40.
683. Elliott WJ, Martin WB, Murphy MB. Comparison of two noninvasive screening tests for renovascular hypertension. Arch Intern Med 1993;153:755-64.
684. van Jaarsveld BC, Krijnen P, Derkx FH, et al. The place of renal scintigraphy in the diagnosis of renal artery stenosis: fifteen years of clinical experience. Arch Intern Med 1997;157:1226-34.
685. Mittal BR, Kumar P, Arora P, et al. Role of captopril renography in the diagnosis of renovascular hypertension. Am J Kidney Dis 1996;28:209-13.
686. Huot SJ, Hansson JH, Dey H, et al. Utility of captopril renal scans for detecting renal artery stenosis. Arch Intern Med 2002; 162:1981-4.
687. Carman TL, Olin JW. Diagnosis of renal artery stenosis: what is the optimal diagnostic test? Curr Interv Cardiol Rep 2000;2:111-8
688. Olin JW. Role of duplex ultrasonography in screening for significant renal artery disease. Urol Clin North Am 1994;21:215-26.
689. Hoffmann U, Edwards JM, Carter S, et al. Role of duplex scanning for the detection of atherosclerotic renal artery disease. Kidney Int 1991;39:1232-9.
690. Kohler TR, Zierler RE, Martin RL, et al. Noninvasive diagnosis of renal artery stenosis by ultrasonic duplex scanning. J Vasc Surg 1986;4:450-6.
691. Taylor DC, Kettler MD, Moneta GL, et al. Duplex ultrasound scanning in the diagnosis of renal artery stenosis: a prospective evaluation. J Vasc Surg 1988;7:363-9.
692. Wilcox CS. Ischemic nephropathy: noninvasive testing. Semin Nephrol 1996;16:43-52.
693. Carman TL, Olin JW, Czum J. Noninvasive imaging of the renal arteries. Urol Clin North Am 2001;28:815-26.
694. Olin JW, Piedmonte MR, Young JR, et al. The utility of duplex ultrasound scanning of the renal arteries for diagnosing significant renal artery stenosis. Ann Intern Med 1995;122:833-8.
695. Hudspeth DA, Hansen KJ, Reavis SW, et al. Renal duplex sonography after treatment of renovascular disease. J Vasc Surg 1993; 18:381-8; discussion 389-90.
696. Hansen KJ, Tribble RW, Reavis SW, et al. Renal duplex sonography: evaluation of clinical utility. J Vasc Surg 1990;12:227-36.
697. Kim SH, Kim WH, Choi BI, et al. Duplex Doppler US in patients with medical renal disease: resistive index vs serum creatinine level. Clin Radiol 1992;45:85-7.
698. Radermacher J, Chavan A, Bleck J, et al. Use of Doppler ultrasonography to predict the outcome of therapy for renal-artery stenosis. N Engl J Med 2001;344:410-7.
699. Leertouwer TC, Gussenhoven EJ, Bosch JL, et al. Stent placement for renal arterial stenosis: where do we stand? A meta-analysis. Radiology 2000;216:78-85.
700. van de Ven PJ, Kaatee R, Beutler JJ, et al. Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: a randomised trial. Lancet 1999;353:282-6.
701. Leertouwer TC, Derkx FH, Pattynama PM, et al. Functional effects of renal artery stent placement on treated and contralateral kidneys. Kidney Int 2002;62:574-9.
702. Zeller T, Muller C, Frank U, et al. Stent angioplasty of severe atherosclerotic ostial renal artery stenosis in patients with diabetes mellitus and nephrosclerosis. Catheter Cardiovasc Interv 2003; 58:510-5.
703. Beregi JP, Elkohen M, Deklunder G, et al. Helical CT angiography compared with arteriography in the detection of renal artery stenosis. AJR Am J Roentgenol 1996;167:495-501.
704. Halpern EJ, Rutter CM, Gardiner GA Jr, et al. Comparison of Doppler US and CT angiography for evaluation of renal artery stenosis. Acad Radiol 1998;5:524-32.
705. Johnson PT, Halpern EJ, Kuszyk BS, et al. Renal artery stenosis: CT angiography—comparison of real-time volume-rendering and

maximum intensity projection algorithms. Radiology 1999; 211:337-43.
706. Kim TS, Chung JW, Park JH, et al. Renal artery evaluation: comparison of spiral CT angiography to intra-arterial DSA. J Vasc Interv Radiol 1998;9:553-9.
707. Rubin GD, Dake MD, Napel S, et al. Spiral CT of renal artery stenosis: comparison of three-dimensional rendering techniques. Radiology 1994;190:181-9.
708. Kawashima A, Sandler CM, Ernst RD, et al. CT evaluation of renovascular disease. Radiographics 2000;20:1321-40.
709. Lufft V, Hoogestraat-Lufft L, Fels LM, et al. Contrast media nephropathy: intravenous CT angiography versus intraarterial digital subtraction angiography in renal artery stenosis: a prospective randomized trial. Am J Kidney Dis 2002;40:236-42.
710. Saloner D. Determinants of image appearance in contrast-enhanced magnetic resonance angiography: a review. Invest Radiol 1998;33:488-95.
711. Leung DA, Hoffmann U, Pfammatter T, et al. Magnetic resonance angiography versus duplex sonography for diagnosing renovascular disease. Hypertension 1999;33:726-31.
712. Loubeyre P, Trolliet P, Cahen R, et al. MR angiography of renal artery stenosis: value of the combination of three-dimensional time-of-flight and three-dimensional phase-contrast MR angiography sequences. AJR Am J Roentgenol 1996;167:489-94.
713. Prince MR, Schoenberg SO, Ward JS, et al. Hemodynamically significant atherosclerotic renal artery stenosis: MR angiographic features. Radiology 1997;205:128-36.
714. Hany TF, Debatin JF, Leung DA, et al. Evaluation of the aortoiliac and renal arteries: comparison of breath-hold, contrast-enhanced, three-dimensional MR angiography with conventional catheter angiography. Radiology 1997;204:357-62.
715. Sueyoshi E, Sakamoto I, Matsuoka Y, et al. Aortoiliac and lower extremity arteries: comparison of three-dimensional dynamic contrast-enhanced subtraction MR angiography and conventional angiography. Radiology 1999;210:683-8.
716. Tan KT, van Beek EJ, Brown PW, et al. Magnetic resonance angiography for the diagnosis of renal artery stenosis: a meta-analysis. Clin Radiol 2002;57:617-24.
717. Mitsuzaki K, Yamashita Y, Sakaguchi T, et al. Abdomen, pelvis, and extremities: diagnostic accuracy of dynamic contrast-enhanced turbo MR angiography compared with conventional angiography-initial experience. Radiology 2000;216:909-15.
717a. Rihal, CS, Textor SC, Grill DE, et al. Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation 2002;105:2259-64.
717b. Parfrey PS, Griffiths SM, Barrett BJ, et al. Contrast material-induced renal failure in patients with diabetes mellitus, renal insufficiency, or both. A prospective controlled study. N Engl J Med 1989;320:143-9.
718. Rundback JH, Sacks D, Kent KC, et al. Guidelines for the reporting of renal artery revascularization in clinical trials. American Heart Association. Circulation 2002;106:1572-85.
719. Leertouwer TC, Pattynama PM, van den Berg-Huysmans A. Incidental renal artery stenosis in peripheral vascular disease: a case for treatment? Kidney Int 2001;59:1480-3.
720. Rihal CS, Textor SC, Breen JF, et al. Incidental renal artery stenosis among a prospective cohort of hypertensive patients undergoing coronary angiography. Mayo Clin Proc 2002;77:309-16.
721. Rossi GP, Cesari M, Chiesura-Corona M, et al. Renal vein renin measurements accurately identify renovascular hypertension caused by total occlusion of the renal artery. J Hypertens 2002;20:975-84.
722. Hughes JS, Dove HG, Gifford RW Jr, et al. Duration of blood pressure elevation in accurately predicting surgical cure of renovascular hypertension. Am Heart J 1981;101:408-13.
723. Maxwell MH, Rudnick MR, Waks AU. New approaches to the diagnosis of renovascular hypertension. Adv Nephrol Necker Hosp 1985;14:285-304.
724. Plouin PF, Chatellier G, Darne B, et al. Blood pressure outcome of angioplasty in atherosclerotic renal artery stenosis: a randomized trial. Essai Multicentrique Medicaments vs Angioplastie (EMMA) Study Group. Hypertension 1998;31:823-9.
725. Webster J, Marshall F, Abdalla M, et al. Randomised comparison of percutaneous angioplasty vs continued medical therapy for hypertensive patients with atheromatous renal artery stenosis. Scottish and Newcastle Renal Artery Stenosis Collaborative Group. J Hum Hypertens 1998;12:329-35.
726. Nordmann AJ, Woo K, Parkes R, et al. Balloon angioplasty or medical therapy for hypertensive patients with atherosclerotic renal artery stenosis? A meta-analysis of randomized controlled trials. Am J Med 2003;114:44-50.
727. Plouin PF. Stable patients with atherosclerotic renal artery stenosis should be treated first with medical management. Am J Kidney Dis 2003;42:851-7.
728. Hollenberg NK. Medical therapy of renovascular hypertension: efficacy and safety of captopril in 269 patients. Cardiovasc Rev Repl 1983;4:852-76.
729. Vetrovec GW, Landwehr DM, Edwards VL. Incidence of renal artery stenosis in hypertensive patients undergoing coronary angiography. J Interv Cardiol 1989;2:69-76.
730. Conlon PJ, Little MA, Pieper K, et al. Severity of renal vascular disease predicts mortality in patients undergoing coronary angiography. Kidney Int 2001;60:1490-7.
731. Weibull H, Bergqvist D, Bergentz SE, et al. Percutaneous transluminal renal angioplasty versus surgical reconstruction of atherosclerotic renal artery stenosis: a prospective randomized study. J Vasc Surg 1993;18:841-50; discussion 850-2.
732. van Jaarsveld BC, Krijnen P, Pieterman H, et al. The effect of balloon angioplasty on hypertension in atherosclerotic renal-artery stenosis. Dutch Renal Artery Stenosis Intervention Cooperative Study Group. N Engl J Med 2000;342:1007-14.
733. Blum U, Krumme B, Flugel P, et al. Treatment of ostial renal-artery stenoses with vascular endoprostheses after unsuccessful balloon angioplasty. N Engl J Med 1997;336:459-65.
734. Tuttle KR, Chouinard RF, Webber JT, et al. Treatment of atherosclerotic ostial renal artery stenosis with the intravascular stent. Am J Kidney Dis 1998;32:611-22.
735. Henry M, Amor M, Henry I, et al. Stents in the treatment of renal artery stenosis: long-term follow-up. J Endovasc Surg 1999;6:42-51.
736. Rocha-Singh KJ, Mishkel GJ, Katholi RE, et al. Clinical predictors of improved long-term blood pressure control after successful stenting of hypertensive patients with obstructive renal artery atherosclerosis. Catheter Cardiovasc Interv 1999;47:167-72.
737. Dorros G, Jaff M, Jain A, et al. Follow-up of primary Palmaz-Schatz stent placement for atherosclerotic renal artery stenosis. Am J Cardiol 1995;75:1051-5.
738. White CJ, Ramee SR, Collins TJ, et al. Renal artery stent placement: utility in lesions difficult to treat with balloon angioplasty. J Am Coll Cardiol 1997;30:1445-50.
739. Lederman RJ, Mendelsohn FO, Santos R, et al. Primary renal artery stenting: characteristics and outcomes after 363 procedures. Am Heart J 2001;142:314-23.
740. Harjai K, Khosla S, Shaw D, et al. Effect of gender on outcomes following renal artery stent placement for renovascular hyperten-

sion. Cathet Cardiovasc Diagn 1997;42:381-6.
741. Bloch MJ, Trost DA, Whitmer J, et al. Ostial renal artery stent placement in patients 75 years of age or older. Am J Hypertens 2001;14:983-8.
742. Tuttle KR. Ischemic nephropathy. Curr Opin Nephrol Hypertens 2001;10:167-73.
743. Tuttle KR. Toward more rational management of ischemic nephropathy: the need for clinical evidence. Am J Kidney Dis 2000;36:863-5.
744. Airoldi F, Palatresi S, Marana I, et al. Angioplasty of atherosclerotic and fibromuscular renal artery stenosis: time course and predicting factors of the effects on renal function. Am J Hypertens 2000;13:1210-7.
745. Losinno F, Zuccala A, Busato F, et al. Renal artery angioplasty for renovascular hypertension and preservation of renal function: long-term angiographic and clinical follow-up. AJR Am J Roentgenol 1994;162:853-7.
746. Geroulakos G, Abel P. Effect of renal-artery stenting on progression of renovascular renal failure. Lancet 1997;349(9068):1840.
747. Dorros G, Jaff M, Mathiak L, et al. Multicenter Palmaz stent renal artery stenosis revascularization registry report: four-year follow-up of 1,058 successful patients. Catheter Cardiovasc Interv 2002;55:182-8.
748. Ying CY, Tifft CP, Gavras H, et al. Renal revascularization in the azotemic hypertensive patient resistant to therapy. N Engl J Med 1984;311:1070-5.
749. Hirshberg B, Sasson T, Grinblat I, et al. Prolonged renal dysfunction secondary to renal-artery stenosis in the elderly—it is never too late. Nephrol Dial Transplant 1998;13:982-4.
750. Harden PN, MacLeod MJ, Rodger RS, et al. Effect of renal-artery stenting on progression of renovascular renal failure. Lancet 1997;349:1133-6.
751. Watson PS, Hadjipetrou P, Cox SV, et al. Effect of renal artery stenting on renal function and size in patients with atherosclerotic renovascular disease. Circulation 2000;102:1671-7.
752. Rocha-Singh KJ, Ahuja RK, Sung CH, et al. Long-term renal function preservation after renal artery stenting in patients with progressive ischemic nephropathy. Catheter Cardiovasc Interv 2002;57:135-41.
752a. Muray S, Martin M, Amoedo ML, et al. Rapid decline in renal function reflects reversibility and predicts the outcome after angioplasty in renal artery stenosis. Am J Kidney Dis 2002;39:60-6.
752b. Beutler JJ, Van Ampting JM, van de Ven PJ, et al. Long-term effects of arterial stenting on kidney function for patients with ostial atherosclerotic renal artery stenosis and renal insufficiency. J Am Soc Nephrol 2001;12: 1475-81.
753. Krishnamurthi V, Novick AC, Myles JL. Atheroembolic renal disease: effect on morbidity and survival after revascularization for atherosclerotic renal artery stenosis. J Urol 1999;161:1093-6.
754. Scolari F, Tardanico R, Zani R, et al. Cholesterol crystal embolism: a recognizable cause of renal disease. Am J Kidney Dis 2000;36:1089-109.
755. Henry M, Klonaris C, Henry I, et al. Protected renal stenting with the PercuSurge GuardWire device: a pilot study. J Endovasc Ther 2001;8:227-37.
756. Pickering TG, Herman L, Devereux RB, et al. Recurrent pulmonary oedema in hypertension due to bilateral renal artery stenosis: treatment by angioplasty or surgical revascularisation. Lancet 1988;2:551-2.
757. Messina LM, Zelenock GB, Yao KA, et al. Renal revascularization for recurrent pulmonary edema in patients with poorly controlled hypertension and renal insufficiency: a distinct subgroup of patients with arteriosclerotic renal artery occlusive disease. J Vasc Surg 1992;15:73-80; discussion 80-2.
758. Weatherford DA, Freeman MB, Regester RF, et al. Surgical management of flash pulmonary edema secondary to renovascular hypertension. Surg Gynecol Obstet 1997;174:160-3.
759. Mansoor S, Shah A, Scoble JE. 'Flash pulmonary oedema'—a diagnosis for both the cardiologist and the nephrologist? Nephrol Dial Transplant 2001;16:1311-3.
760. Planken II, Rietveld AP. Rapid onset pulmonary edema (flash edema) in renal artery stenosis. Neth J Med 1998;52:116-9.
761. Gray BH, Olin JW, Childs MB, et al. Clinical benefit of renal artery angioplasty with stenting for the control of recurrent and refractory congestive heart failure. Vasc Med 2002;7:275-9.
762. Azizi M, Lavergne T, Day M, et al. Renal artery stenosis and congestive heart failure. Lancet 1993;342(8866):302.
763. Missouris CG, Buckenham T, Vallance PJ, et al. Renal artery stenosis masquerading as congestive heart failure. Lancet 1993; 341:1521-2.
764. Tami LF, McElderry MW, al-Adli NM, et al. Renal artery stenosis presenting as crescendo angina pectoris. Cathet Cardiovasc Diagn 1995;35:252-6.
765. Gross CM, Kramer J, Waigand J, et al. Ostial renal artery stent placement for atherosclerotic renal artery stenosis in patients with coronary artery disease. Cathet Cardiovasc Diagn 1998;45:1-8.
766. Bloch MJ, Trost DW, Pickering TG, et al. Prevention of recurrent pulmonary edema in patients with bilateral renovascular disease through renal artery stent placement. Am J Hypertens 1999;12(1 pt 1):1-7.
767. Khosla S, White CJ, Collins TJ, et al. Effects of renal artery stent implantation in patients with renovascular hypertension presenting with unstable angina or congestive heart failure. Am J Cardiol 1997;80:363-6.
768. Tegtmeyer CJ, Selby JB, Hartwell GD, et al. Results and complications of angioplasty in fibromuscular disease. Circulation 1991;83(2 suppl):I155-61.
769. Brawn LA, Ramsay LE. Is "improvement" real with percutaneous transluminal angioplasty in the management of renovascular hypertension? Lancet 1987;2:1313-6.
770. Cicuto KP, McLean GK, Oleaga JA, et al. Renal artery stenosis: anatomic classification for percutaneous transluminal angioplasty. AJR Am J Roentgenol 1981;137:599-601.
771. Ramsay LE, Waller PC. Blood pressure response to percutaneous transluminal angioplasty for renovascular hypertension: an overview of published series. BMJ 1990;300:569-72.
772. Ives NJ, Wheatley K, Stowe RL, et al. Continuing uncertainty about the value of percutaneous revascularization in atherosclerotic renovascular disease: a meta-analysis of randomized trials. Nephrol Dial Transplant 2003;18:298-304.
773. Sos TA, Pickering TG, Sniderman K, et al. Percutaneous transluminal renal angioplasty in renovascular hypertension due to atheroma or fibromuscular dysplasia. N Engl J Med 1983; 309:274-9.
774. Libertino JA, Beckmann CF. Surgery and percutaneous angioplasty in the management of renovascular hypertension. Urol Clin North Am 1994;21:235-43.
775. Canzanello VJ, Millan VG, Spiegel JE, et al. Percutaneous transluminal renal angioplasty in management of atherosclerotic renovascular hypertension: results in 100 patients. Hypertension 1989;13:163-72.
776. Klinge J, Mali WP, Puijlaert CB, et al. Percutaneous transluminal renal angioplasty: initial and long-term results. Radiology 1989;171:501-6.

777. Plouin PF, Darne B, Chatellier G, et al. Restenosis after a first percutaneous transluminal renal angioplasty. Hypertension 1993;21:89-96.
778. Martin LG, Cork RD, Kaufman SL. Long-term results of angioplasty in 110 patients with renal artery stenosis. J Vasc Interv Radiol 1992;3:619-26.
779. Dorros G, Prince C, Mathiak L. Stenting of a renal artery stenosis achieves better relief of the obstructive lesion than balloon angioplasty. Cathet Cardiovasc Diagn 1993;29:191-8.
780. Isles CG, Robertson S, Hill D. Management of renovascular disease: a review of renal artery stenting in ten studies. QJM 1999;92:159-67.
781. Stanley JC. The evolution of surgery for renovascular occlusive disease. Cardiovasc Surg 1994;2:195-202.
782. Stanley JC. David M. Hume memorial lecture. Surgical treatment of renovascular hypertension. Surg Gynecol Obstet 1997;174: 102-10.
783. van Bockel JH, van Schilfgaarde R, Felthuis W, et al. Long-term results of in situ and extracorporeal surgery for renovascular hypertension caused by fibrodysplasia. J Vasc Surg 1987;6:355-64.
784. Stanley JC, Ernst CB, Fry WJ. Fate of 100 aortorenal vein grafts: characteristics of late graft expansion, aneurysmal dilatation, and stenosis. Surgery 1973;74:931-44.
785. Khauli RB, Novick AC, Ziegelbaum M. Splenorenal bypass in the treatment of renal artery stenosis: experience with sixty-nine cases. J Vasc Surg 1985;2:547-51.
786. Stanley JC, Fry WJ. Renovascular hypertension secondary to arterial fibrodysplasia in adults: criteria for operation and results of surgical therapy. Arch Surg 1975;110:922-8.
787. Novick AC, Straffon RA, Stewart BH, et al. Surgical treatment of renovascular hypertension in the pediatric patient. J Urol 1978; 119:794-9.
788. Berkowitz HD, O'Neill JA Jr. Renovascular hypertension in children. Surgical repair with special reference to the use of reinforced vein grafts. J Vasc Surg 1989;9:46-55.
789. Palmaz JC. The current status of vascular intervention in ischemic nephropathy. J Vasc Interv Radiol 1998;9:539-43.
790. Martin LG, Rees CR, O'Bryant T. Percutaneous angioplasty of the renal arteries. In: Rutherford RB, ed. Vascular Surgery 5th ed. Philadelphia, Pa: WB Saunders; 2000:1611-39.
791. Slonim SM, Dake MD. Radiographic evaluation and treatment of renovascular disease. In: Strandness DE Jr, VanBreda A, eds. Surgical & Interventional Therapy. New York: Churchill Livingstone; 1994:721-41.
792. Hansen KJ, Thomason RB, Craven TE, et al. Surgical management of dialysis-dependent ischemic nephropathy. J Vasc Surg 1995;21:197-209; discussion 209-11.
793. Hansen KJ, Starr SM, Sands RE, et al. Contemporary surgical management of renovascular disease. J Vasc Surg 1992;16:319-30; discussion 330-1.
794. Wong JM, Hansen KJ, Oskin TC, et al. Surgery after failed percutaneous renal artery angioplasty. J Vasc Surg 1999;30:468-82.
795. Cambria RP, Brewster DC, L'Italien G, et al. Simultaneous aortic and renal artery reconstruction: evolution of an eighteen-year experience. J Vasc Surg 1995;21:916-24; discussion 925.
796. Dougherty MJ, Hallett JW Jr, Naessens J, et al. Renal endarterectomy vs. bypass for combined aortic and renal reconstruction: is there a difference in clinical outcome? Ann Vasc Surg 1995;9:87-94.
797. Stoney RJ, Messina LM, Goldstone J, et al. Renal endarterectomy through the transected aorta: a new technique for combined aortorenal atherosclerosis—a preliminary report. J Vasc Surg 1989; 9:224-33. Erratum in: J Vasc Surg 1989;10:19.
798. Stanley JC, Whitehouse WM Jr, Zelenock GB, et al. Reoperation for complications of renal artery reconstructive surgery undertaken for treatment of renovascular hypertension. J Vasc Surg 1985;2:133-44.
799. Hansen KJ, Deitch JS, Oskin TC, et al. Renal artery repair: consequence of operative failures. Ann Surg 1998;227:678-89; discussion 689-90.
800. Whitehouse WM Jr, Kazmers A, Zelenock GB, et al. Chronic total renal artery occlusion: effects of treatment on secondary hypertension and renal function. Surgery 1981;89:753-63.
801. Oskin TC, Hansen KJ, Deitch JS, et al. Chronic renal artery occlusion: nephrectomy versus revascularization. J Vasc Surg 1999; 29:140-9.
802. Novick AC. Surgical correction of renovascular hypertension. Surg Clin North Am 1988;68:1007-25.
803. Cambria RP, Brewster DC, L'Italien GJ, et al. The durability of different reconstructive techniques for atherosclerotic renal artery disease. J Vasc Surg 1994;20:76-85; discussion 86-7.
804. Novick AC, Ziegelbaum M, Vidt DG, et al. Trends in surgical revascularization for renal artery disease: ten years' experience. JAMA 1987;257:498-501.
805. Libertino JA, Bosco PJ, Ying CY, et al. Renal revascularization to preserve and restore renal function. J Urol 1992;147:1485-7.
806. Clair DG, Belkin M, Whittemore AD, et al. Safety and efficacy of transaortic renal endarterectomy as an adjunct to aortic surgery. J Vasc Surg 1995;21:926-33; discussion 934.
807. Xue F, Bettmann MA, Langdon DR, et al. Outcome and cost comparison of percutaneous transluminal renal angioplasty, renal arterial stent placement, and renal arterial bypass grafting. Radiology 1999;212:378-84.
808. Ottinger LW, Austen WG. A study of 136 patients with mesenteric infarction. Surg Gynecol Obstet 1967;124:251-61.
809. Hertzer NR, Beven EG, Humphries AW. Acute intestinal ischemia. Am Surg 1978;44:744-9.
810. Bergan JJ. Recognition and treatment of intestinal ischemia. Surg Clin North Am 1967;47:109-26.
811. Krupski WC, Effeney DJ, Ehrenfeld WK. Spontaneous dissection of the superior mesenteric artery. J Vasc Surg 1985;2:731-4.
812. Wolf EA Jr, Sumner DS, Strandness DE Jr. Disease of the mesenteric circulation in patients with thromboangiitis obliterans. Vasc Surg 1972;6:218-23.
813. Slater H, Elliott DW. Primary mesenteric infarction. Surg Gynecol Obstet 1972;123:309-11.
814. Singh RP, Shah RC, Lee ST. Acute mesenteric vascular occlusion: a review of thirty-two patients. Surgery 1975;78:613-7.
815. Smith JS Jr, Patterson LT. Acute mesenteric infarction. Am Surg 1976;42:562-7.
816. Kairaluoma MI, Karkola P, Heikkinen D, et al. Mesenteric infarction. Surg Gynecol Obstet 1977;133:188-93.
817. Sachs SM, Morton JH, Schwartz SI. Acute mesenteric ischemia. Surgery 1982;92:646-53.
818. Levy PJ, Krausz MM, Manny J. Acute mesenteric ischemia: improved results—a retrospective analysis of ninety-two patients. Surgery 1990;107:372-80.
819. Bulkley GB, Zuidema GD, Hamilton SR, et al. Intraoperative determination of small intestinal viability following ischemic injury: a prospective, controlled trial of two adjuvant methods (Doppler and fluorescein) compared with standard clinical judgment. Ann Surg 1981;193:628-37.
820. Gallego AM, Ramirez P, Rodriguez JM, et al. Role of urokinase in the superior mesenteric artery embolism. Surgery 1996; 120:111-3.

821. McBride KD, Gaines PA. Thrombolysis of a partially occluding superior mesenteric artery thromboembolus by infusion of streptokinase. Cardiovasc Intervent Radiol 1994;17:164-6.
822. Schoenbaum SW, Pena C, Koenigsberg P, et al. Superior mesenteric artery embolism: treatment with intraarterial urokinase. J Vasc Interv Radiol 1992;3:485-90.
823. Boley SJ, Sprayregan S, Siegelman SS, et al. Initial results from an aggressive roentgenological and surgical approach to acute mesenteric ischemia. Surgery 1977;82:848-55.
824. Kawauchi M, Tada Y, Asano K, et al. Angiographic demonstration of mesenteric arterial changes in postcoarctectomy syndrome. Surgery 1985;98:602-4.
825. Gewertz BL, Zarins CK. Postoperative vasospasm after antegrade mesenteric revascularization: a report of three cases. J Vasc Surg 1991;14:382-5.
826. Siegelman SS, Sprayregen S, Boley SJ. Angiographic diagnosis of mesenteric arterial vasoconstriction. Radiology 1974;112:533-42.
827. Ende N. Infarction of the bowel in cardiac failure. N Engl J Med 1958;258:879-81.
828. Greene FL, Ariyan S, Stansel HC Jr. Mesenteric and peripheral vascular ischemia secondary to ergotism. Surgery 1977;81:176-9.
829. Nalbandian H, Sheth N, Dietrich R, et al. Intestinal ischemia caused by cocaine ingestion: report of two cases. Surgery 1985;97:374-6.
830. Cheatham JE Jr, Williams GR, Thompson WM, et al. Coarctation: a review of 80 children and adolescents. Surg Gynecol Obstet 1979;138:889-93.
831. Merhoff GC, Porter JM. Ergot intoxication: historical review and description of unusual clinical manifestations. Ann Surg 1974;180:773-9.
832. Fisher DF Jr, Fry WJ. Collateral mesenteric circulation. Surg Gynecol Obstet 1987;164:487-92.
833. Mikkelsen WP. Intestinal angina: its surgical significance. Surg Gynecol Obstet 1957;94:262-7; discussion, 267-9.
834. Buchardt Hansen HJ. Abdominal angina: results of arterial reconstruction in 12 patients. Acta Chir Scand 1976;142:319-25.
835. Hollier LH, Bernatz PE, Pairolero PC, et al. Surgical management of chronic intestinal ischemia: a reappraisal. Surgery 1981;90:940-6.
836. Johnston KW, Lindsay TF, Walker PM, et al. Mesenteric arterial bypass grafts: early and late results and suggested surgical approach for chronic and acute mesenteric ischemia. Surgery 1995;118:1-7.
837. Moneta GL, Yeager RA, Dalman R, et al. Duplex ultrasound criteria for diagnosis of splanchnic artery stenosis or occlusion. J Vasc Surg 1991;14:511-8; discussion 518-20.
838. Moneta GL, Lee RW, Yeager RA, et al. Mesenteric duplex scanning: a blinded prospective study. J Vasc Surg 1993;17:79-84; discussion 85-6.
839. Zwolak RM, Fillinger MF, Walsh DB, et al. Mesenteric and celiac duplex scanning: a validation study. J Vasc Surg 1998;27:1078-87; discussion 1088.
840. Gentile AT, Moneta GL, Lee RW, et al. Usefulness of fasting and postprandial duplex ultrasound examinations for predicting high-grade superior mesenteric artery stenosis. Surg Gynecol Obstet 1995;169:476-9.
841. Thomas JH, Blake K, Pierce GE, et al. The clinical course of asymptomatic mesenteric arterial stenosis. J Vasc Surg 1998; 27:840-4.
842. Connolly JE, Stemmer EA. Intestinal gangrene as the result of mesenteric arterial steal. Surg Gynecol Obstet 1973;126:197-204.
843. Furrer J, Gruntzig A, Kugelmeier J, et al. Treatment of abdominal angina with percutaneous dilatation of an arteria mesenterica superior stenosis: preliminary communication. Cardiovasc Intervent Radiol 1980;3:43-4.
844. Golden DA, Ring EJ, McLean GK, et al. Percutaneous transluminal angioplasty in the treatment of abdominal angina. AJR Am J Roentgenol 1982;139:247-9.
845. Odurny A, Sniderman KW, Colapinto RF. Intestinal angina: percutaneous transluminal angioplasty of the celiac and superior mesenteric arteries. Radiology 1988;167:59-62.
846. Roberts L Jr, Wertman DA Jr, Mills SR, et al. Transluminal angioplasty of the superior mesenteric artery: an alternative to surgical revascularization. AJR Am J Roentgenol 1983;141:1039-42.
847. Levy PJ, Haskell L, Gordon RL. Percutaneous transluminal angioplasty of splanchnic arteries: an alternative method to elective revascularisation in chronic visceral ischaemia. Eur J Radiol 1987;7:239-42.
848. McShane MD, Proctor A, Spencer P, et al. Mesenteric angioplasty for chronic intestinal ischaemia. Eur J Vasc Surg 1992;6:333-6.
849. Allen RC, Martin GH, Rees CR, et al. Mesenteric angioplasty in the treatment of chronic intestinal ischemia. J Vasc Surg 1996;24:415-21; discussion 421-3.
850. Kasirajan K, O'Hara PJ, Gray BH, et al. Chronic mesenteric ischemia: open surgery versus percutaneous angioplasty and stenting. J Vasc Surg 2001;33:63-71.
850a. Rose SC, Quigley TM, Raker EJ. Revascularization for chronic mesenteric ischemia: comparison off operative arterial bypass grafting and percutaneous transluminal angioplasty. J Vasc Interv Radiol 1995;6:339-49.
850b. Bowser AN. Revascularization for chronic mesenteric ischemia: comparison of endovascular and open surgical intervention (Government Rep. No. CI02-4). University of South Florida, Jan. 17, 2002.
851. Jimenez JG, Huber TS, Ozaki CK, et al. Durability of antegrade synthetic aortomesenteric bypass for chronic mesenteric ischemia. J Vasc Surg 2002;35:1078-84. Erratum in: J Vasc Surg 2002;36:548.
852. Park WM, Cherry KJ Jr, Chua HK, et al. Current results of open revascularization for chronic mesenteric ischemia: a standard for comparison. J Vasc Surg 2002;35:853-9.
853. Cunningham CG, Reilly LM, Rapp JH, et al. Chronic visceral ischemia. Three decades of progress. Ann Surg 1991;214:276-87; discussion 287-8.
854. Kieny R, Batellier J, Kretz JG. Aortic reimplantation of the superior mesenteric artery for atherosclerotic lesions of the visceral arteries: sixty cases. Ann Vasc Surg 1990;4:122-5.
855. Foley MI, Moneta GL, Abou-Zamzam AM Jr, et al. Revascularization of the superior mesenteric artery alone for treatment of intestinal ischemia. J Vasc Surg 2000;32:37-47.
856. Beebe HG, MacFarlane S, Raker EJ. Supraceliac aortomesenteric bypass for intestinal ischemia. J Vasc Surg 1987;5:749-54.
857. Rapp JH, Reilly LM, Qvarfordt PG, et al. Durability of endarterectomy and antegrade grafts in the treatment of chronic visceral ischemia. J Vasc Surg 1986;3:799-806.
858. Moawad J, McKinsey JF, Wyble CW, et al. Current results of surgical therapy for chronic mesenteric ischemia. Arch Surg 1997;132:613-8; discussion 618-9.
859. Ebaugh JL, Garcia ND, Matsumura JS. Screening and surveillance for abdominal aortic aneurysms: who needs it and when. Semin Vasc Surg 2001;14:193-9.
860. Alcorn HG, Wolfson SK Jr, Sutton-Tyrrell K, et al. Risk factors for abdominal aortic aneurysms in older adults enrolled in The Cardiovascular Health Study. Arterioscler Thromb Vasc Biol

1996;16:963-70.
861. Pedersen OM, Aslaksen A, Vik-Mo H. Ultrasound measurement of the luminal diameter of the abdominal aorta and iliac arteries in patients without vascular disease. J Vasc Surg 1993;17:596-601.
862. Lanne T, Sandgren T, Sonesson B. A dynamic view on the diameter of abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 1998;15:308-12.
863. Johnston KW, Rutherford RB, Tilson MD, et al. 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 J Vasc Surg 1991;13:452-8.
864. Pearce WH, Slaughter MS, LeMaire S, et al. Aortic diameter as a function of age, gender, and body surface area. Surgery 1993; 114:691-7.
865. Sandgren T, Sonesson B, Ahlgren AR, et al. Factors predicting the diameter of the popliteal artery in healthy humans. J Vasc Surg 1998;28:284-9.
866. Sonesson B, Lanne T, Hansen F, et al. Infrarenal aortic diameter in the healthy person. Eur J Vasc Surg 1994;8:89-95.
867. Lawrence-Brown MM, Norman PE, Jamrozik K, et al. Initial results of ultrasound screening for aneurysm of the abdominal aorta in Western Australia: relevance for endoluminal treatment of aneurysm disease. Cardiovasc Surg 2001;9:234-40.
868. Bengtsson H, Sonesson B, Bergqvist D. Incidence and prevalence of abdominal aortic aneurysms, estimated by necropsy studies and population screening by ultrasound. Ann N Y Acad Sci 1996;800:1-24.
869. Jamrozik K, Norman PE, Spencer CA, et al. Screening for abdominal aortic aneurysm: lessons from a population-based study. Med J Aust 2000;173:345-50.
870. Lederle FA, Johnson GR, Wilson SE, et al. The aneurysm detection and management study screening program: validation cohort and final results. Aneurysm Detection and Management Veterans Affairs Cooperative Study Investigators. Arch Intern Med 2000;160:1425-30.
871. Singh K, Bonaa KH, Jacobsen BK, et al. Prevalence of and risk factors for abdominal aortic aneurysms in a population-based study: the Tromso Study. Am J Epidemiol 2001;154:236-44.
872. Pleumeekers HJ, Hoes AW, van der Does E, et al. Aneurysms of the abdominal aorta in older adults. The Rotterdam Study. Am J Epidemiol 1995;142:1291-9.
873. Vazquez C, Sakalihasan N, D'Harcour JB, et al. Routine ultrasound screening for abdominal aortic aneurysm among 65- and 75-year-old men in a city of 200,000 inhabitants. Ann Vasc Surg 1998;12:544-9.
874. Boll AP, Verbeek AL, van de Lisdonk EH, et al. High prevalence of abdominal aortic aneurysm in a primary care screening programme. Br J Surg 1998;85:1090-4.
875. Wilmink AB, Quick CR. Epidemiology and potential for prevention of abdominal aortic aneurysm. Br J Surg 1998;85:155-62.
876. Takei H, Ishikawa S, Otaki A, et al. Screening for abdominal aortic aneurysm and occlusive peripheral vascular disease in Japanese residents. Surg Today 1995;25:608-11.
877. Adachi K, Iwasawa T, Ono T. Screening for abdominal aortic aneurysms during a basic medical checkup in residents of a Japanese rural community. Surg Today 2000;30:594-9.
878. Spark JI, Baker JL, Vowden P, et al. Epidemiology of abdominal aortic aneurysms in the Asian community. Br J Surg 2001;88:382-4.
879. Sandgren T, Sonesson B, Ryden-Ahlgren, Lanne T. Arterial dimensions in the lower extremities of patients with abdominal aortic aneurysms—no indications of a generalized dilating diathesis. J Vasc Surg 2001;34:1079-84.
880. Lawrence PF, Wallis C, Dobrin PB, et al. Peripheral aneurysms and arteriomegaly: is there a familial pattern? J Vasc Surg 1998;28:599-605.
881. Verloes A, Sakalihasan N, Koulischer L, et al. Aneurysms of the abdominal aorta: familial and genetic aspects in three hundred thirteen pedigrees. J Vasc Surg 1995;21:646-55.
882. Darling RC 3rd, Brewster DC, Darling RC, et al. Are familial abdominal aortic aneurysms different? J Vasc Surg 1989;10:39-43.
883. Webster MW, Ferrell RE, St Jean PL, et al. Ultrasound screening of first-degree relatives of patients with an abdominal aortic aneurysm. J Vasc Surg 1991;13:9-13; discussion 13-4.
884. Bengtsson H, Sonesson B, Lanne T, et al. Prevalence of abdominal aortic aneurysm in the offspring of patients dying from aneurysm rupture. Br J Surg 1992;79:1142-3.
885. Hirose H, Tilson MD. Abdominal aortic aneurysm as an autoimmune disease. Ann N Y Acad Sci 2001;947:416-8.
886. Lindholt JS, Jorgensen B, Fasting H, et al. Plasma levels of plasmin-antiplasmin-complexes are predictive for small abdominal aortic aneurysms expanding to operation-recommendable sizes. J Vasc Surg 2001;34:611-5.
887. Adams DC, Tulloh BR, Galloway SW, et al. Familial abdominal aortic aneurysm: prevalence and implications for screening. Eur J Vasc Surg 1993;7:709-12.
888. Fitzgerald P, Ramsbottom D, Burke P, et al. Abdominal aortic aneurysm in the Irish population: a familial screening study. Br J Surg 1995;82:483-6.
889. van der Graaf Y, Akkersdijk GJ, Hak E, et al. Results of aortic screening in the brothers of patients who had elective aortic aneurysm repair. Br J Surg 1998;85:778-80.
890. Jaakkola P, Kuivaniemi H, Partanen K, et al. Familial abdominal aortic aneurysms: screening of 71 families. Eur J Surg 1996;162:611-7.
891. Leier CV, Baker PB, Kilman JW, et al. Cardiovascular abnormalities associated with adult polycystic kidney disease. Ann Intern Med 1984;100:683-8.
892. Torra R, Nicolau C, Badenas C, et al. Abdominal aortic aneurysms and autosomal dominant polycystic kidney disease. J Am Soc Nephrol 1996;7:2483-6.
893. McConathy WJ, Alaupovic P, Woolcock N, et al. Lipids and apolipoprotein profiles in men with aneurysmal and stenosing aorto-iliac atherosclerosis. Eur J Vasc Surg 1989;3:511-4.
894. Schillinger M, Domanovits H, Ignatescu M, et al. Lipoprotein (a) in patients with aortic aneurysmal disease. J Vasc Surg 2002;36:25-30.
895. Simons PC, Algra A, Bots ML, et al. Common carotid intima-media thickness in patients with peripheral arterial disease or abdominal aortic aneurysm: the SMART study. Second Manifestations of ARTerial disease. Atherosclerosis 1999;146: 243-8.
896. Davies MJ. Aortic aneurysm formation: lessons from human studies and experimental models. Circulation 1998;98:193-5.
897. Goodall S, Porter KE, Bell PR, et al. Enhanced invasive properties exhibited by smooth muscle cells are associated with elevated production of MMP-2 in patients with aortic aneurysms. Eur J Vasc Endovasc Surg 2002;24:72-80.
898. Reilly JM, Brophy CM, Tilson MD. Characterization of an elastase from aneurysmal aorta which degrades intact aortic elastin. Ann Vasc Surg 1992;6:499-502.
899. Anidjar S, Dobrin PB, Eichorst M, et al. Correlation of inflammatory infiltrate with the enlargement of experimental aortic

aneurysms. J Vasc Surg 1992;16:139-47.
900. Pearce WH, Koch AE. Cellular components and features of immune response in abdominal aortic aneurysms. Ann N Y Acad Sci 1996;800:175-85.
901. Louwrens HD, Kwaan HC, Pearce WH, et al. Plasminogen activator and plasminogen activator inhibitor expression by normal and aneurysmal human aortic smooth muscle cells in culture. Eur J Vasc Endovasc Surg 1995;10:289-93.
902. Sakamaki F, Oya H, Nagaya N, et al. Higher prevalence of obstructive airway disease in patients with thoracic or abdominal aortic aneurysm. J Vasc Surg 2002;36:35-40.
903. Lindholt JS, Heickendorff L, Antonsen S, et al. Natural history of abdominal aortic aneurysm with and without coexisting chronic obstructive pulmonary disease. J Vasc Surg 1998;28:226-33.
904. Yajima N, Masuda M, Miyazaki M, et al. Oxidative stress is involved in the development of experimental abdominal aortic aneurysm: a study of the transcription profile with complementary DNA microarray. J Vasc Surg 2002;36:379-85.
905. Baxter BT, Pearce WH, Waltke EA, et al. Prolonged administration of doxycycline in patients with small asymptomatic abdominal aortic aneurysms: report of a prospective (Phase II) multicenter study. J Vasc Surg 2002;36:1-12.
906. Nagashima H, Aoka Y, Sakomura Y, et al. A 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor, cerivastatin, suppresses production of matrix metalloproteinase-9 in human abdominal aortic aneurysm wall. J Vasc Surg 2002;36:158-63.
907. Gsell O. Wandnekrosen der Aorta als selbständige Erkrankung und ihre Beziehung zur Spontanruptur. Virchows Arch Pathol Anat Physiol Klin Med 1928:1-36.
908. Erdheim J. Medionecrosis aortae idiopathica (cystica). Virchows Arch Pathol Anat Physiol Klin Med 1929:454-79.
909. Marfan AB. Un cas de déformation congénital des quatre membres plus prononcée aux extrémitiés caractérisée par l'allongement des os avec un certain degré d'amincissement. Bull Mém Soc Méd Hôp 1896;13:220-6.
910. Jondeau G, Delorme G, Guiti C. [Marfan syndrome] Rev Prat. 2002;52:1089-93.
911. Hollister DW, Godfrey M, Sakai LY, et al. Immunohistologic abnormalities of the microfibrillar-fiber system in the Marfan syndrome. N Engl J Med 1990;323:152-9.
912. Dietz HC, Cutting GR, Pyeritz RE, et al. Marfan syndrome caused by a recurrent de novo missense mutation in the fibrillin gene. Nature 1991;352:337-9.
913. Lee B, Godfrey M, Vitale E, et al. Linkage of Marfan syndrome and a phenotypically related disorder to two different fibrillin genes. Nature 1991;352:330-4.
914. Tsipouras P, Del Mastro R, Sarfarazi M, et al. Genetic linkage of the Marfan syndrome, ectopia lentis, and congenital contractural arachnodactyly to the fibrillin genes on chromosomes 15 and 5. The International Marfan Syndrome Collaborative Study. N Engl J Med 1992;326:905-9.
915. Francke U, Furthmayr H. Marfan's syndrome and other disorders of fibrillin. N Engl J Med 1994;330:1384-5.
916. Kainulainen K, Savolainen A, Palotie A, et al. Marfan syndrome: exclusion of genetic linkage to five genes coding for connective tissue components in the long arm of chromosome 2. Hum Genet 1990;84:233-6.
917. Maslen CL, Corson GM, Maddox BK, et al. Partial sequence of a candidate gene for the Marfan syndrome. Nature 1991;352:334-7.
918. Jeremy RW, Huang H, Hwa J, et al. Relation between age, arterial distensibility, and aortic dilatation in the Marfan syndrome. Am J Cardiol 1994;74:369-73.
919. Pepin M, Schwarze U, Superti-Furga A, et al. Clinical and genetic features of Ehlers-Danlos syndrome type IV, the vascular type. N Engl J Med 2000;342:673-80. Erratum in: N Engl J Med 2001;344:392.
920. Francke U, Berg MA, Tynan K, et al. A Gly1127Ser mutation in an EGF-like domain of the fibrillin-1 gene is a risk factor for ascending aortic aneurysm and dissection. Am J Hum Genet 1995;56:1287-96.
921. Rasmussen TE, Hallett JW Jr. Inflammatory aortic aneurysms: a clinical review with new perspectives in pathogenesis. Ann Surg 1997;225:155-64.
922. Bonamigo TP, Bianco C, Becker M, et al. Inflammatory aneurysms of infra-renal abdominal aorta: a case-control study. Minerva Cardioangiol 2002;50:253-8.
923. Cavallaro A, Sapienza P, di Marzo L, et al. [Inflammatory aneurysm of the abdominal aorta: study of 355 patients with aortic aneurysm] Recenti Prog Med 2001;92:269-73. Italian.
924. Pennell RC, Hollier LH, Lie JT, et al. Inflammatory abdominal aortic aneurysms: a thirty-year J Vasc Surg 1985;2:859-69.
925. Munshi IA, Rhee SW, Pane T, et al. Clostridium septicum mycotic aortic aneurysm. Surg Gynecol Obstet 2002;184:54-5.
926. Fiessinger JN, Paul JF. [Inflammatory and infectious aortitis] Rev Prat. 2002;52:1094-9.
927. Hagino RT, Clagett GP, Valentine RJ. A case of Pott's disease of the spine eroding into the suprarenal aorta. J Vasc Surg 1996;24:482-6.
928. Vammen S, Vorum H, Ostergaard L, et al. Immunoblotting analysis of abdominal aortic aneurysms using antibodies against Chlamydia pneumoniae recombinant MOMP. Eur J Vasc Endovasc Surg 2002;24:81-5.
929. Loehe F, Bittmann I, Weilbach C, et al. Chlamydia pneumoniae in atherosclerotic lesions of patients undergoing vascular surgery. Ann Vasc Surg 2002;16:467-73.
930. Vammen S, Lindholt JS, Ostergaard L, et al. Randomized double-blind controlled trial of roxithromycin for prevention of abdominal aortic aneurysm expansion. Br J Surg 2001;88:1066-72. Erratum in: Br J Surg 2002;89:120-1.
931. Bengtsson H, Nilsson P, Bergqvist D. Natural history of abdominal aortic aneurysm detected by screening. Br J Surg 1993; 80:718-20.
932. Englund R, Hudson P, Hanel K, et al. Expansion rates of small abdominal aortic aneurysms. Aust N Z J Surg 1998;68:21-4.
933. Grimshaw GM, Thompson JM, Hamer JD. A statistical analysis of the growth of small abdominal aortic aneurysms. Eur J Vasc Surg 1994;8:741-6.
934. Santilli SM, Littooy FN, Cambria RA, et al. Expansion rates and outcomes for the 3.0-cm to the 3.9-cm infrarenal abdominal aortic aneurysm. J Vasc Surg 2002;35:666-71.
935. Nevitt MP, Ballard DJ, Hallett JW Jr. Prognosis of abdominal aortic aneurysms. A population-based study. N Engl J Med 1989; 321:1009-14.
936. Cronenwett JL, Sargent SK, Wall MH, et al. Variables that affect the expansion rate and outcome of small abdominal aortic aneurysms. J Vasc Surg 1990;11:260-8; discussion 268-9.
937. Bengtsson H, Bergqvist D, Ekberg O, et al. Expansion pattern and risk of rupture of abdominal aortic aneurysms that were not operated on. Eur J Surg 1993;159:461-7.
938. Taylor LM Jr, Porter JM. Basic data related to clinical decision-making in abdominal aortic aneurysms. Ann Vasc Surg 1987;1:502-4.
939. Hollier LH, Taylor LM, Ochsner J. Recommended indications for operative treatment of abdominal aortic aneurysms: report of a

subcommittee of the Joint Council of the Society for Vascular Surgery and the North American Chapter of the International Society for Cardiovascular Surgery J Vasc Surg 1992;15:1046-56.
940. Hallin A, Bergqvist D, Holmberg L. Literature review of surgical management of abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2001;22:197-204.
941. Lederle FA, Wilson SE, Johnson GR, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002;346:1437-44.
942. Mealy K, Salman A. The true incidence of ruptured abdominal aortic aneurysms. Eur J Vasc Surg 1988;2:405-8.
943. Johansen K, Kohler TR, Nicholls SC, et al. Ruptured abdominal aortic aneurysm: the Harborview experience. J Vasc Surg 1991;13:240-5; discussion 245-7.
944. Heikkinen M, Salenius J, Zeitlin R, et al. The fate of AAA patients referred electively to vascular surgical unit. Scand J Surg 2002;91:345-52.
945. Szilagyi DE, Smith RF, DeRusso FJ, et al. Contribution of abdominal aortic aneurysmectomy to prolongation of life. Ann Surg 1966;164:678-99.
946. Cronenwett JL, Murphy TF, Zelenock GB, et al. Actuarial analysis of variables associated with rupture of small abdominal aortic aneurysms. Surgery 1985;98:472-83.
947. Powell JT, Brown LC. The natural history of abdominal aortic aneurysms and their risk of rupture. Acta Chir Belg 2001;101:11-6.
948. Chang JB, Stein TA, Liu JP, et al. Risk factors associated with rapid growth of small abdominal aortic aneurysms. Surgery 1997;121:117-22.
949. Axelrod DA, Henke PK, Wakefield TW, et al. Impact of chronic obstructive pulmonary disease on elective and emergency abdominal aortic aneurysm repair. J Vasc Surg 2001;33:72-6.
950. Conway KP, Byrne J, Townsend M, et al. Prognosis of patients turned down for conventional abdominal aortic aneurysm repair in the endovascular and sonographic era: Szilagyi revisited? J Vasc Surg 2001;33:752-7.
951. Watson CJ, Walton J, Shaw E, et al. What is the long-term outcome for patients with very small abdominal aortic aneurysms? Eur J Vasc Endovasc Surg 1997;14:299-304.
952. Brown PM, Pattenden R, Gutelius JR. The selective management of small abdominal aortic aneurysms: the Kingston study. J Vasc Surg 1992;15:21-5; discussion 25-7.
953. Scott RA, Tisi PV, Ashton HA, et al. Abdominal aortic aneurysm rupture rates: a 7-year follow-up of the entire abdominal aortic aneurysm population detected by screening. J Vasc Surg 1998; 28:124-8.
954. Katz DA, Littenberg B, Cronenwett JL. Management of small abdominal aortic aneurysms: early surgery vs watchful waiting. JAMA 1992;268:2678-86.
955. Hertzer NR, Young JR, Beven EG, et al. Late results of coronary bypass in patients with infrarenal aortic aneurysms. The Cleveland Clinic Study. Ann Surg 1987;205:360-7.
956. Perko MJ, Schroeder TV, Olsen PS, et al. Natural history of abdominal aortic aneurysm: a survey of 63 patients treated nonoperatively. Ann Vasc Surg 1993;7:113-6.
957. Galland RB, Whiteley MS, Magee TR. The fate of patients undergoing surveillance of small abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 1998;16:104-9.
958. Jones L, Pressdee DJ, Lamont PM, et al. A phase contrast (PC) rephase/dephase sequence of magnetic resonance angiography (MRA): a new technique for imaging distal run-off in the preoperative evaluation of peripheral vascular disease. Clin Radiol 1998;53:333-7.
959. Biancari F, Ylonen K, Anttila V, et al. Durability of open repair of infrarenal abdominal aortic aneurysm: a 15-year follow-up study. J Vasc Surg 2002;35:87-93.
960. Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. The UK Small Aneurysm Trial Participants. Lancet 1998;352:1649-55.
961. Brown LC, Powell JT. Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. UK Small Aneurysm Trial Participants. Ann Surg 1999;230:289-96; discussion 296-7.
962. Lederle FA, Johnson GR, Wilson SE, et al. Rupture rate of large abdominal aortic aneurysms in patients refusing or unfit for elective repair. JAMA 2002;287:2968-72.
963. United Kingdom Small Aneurysm Trial Participants. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002;346:1445-52.
964. Brewster DC, Cronenwett JL, Hallett JW Jr, et al. Guidelines for the treatment of abdominal aortic aneurysms: report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery J Vasc Surg 2003;37:1106-17.
965. Krupski WC, Selzman CH, Floridia R, et al. Contemporary management of isolated iliac aneurysms. J Vasc Surg 1998;28:1-11; discussion 11-3.
966. Kasirajan V, Hertzer NR, Beven EG, et al. Management of isolated common iliac artery aneurysms. Cardiovasc Surg 1998;6:171-7.
967. Carpenter JP, Barker CF, Roberts B, et al. Popliteal artery aneurysms: current management and outcome. J Vasc Surg 1994; 19:65-72; discussion 72-3.
968. Varga ZA, Locke-Edmunds JC, Baird RN. A multicenter study of popliteal aneurysms. Joint Vascular Research Group. J Vasc Surg 1994;20:171-7.
969. Vowden P, Wilkinson D, Ausobsky JR, et al. A comparison of three imaging techniques in the assessment of an abdominal aortic aneurysm. J Cardiovasc Surg (Torino) 1989;30:891-6.
970. Muluk SC, Gertler JP, Brewster DC, et al. Presentation and patterns of aortic aneurysms in young patients. J Vasc Surg 1994;20:880-6; discussion 887-8.
971. Kiell CS, Ernst CB. Advances in management of abdominal aortic aneurysm. Adv Surg 1993;26:73-98.
972. Newman AB, Arnold AM, Burke GL, et al. Cardiovascular disease and mortality in older adults with small abdominal aortic aneurysms detected by ultrasonography: the cardiovascular health study. Ann Intern Med 2001;134:182-90.
973. Zarins CK, Harris EJ Jr. Operative repair for aortic aneurysms: the gold standard. J Endovasc Surg 1997;4:232-41.
974. Crawford ES, Cohen ES. Aortic aneurysm: a multifocal disease. Presidential address. Arch Surg 1982;117:1393-400.
975. Bickerstaff LK, Pairolero PC, Hollier LH, et al. Thoracic aortic aneurysms: a population-based study. Surgery 1982;92:1103-8.
976. Pressler V, McNamara JJ. Aneurysm of the thoracic aorta: review of 260 cases. J Thorac Cardiovasc Surg 1985;89:50-4.
977. Lederle FA, Simel DL. The rational clinical examination: does this patient have abdominal aortic aneurysm? JAMA 1999; 281:77-82.
978. May AG, DeWeese JA, Frank I, et al. Surgical treatment of abdominal aortic aneurysms. Surgery 1968;63:711-21.
979. Nichols GB, Schilling PJ. Pseudo-retroperitoneal gas in rupture of aneurysm of abdominal aorta. Am J Roentgenol Radium Ther Nucl Med 1975;125:134-7.
980. JANOWER ML. Ruptured arteriosclerotic aneurysms of the abdominal aorta: roentgenographic findings on plain films. N

Engl J Med 1961;265:12-5.
981. Littooy FN, Steffan G, Greisler HP, et al. Use of sequential B-mode ultrasonography to manage abdominal aortic aneurysms. Arch Surg 1989;124:419-21.
982. Hara AK, Johnson CD, MacCarty RL, et al. Incidental extracolonic findings at CT colonography. Radiology 2000;215:353-7.
983. Flanigan RC, McKay TC, Olson M, et al. Limited efficacy of preoperative computed tomographic scanning for the evaluation of lymph node metastasis in patients before radical prostatectomy. Urology 1996;48:428-32.
984. Aoyagi K, Watanabe N, Yukihiro M, et al. Incidental detection of arterial aneurysms with Tc-99m human serum albumin. Clin Nucl Med 1996;21:485-6.
985. Phillips SM, King D. The role of ultrasound to detect aortic aneurysms in "urological" patients. Eur J Vasc Surg 1993;7:298-300.
986. Howe SF, Taylor RJ, Halloran BG, et al. Management of synchronous renal cell carcinoma and aortic disease. Surg Gynecol Obstet 1995;170:231-4.
987. Kumar A, Pham DH, Meindok H, et al. Diagnosis of bleeding mycotic iliac aneurysm on technetium-99m renal scan. J Nucl Med 1992;33:1548-9.
988. Akkersdijk GJ, Puylaert JB, de Vries AC. Abdominal aortic aneurysm as an incidental finding in abdominal ultrasonography. Br J Surg 1991;78:1261-3.
989. Thompson GT. Incidental findings on gallbladder sonography. Can Assoc Radiol J 1987;38:40-1.
990. Moreno AJ, Brown JM, Spicer MJ, et al. Ruptured abdominal aortic aneurysm identified incidental to bone scintigraphy. Eur J Nucl Med 1983;8:546-8.
991. Hautumm B, Grauel H. Aortic aneurysm in urology. Int Urol Nephrol 1982;14:3-11.
992. Spittell PC, Ehrsam JE, Anderson L, et al. Screening for abdominal aortic aneurysm during transthoracic echocardiography in a hypertensive patient population. J Am Soc Echocardiogr 1997;10:722-7.
993. Derbyshire ND, Lindsell DR, Collin J, et al. Opportunistic screening for abdominal aortic aneurysm. J Med Screen 1994;1:220-2.
994. Wolf YG, Otis SM, Schwend RB, et al. Screening for abdominal aortic aneurysms during lower extremity arterial evaluation in the vascular laboratory. J Vasc Surg 1995;22:417-21; discussion 421-3.
995. Rosch J, Keller FS, Porter JM, et al. Value of angiography in the management of abdominal aortic aneurysm. Cardiovasc Radiol 1978;1:83-94.
996. Bandyk DF. Preoperative imaging of aortic aneurysms: conventional and digital subtraction angiography, computed tomography scanning, and magnetic resonance imaging. Surg Clin North Am 1989;69:721-35.
997. Siegel CL, Cohan RH. CT of abdominal aortic aneurysms. AJR Am J Roentgenol 1994;163:17-29.
998. Turnipseed WD, Acher CW, Detmer DE, et al. Digital subtraction angiography and B-mode ultrasonography for abdominal and peripheral aneurysms. Surgery 1982;92:619-26.
999. Maloney JD, Pairolero PC, Smith SF Jr, et al. Ultrasound evaluation of abdominal aortic aneurysms. Circulation 1977;56(3 suppl):II80-5.
1000. Ellis M, Powell JT, Greenhalgh RM. Limitations of ultrasonography in surveillance of small abdominal aortic aneurysms. Br J Surg 1991;78:614-6.
1001. Andrews SM, Cuming R, Macsweeney ST, et al. Assessment of feasibility for endovascular prosthetic tube correction of aortic aneurysm. Br J Surg 1995;82:917-9.
1002. Diwan A, Sarkar R, Stanley JC, et al. Incidence of femoral and popliteal artery aneurysms in patients with abdominal aortic aneurysms. J Vasc Surg 2000;31:863-9.
1003. Helvie MA, Rubin JM, Silver TM, et al. The distinction between femoral artery pseudoaneurysms and other causes of groin masses: value of duplex Doppler sonography. AJR Am J Roentgenol 1988;150:1177-80.
1004. Bluth EI, Merritt CR, Sullivan MA. Gray-scale ultrasound evaluation of the lower extremities. JAMA 1982;247:3127-9.
1005. Hirsch JH, Thiele BL, Carter SS, et al. Aortic and lower extremity arterial aneurysms. J Clin Ultrasound 1981;9:29-31.
1006. Neiman HL, Yao JS, Silver TM. Gray-scale ultrasound diagnosis of peripheral arterial aneurysms. Radiology 1979;130:413-6.
1007. Collins GJ Jr, Rich NM, Phillips J, et al. Ultrasound diagnosis of popliteal arterial aneurysms. Am Surg 1976;42:853-8.
1008. Atallah C, al Hassan HK, Neglen P. Superficial femoral artery aneurysm—an uncommon site of aneurysm formation. Eur J Vasc Endovasc Surg 1995;10:502-4.
1009. Lindholt JS, Henneberg EW, Fasting H, et al. Hospital based screening of 65-73 year old men for abdominal aortic aneurysms in the county of Viborg, Denmark. J Med Screen 1996;3:43-6.
1010. Pleumeekers HJ, Hoes AW, Hofman A, et al. Selecting subjects for ultrasonographic screening for aneurysms of the abdominal aorta: four different strategies. Int J Epidemiol 1999;28:682-6.
1011. Lindholt JS, Vammen S, Juul S, et al. The validity of ultrasonographic scanning as screening method for abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 1999;17:472-5.
1012. Nasim A, Thompson MM, Sayers RD, et al. Role of magnetic resonance angiography for assessment of abdominal aortic aneurysm before endoluminal repair. Br J Surg 1998;85:641-4.
1013. Taylor SM, Mills JL, Fujitani RM. The juxtarenal abdominal aortic aneurysm: a more common problem than previously realized? Arch Surg 1994;129:734-7.
1014. Tennant WG, Hartnell GG, Baird RN, et al. Radiologic investigation of abdominal aortic aneurysm disease: comparison of three modalities in staging and the detection of inflammatory change. J Vasc Surg 1993;17:703-9.
1015. Lamah M, Darke S. Value of routine computed tomography in the preoperative assessment of abdominal aneurysm replacement. World J Surg 1999;23:1076-80; discussion 1080-1.
1016. Fillinger MF. Imaging of the thoracic and thoracoabdominal aorta. Semin Vasc Surg 2000;13:247-63.
1017. Errington ML, Ferguson JM, Gillespie IN, et al. Complete preoperative imaging assessment of abdominal aortic aneurysm with spiral CT angiography. Clin Radiol 1997;52:369-77.
1018. Rubin GD, Armerding MD, Dake MD, et al. Cost identification of abdominal aortic aneurysm imaging by using time and motion analyses. Radiology 2000;215:63-70.
1019. Galt SW, Pearce WH. Preoperative assessment of abdominal aortic aneurysms: noninvasive imaging versus routine arteriography. Semin Vasc Surg 1995;8:103-7.
1020. Coulam CH, Rubin GD. Acute aortic abnormalities. Semin Roentgenol 2001;36:148-64.
1021. Papanicolaou N, Wittenberg J, Ferrucci JT Jr, et al. Preoperative evaluation of abdominal aortic aneurysms by computed tomography. AJR Am J Roentgenol 1986;146:711-5.
1022. Vicaretti M, Young N, Jenkins J, et al. Helical computed tomography in the assessment of abdominal aortic pathology. Australas Radiol 1997;41:125-31.
1023. Eriksson I, Forsberg JO, Hemmingsson A, et al. Preoperative evaluation of abdominal aortic aneurysms: is there a need for

aortography? Acta Chir Scand 1981;147:533-7.
1024. Broeders IA, Blankensteijn JD. Preoperative imaging of the aortoiliac anatomy in endovascular aneurysm surgery. Semin Vasc Surg 1999;12:306-14.
1025. Ludman CN, Yusuf SW, Whitaker SC, et al. Feasibility of using dynamic contrast-enhanced magnetic resonance angiography as the sole imaging modality prior to endovascular repair of abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2000;19:524-30.
1026. Hovsepian DM, Siegel BA, Kimbiris G, et al. Tc-99m sulfur colloid scintigraphy for detecting perigraft flow following endovascular aortic aneurysm repair: a feasibility study. Cardiovasc Intervent Radiol 1999;22:447-51.
1027. Hanson SR, Kotze HF, Pieters H, et al. Analysis of indium-111 platelet kinetics and imaging in patients with aortic grafts and abdominal aortic aneurysms. Arteriosclerosis 1990;10:1037-44.
1028. Prince MR, Yucel EK, Kaufman JA, et al. Dynamic gadolinium-enhanced three-dimensional abdominal MR arteriography. J Magn Reson Imaging 1993;3:877-81.
1029. Frayne R, Grist TM, Swan JS, et al. 3D MR DSA: effects of injection protocol and image masking. J Magn Reson Imaging 2000;12:476-87.
1030. Yamashita Y, Mitsuzaki K, Tang Y, et al. Gadolinium-enhanced breath-hold three-dimensional time-of-flight MR angiography of the abdominal and pelvic vessels: the value of ultrafast MP-RAGE sequences. J Magn Reson Imaging 1997;7:623-8.
1031. Thurnher SA, Dorffner R, Thurnher MM, et al. Evaluation of abdominal aortic aneurysm for stent-graft placement: comparison of gadolinium-enhanced MR angiography versus helical CT angiography and digital subtraction angiography. Radiology 1997;205:341-52.
1032. Scott RA, Ashton HA, Kay DN. Abdominal aortic aneurysm in 4237 screened patients: prevalence, development and management over 6 years. Br J Surg 1991;78:1122-5.
1033. Grimshaw GM, Thompson JM. The abnormal aorta: a statistical definition and strategy for monitoring change. Eur J Vasc Endovasc Surg 1995;10:95-100.
1034. Scott RA, Vardulaki KA, Walker NM, et al. The long-term benefits of a single scan for abdominal aortic aneurysm (AAA) at age 65. Eur J Vasc Endovasc Surg 2001;21:535-40.
1035. Cole CW, Hill GB, Millar WJ, et al. Selective screening for abdominal aortic aneurysm. Chronic Dis Can. 1996;17:51-5.
1036. Multicentre Aneurysm Screening Study Group. Multicentre aneurysm screening study (MASS): cost effectiveness analysis of screening for abdominal aortic aneurysms based on four year results from randomised controlled trial. BMJ 2002;325:1135.
1037. Ashton HA, Buxton MJ, Day NE, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial. Lancet 2002;360:1531-9.
1038. Connelly JB, Hill GB, Millar WJ. The detection and management of abdominal aortic aneurysm: a cost-effectiveness analysis. Clin Invest Med 2002;25:127-33.
1039. Soisalon-Soininen S, Rissanen P, Pentikainen T, et al. Cost-effectiveness of screening for familial abdominal aortic aneurysms. Vasa 2001;30:262-70.
1040. Lee TY, Korn P, Heller JA, et al. The cost-effectiveness of a "quick-screen" program for abdominal aortic aneurysms. Surgery 2002;132:399-407.
1041. Fleming C, Whitlock EP, Beil TL, et al. Screening for abdominal aortic aneurysm: a best-evidence systematic review for the U.S. Preventive Services Task Force. Ann Intern Med 2005;142:203-11.
1042. Meenan RT, Fleming C, Whitlock EP, et al. Cost-Effectiveness Analyses of Population-Based Screening for Abdominal Aortic Aneurysm. Evidence Synthesis. AHRQ Publication No. 05-0569-C, February 2005. Agency for Healthcare Research and Quality, Rockville, Md. Available at: http://www.ahrq.gov/clinic/uspstf05/aaascr/aaacost.htm. Accessed July 16, 2005.
1043. Brophy C, Tilson JE, Tilson MD. Propranolol delays the formation of aneurysms in the male blotchy mouse. J Surg Res 1988; 44:687-9.
1044. Ricci MA, Slaiby JM, Gadowski GR, et al. Effects of hypertension and propranolol upon aneurysm expansion in the Anidjar/Dobrin aneurysm model. Ann N Y Acad Sci 1996; 800:89-96.
1045. Leach SD, Toole AL, Stern H, et al. Effect of beta-adrenergic blockade on the growth rate of abdominal aortic aneurysms. Arch Surg 1988;123:606-9.
1046. Gadowski GR, Pilcher DB, Ricci MA. Abdominal aortic aneurysm expansion rate: effect of size and beta-adrenergic blockade. J Vasc Surg 1994;19:727-31.
1047. Lindholt JS, Henneberg EW, Juul S, et al. Impaired results of a randomised double blinded clinical trial of propranolol versus placebo on the expansion rate of small abdominal aortic aneurysms. Int Angiol 1999;18:52-7.
1048. Shores J, Berger KR, Murphy EA, et al. Progression of aortic dilatation and the benefit of long-term beta-adrenergic blockade in Marfan's syndrome. N Engl J Med 1994;330:1335-41.
1049. Kertai MD, Boersma E, Westerhout CM, et al. A combination of statins and beta-blockers is independently associated with a reduction in the incidence of perioperative mortality and nonfatal myocardial infarction in patients undergoing abdominal aortic aneurysm surgery. Eur J Vasc Endovasc Surg 2004;28:343-52.
1050. Fleisher LA, Eagle KA. Clinical practice: lowering cardiac risk in noncardiac surgery. N Engl J Med 2001;345:1677-82.
1051. Auerbach AD, Goldman L. Beta-blockers and reduction of cardiac events in noncardiac surgery: scientific JAMA 2002; 287:1435-44.
1052. Cook TA, Galland RB. A prospective study to define the optimum rescreening interval for small abdominal aortic aneurysm. Cardiovasc Surg 1996;4:441-4.
1053. Finlayson SR, Birkmeyer JD, Fillinger MF, et al. Should endovascular surgery lower the threshold for repair of abdominal aortic aneurysms? J Vasc Surg 1999;29:973-85.
1054. Thompson RW. Detection and management of small aortic aneurysms. N Engl J Med 2002;346:1484-6.
1055. d'Audiffret A, Santilli S, Tretinyak A, et al. Fate of the ectatic infrarenal aorta: expansion rates and outcomes. Ann Vasc Surg 2002;16:534-6.
1056. Hallett JW Jr, Naessens JM, Ballard DJ. Early and late outcome of surgical repair for small abdominal aortic aneurysms: a population-based analysis. J Vasc Surg 1993;18:684-91.
1057. Koskas F, Kieffer E. Long-term survival after elective repair of infrarenal abdominal aortic aneurysm: results of a prospective multicentric study. Association for Academic Research in Vascular Surgery (AURC). Ann Vasc Surg 1997;11:473-81.
1058. Aune S. Risk factors and operative results of patients aged less than 66 years operated on for asymptomatic abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2001;22:240-3.
1059. Brady AR, Fowkes FG, Thompson SG, et al. Aortic aneurysm diameter and risk of cardiovascular mortality. Arterioscler Thromb Vasc Biol 2001;21:1203-7.
1060. Starr JE, Hertzer NR, Mascha EJ, et al. Influence of gender on cardiac risk and survival in patients with infrarenal aortic aneurysms. J Vasc Surg 1996;23:870-80.

1061. Crawford ES, Saleh SA, Babb JW 3rd, et al. Infrarenal abdominal aortic aneurysm: factors influencing survival after operation performed over a 25-year period. Ann Surg 1981;193:699-709.
1062. Hollier LH, Plate G, O'Brien PC, et al. Late survival after abdominal aortic aneurysm repair: influence of coronary artery disease. J Vasc Surg 1984;1:290-9.
1063. Reigel MM, Hollier LH, Kazmier FJ, et al. Late survival in abdominal aortic aneurysm patients: the role of selective myocardial revascularization on the basis of clinical symptoms. J Vasc Surg 1987;5:222-7.
1064. Glance LG. Selective preoperative cardiac screening improves five-year survival in patients undergoing major vascular surgery: a cost-effectiveness analysis. J Cardiothorac Vasc Anesth 1999;13:265-71.
1065. Golden MA, Whittemore AD, Donaldson MC, et al. Selective evaluation and management of coronary artery disease in patients undergoing repair of abdominal aortic aneurysms: a 16-year experience. Ann Surg 1990;212:415-20; discussion 420-3.
1066. Lachapelle K, Graham AM, Symes JF. Does the clinical evaluation of the cardiac status predict outcome in patients with abdominal aortic aneurysms? J Vasc Surg 1992;15:964-70; discussion 970-1.
1067. Suggs WD, Smith RB 3rd, Weintraub WS, et al. Selective screening for coronary artery disease in patients undergoing elective repair of abdominal aortic aneurysms. J Vasc Surg 1993;18:349-55; discussion 355-7.
1068. Hertzer NR, Mascha EJ, Karafa MT, et al. Open infrarenal abdominal aortic aneurysm repair: the Cleveland Clinic experience from 1989 to 1998. J Vasc Surg 2002;35:1145-54.
1069. McFalls EO, Ward HB, Moritz TE, et al. Coronary-artery revascularization before elective major vascular surgery. N Engl J Med 2004;351:2795-804. Erratum in: N Engl J Med 2005; 95:19.
1070. Darling RC 3rd, Cordero JA Jr, Chang BB, et al. Advances in the surgical repair of ruptured abdominal aortic aneurysms. Cardiovasc Surg 1996;4:720-3.
1071. Sicard GA, Reilly JM, Rubin BG, et al. Transabdominal versus retroperitoneal incision for abdominal aortic surgery: report of a prospective randomized trial. J Vasc Surg 1995;21:174-81; discussion 181-3.
1072. Cambria RP, Brewster DC, Abbott WM, et al. Transperitoneal versus retroperitoneal approach for aortic reconstruction: a randomized prospective study. J Vasc Surg 1990;11:314-24; discussion 324-5.
1073. Sieunarine K, Lawrence-Brown MM, Goodman MA. Comparison of transperitoneal and retroperitoneal approaches for infrarenal aortic surgery: early and late results. Cardiovasc Surg 1997;5:71-6.
1074. Blankensteijn JD, Lindenburg FP, Van der Graaf Y, et al. Influence of study design on reported mortality and morbidity rates after abdominal aortic aneurysm repair. Br J Surg 1998;85:1624-30.
1075. Lawrence PF, Gazak C, Bhirangi L, et al. The epidemiology of surgically repaired aneurysms in the United States. J Vasc Surg 1999;30:632-40.
1076. Heller JA, Weinberg A, Arons R, et al. Two decades of abdominal aortic aneurysm repair: have we made any progress? J Vasc Surg 2000;32:1091-100.
1077. Huber TS, Wang JG, Derrow AE, et al. Experience in the United States with intact abdominal aortic aneurysm repair. J Vasc Surg 2001;33:304-10; discussion 310-1.
1078. Dimick JB, Stanley JC, Axelrod DA, et al. Variation in death rate after abdominal aortic aneurysmectomy in the United States: impact of hospital volume, gender, and age. Ann Surg 2002; 235:579-85.
1079. Lloyd WE, Paty PS, Darling RC 3rd, et al. Results of 1000 consecutive elective abdominal aortic aneurysm repairs. Cardiovasc Surg 1996;4:724-6.
1080. Menard MT, Chew DK, Chan RK, et al. Outcome in patients at high risk after open surgical repair of abdominal aortic aneurysm. J Vasc Surg 2003;37:285-92.
1081. Ernst CB. Abdominal aortic aneurysm. N Engl J Med 1993; 328:1167-72.
1082. Johnston KW, Scobie TK. Multicenter prospective study of nonruptured abdominal aortic aneurysms. I. Population and operative management. J Vasc Surg 1988;7:69-81.
1083. Richardson JD, Main KA. Repair of abdominal aortic aneurysms. A statewide experience. Arch Surg 1991;126:614-6.
1084. Hannan EL, Kilburn H Jr, O'Donnell JF, et al. A longitudinal analysis of the relationship between in-hospital mortality in New York State and the volume of abdominal aortic aneurysm surgeries performed. Health Serv Res. 1992;27:517-42.
1085. Johnston KW. Nonruptured abdominal aortic aneurysm: six-year follow-up results from the multicenter prospective Canadian aneurysm study. Canadian Society for Vascular Surgery Aneurysm Study Group. J Vasc Surg 1994;20:163-70.
1086. Katz DJ, Stanley JC, Zelenock GB. Operative mortality rates for intact and ruptured abdominal aortic aneurysms in Michigan: an eleven-year statewide experience. J Vasc Surg 1994;19:804-15; discussion 816-7.
1087. Kazmers A, Jacobs L, Perkins A, et al. Abdominal aortic aneurysm repair in Veterans Affairs medical centers. J Vasc Surg 1996;23:191-200.
1088. Wen SW, Simunovic M, Williams JI, et al. Hospital volume, calendar age, and short term outcomes in patients undergoing repair of abdominal aortic aneurysms: the Ontario experience, 1988-92. J Epidemiol Community Health 1996;50:207-13.
1089. Kantonen I, Lepantalo M, Salenius JP, et al. Mortality in abdominal aortic aneurysm surgery—the effect of hospital volume, patient mix and surgeon's case load. Eur J Vasc Endovasc Surg 1997;14:375-9.
1090. Bradbury AW, Adam DJ, Makhdoomi KR, et al. A 21-year experience of abdominal aortic aneurysm operations in Edinburgh. Br J Surg 1998;85:645-7.
1091. Manheim LM, Sohn MW, Feinglass J, et al. Hospital vascular surgery volume and procedure mortality rates in California,982-1994. J Vasc Surg 1998;28:45-56; discussion 56-8.
1092. Dardik A, Lin JW, Gordon TA, et al. Results of elective abdominal aortic aneurysm repair in the 1990s: a population-based analysis of 2335 cases. J Vasc Surg 1999;30:985-95.
1093. Pearce WH, Parker MA, Feinglass J, et al. The importance of surgeon volume and training in outcomes for vascular surgical procedures. J Vasc Surg 1999;29:768-76; discussion 777-8.
1094. Sollano JA, Gelijns AC, Moskowitz AJ, et al. Volume-outcome relationships in cardiovascular operations: New York State, 1990-1995. J Thorac Cardiovasc Surg 1999;117:419-28; discussion 428-30.
1095. Kazmers A, Perkins AJ, Jacobs LA. Aneurysm rupture is independently associated with increased late mortality in those surviving abdominal aortic aneurysm repair. J Surg Res 2001;95:50-3.
1096. Huber TS, Seeger JM. Dartmouth Atlas of Vascular Health Care review: impact of hospital volume, surgeon volume, and training on outcome. J Vasc Surg 2001;34:751-6.
1097. Panneton JM, Lassonde J, Laurendeau F. Ruptured abdominal aortic aneurysm: impact of comorbidity and postoperative com-

plications on outcome. Ann Vasc Surg 1995;9:535-41.
1098. Seiwert AJ, Elmore JR, Youkey JR, et al. Samuels Award. Ruptured abdominal aortic aneurysm repair: the financial analysis. Surg Gynecol Obstet 1995;170:91-6.
1099. Barry MC, Burke PE, Sheehan S, et al. An "all comers" policy for ruptured abdominal aortic aneurysms: how can results be improved? Eur J Surg 1998;164:263-70.
1100. Noel AA, Gloviczki P, Cherry KJ Jr, et al. Ruptured abdominal aortic aneurysms: the excessive mortality rate of conventional repair. J Vasc Surg 2001;34:41-6.
1101. Hertzer NR, Avellone JC, Farrell CJ, et al. The risk of vascular surgery in a metropolitan community: with observations on surgeon experience and hospital size. J Vasc Surg 1984;1:13-21.
1102. Johnston KW. Ruptured abdominal aortic aneurysm: six-year follow-up results of a multicenter prospective study. Canadian Society for Vascular Surgery Aneurysm Study Group. J Vasc Surg 1994;19:888-900.
1103. Amundsen S, Skjaerven R, Trippestad A, et al. Abdominal aortic aneurysms—a study of factors influencing postoperative mortality. Norwegian Aortic Aneurysm Trial. Eur J Vasc Surg 1989;3:405-9.
1104. Gloviczki P, Pairolero PC, Mucha P Jr, et al. Ruptured abdominal aortic aneurysms: repair should not be denied. J Vasc Surg 1992;15:851-7; discussion 857-9.
1105. Halpern VJ, Kline RG, D'Angelo AJ, et al. Factors that affect the survival rate of patients with ruptured abdominal aortic aneurysms. J Vasc Surg 1997;26:939-45; discussion 945-8.
1106. Steyerberg EW, Kievit J, de Mol Van Otterloo JC, et al. Perioperative mortality of elective abdominal aortic aneurysm surgery: a clinical prediction rule based on literature and individual patient data. Arch Intern Med 1995;155:1998-2004.
1107. Brady AR, Fowkes FG, Greenhalgh RM, et al. Risk factors for postoperative death following elective surgical repair of abdominal aortic aneurysm: results from the UK Small Aneurysm Trial. On behalf of the UK Small Aneurysm Trial participants. Br J Surg 2000;87:742-9.
1108. Kazmers A, Perkins AJ, Jacobs LA. Outcomes after abdominal aortic aneurysm repair in those > or =80 years of age: recent Veterans Affairs experience. Ann Vasc Surg 1998;12:106-12.
1109. O'Hara PJ, Hertzer NR, Krajewski LP, et al. Ten-year experience with abdominal aortic aneurysm repair in octogenarians: early results and late outcome. J Vasc Surg 1995;21:830-7; discussion 837-8.
1110. Harris KA, Ameli FM, Lally M, et al. Abdominal aortic aneurysm resection in patients more than 80 years old. Surg Gynecol Obstet 1986;162:536-8.
1111. Collins TC, Johnson M, Daley J, et al. Preoperative risk factors for 30-day mortality after elective surgery for vascular disease in Department of Veterans Affairs hospitals: is race important? J Vasc Surg 2001;34:634-40.
1112. Shackley P, Slack R, Booth A, et al. Is there a positive volume-outcome relationship in peripheral vascular surgery? Results of a systematic Eur J Vasc Endovasc Surg 2000;20:326-35.
1113. Amundsen S, Skjaerven R, Trippestad A, et al. Abdominal aortic aneurysms: is there an association between surgical volume, surgical experience, hospital type and operative mortality? Members of the Norwegian Abdominal Aortic Aneurysm Trial. Acta Chir Scand 1990;156:323-7; discussion 327-8.
1114. Soisalon-Soininen S, Salo JA, Takkunen O, et al. Comparison of long-term survival after repair of ruptured and non-ruptured abdominal aortic aneurysm. Vasa 1995;24:42-8.
1115. Cho JS, Gloviczki P, Martelli E, et al. Long-term survival and late complications after repair of ruptured abdominal aortic aneurysms. J Vasc Surg 1998;27:813-9; discussion 819-20.
1116. Feinglass J, Cowper D, Dunlop D, et al. Late survival risk factors for abdominal aortic aneurysm repair: experience from fourteen Department of Veterans Affairs hospitals. Surgery 1995;118:16-24.
1117. Stonebridge PA, Callam MJ, Bradbury AW, et al. Comparison of long-term survival after successful repair of ruptured and non-ruptured abdominal aortic aneurysm. Br J Surg 1993;80:585-6.
1118. Norman PE, Semmens JB, Lawrence-Brown MM. Long-term relative survival following surgery for abdominal aortic aneurysm: a review. Cardiovasc Surg 2001;9:219-24.
1119. Evans SM, Adam DJ, Brittenden J, et al. Vascular Surgical Society of Great Britain and Ireland: long-term survival following repair of ruptured abdominal aortic aneurysm in patients over 75 years of age. Br J Surg 1999;86:696.
1120. Hallett JW Jr, Marshall DM, Petterson TM, et al. Graft-related complications after abdominal aortic aneurysm repair: reassurance from a 36-year population-based experience. J Vasc Surg 1997;25:277-84; discussion 285-6.
1121. Crawford ES, Beckett WC, Greer MS. Juxtarenal infrarenal abdominal aortic aneurysm: special diagnostic and therapeutic considerations. Ann Surg 1986;203:661-70.
1122. Ayari R, Paraskevas N, Rosset E, et al. Juxtarenal aneurysm: comparative study with infrarenal abdominal aortic aneurysm and proposition of a new classification. Eur J Vasc Endovasc Surg 2001;22:169-74.
1123. Faggioli G, Stella A, Freyrie A, et al. Early and long-term results in the surgical treatment of juxtarenal and pararenal aortic aneurysms. Eur J Vasc Endovasc Surg 1998;15:205-11.
1124. Qvarfordt PG, Stoney RJ, Reilly LM, et al. Management of pararenal aneurysms of the abdominal aorta. J Vasc Surg 1986;3:84-93.
1125. Nypaver TJ, Shepard AD, Reddy DJ, et al. Repair of pararenal abdominal aortic aneurysms: an analysis of operative management. Arch Surg 1993;128:803-11; discussion 811-3.
1126. Jean-Claude JM, Reilly LM, Stoney RJ, et al. Pararenal aortic aneurysms: the future of open aortic aneurysm repair. J Vasc Surg 1999;29:902-12.
1127. Anagnostopoulos PV, Shepard AD, Pipinos II, et al. Factors affecting outcome in proximal abdominal aortic aneurysm repair. Ann Vasc Surg 2001;15:511-9.
1128. Cox GS, O'Hara PJ, Hertzer NR, et al. Thoracoabdominal aneurysm repair: a representative experience. J Vasc Surg 1992; 15:780-7; discussion 787-8.
1129. Svensson LG, Crawford ES, Hess KR, et al. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993;17:357-68; discussion 368-70.
1130. Coselli JS, LeMaire SA, Buket S, et al. Subsequent proximal aortic operations in 123 patients with previous infrarenal abdominal aortic aneurysm surgery. J Vasc Surg 1995;22:59-67.
1131. Schwartz LB, Belkin M, Donaldson MC, et al. Improvement in results of repair of type IV thoracoabdominal aortic aneurysms. J Vasc Surg 1996;24:74-81.
1132. Dunning PG, Dudgill S, Brown AS, et al. Vascular Surgical Society of Great Britain and Ireland: total abdominal approach for repair of type IV thoracoabdominal aortic aneurysm. Br J Surg 1999;86:696.
1133. Martin GH, O'Hara PJ, Hertzer NR, et al. Surgical repair of aneurysms involving the suprarenal, visceral, and lower thoracic aortic segments: early results and late outcome. J Vasc Surg 2000;31:851-62.
1134. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc

Surg 1991;5:491-9.
1135. Anderson PL, Arons RR, Moskowitz AJ, et al. A statewide experience with endovascular abdominal aortic aneurysm repair: rapid diffusion with excellent early results. J Vasc Surg 2004;39:10-9.
1136. Jacobs TS, Won J, Gravereaux EC, et al. Mechanical failure of prosthetic human implants: a 10-year experience with aortic stent graft devices. J Vasc Surg 2003;37:16-26.
1137. Zarins CK; AneuRx Clinical Investigators. The US AneuRx Clinical Trial: 6-year clinical update 2002. J Vasc Surg 2003; 37:904-8.
1138. Dillavou ED, Muluk SC, Rhee RY, et al. Does hostile neck anatomy preclude successful endovascular aortic aneurysm repair? J Vasc Surg 2003;38:657-63.
1139. Arko FR, Filis KA, Seidel SA, et al. How many patients with infrarenal aneurysms are candidates for endovascular repair? The Northern California experience. J Endovasc Ther 2004;11:33-40.
1140. Carpenter JP, Baum RA, Barker CF, et al. Impact of exclusion criteria on patient selection for endovascular abdominal aortic aneurysm repair. J Vasc Surg 2001;34:1050-4.
1141. Becker GJ, Kovacs M, Mathison MN, et al. Risk stratification and outcomes of transluminal endografting for abdominal aortic aneurysm: 7-year experience and long-term follow-up. J Vasc Interv Radiol 2001;12:1033-46.
1142. Mathison M, Becker GJ, Katzen BT, et al. The influence of female gender on the outcome of endovascular abdominal aortic aneurysm repair. J Vasc Interv Radiol 2001;12:1047-51.
1143. Wolf YG, Arko FR, Hill BB, et al. Gender differences in endovascular abdominal aortic aneurysm repair with the AneuRx stent graft. J Vasc Surg 2002;35:882-6.
1144. Veith FJ, Baum RA, Ohki T, et al. Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. J Vasc Surg 2002;35:1029-35.
1145. White RA, Donayre C, Walot I, et al. Abdominal aortic aneurysm rupture following endoluminal graft deployment: report of a predictable event. J Endovasc Ther 2000;7:257-62.
1146. Abraham CZ, Chuter TA, Reilly LM, et al. Abdominal aortic aneurysm repair with the Zenith stent graft: short to midterm results. J Vasc Surg 2002;36:217-24; discussion 224-5.
1147. Zarins CK, Wolf YG, Lee WA, et al. Will endovascular repair replace open surgery for abdominal aortic aneurysm repair? Ann Surg 2000;232:501-7.
1148. Zarins CK, White RA, Moll FL, et al. The AneuRx stent graft: four-year results and worldwide experience 2000. J Vasc Surg 2001;33(2 suppl):S135-45. Erratum in: J Vasc Surg 2001; 33:1318.
1149. Sapirstein W, Chandeysson P, Wentz C. The Food and Drug Administration approval of endovascular grafts for abdominal aortic aneurysm: an 18-month retrospective. J Vasc Surg 2001;34:180-3.
1150. Harris PL, Buth J. An update on the important findings from the EUROSTAR EVAR registry. Vascular. 2004;12:33-8.
1151. Steinmetz E, Rubin BG, Sanchez LA, et al. Type II endoleak after endovascular abdominal aortic aneurysm repair: a conservative approach with selective intervention is safe and cost-effective. J Vasc Surg 2004;39:306-13.
1152. Stelter W, Umscheid T, Ziegler P. Three-year experience with modular stent-graft devices for endovascular AAA treatment. J Endovasc Surg 1997;4:362-9.
1153. Amesur NB, Zajko AB, Orons PD, et al. Endovascular treatment of iliac limb stenoses or occlusions in 31 patients treated with the Ancure endograft. J Vasc Interv Radiol 2000;11:421-8.
1154. Baum RA, Shetty SK, Carpenter JP, et al. Limb kinking in supported and unsupported abdominal aortic stent-grafts. J Vasc Interv Radiol 2000;11:1165-71.
1155. Fairman RM, Baum RA, Carpenter JP, et al. Limb interventions in patients undergoing treatment with an unsupported bifurcated aortic endograft system: a review of the Phase II EVT Trial. J Vasc Surg 2002;36:118-26.
1156. Greenberg RK, Lawrence-Brown M, Bhandari G, et al. An update of the Zenith endovascular graft for abdominal aortic aneurysms: initial implantation and mid-term follow-up data. J Vasc Surg 2001;33(2 suppl):S157-64.
1157. Conners MS 3rd, Sternbergh WC 3rd, Carter G, et al. Endograft migration one to four years after endovascular abdominal aortic aneurysm repair with the AneuRx device: a cautionary note. J Vasc Surg 2002;36:476-84.
1158. Makaroun MS, Deaton DH. Is proximal aortic neck dilatation after endovascular aneurysm exclusion a cause for concern? J Vasc Surg 2001;33(2 suppl):S39-45.
1159. Matsumura JS, Chaikof EL. Continued expansion of aortic necks after endovascular repair of abdominal aortic aneurysms. EVT Investigators. EndoVascular Technologies, Inc. J Vasc Surg 1998;28:422-30; discussion 430-1.
1160. de Virgilio C, Bui H, Donayre C, et al. Endovascular vs open abdominal aortic aneurysm repair: a comparison of cardiac morbidity and mortality. Arch Surg 1999;134:947-50; discussion 950-1.
1161. Aziz IN, Lee JT, Kopchok GE, et al. Cardiac risk stratification in patients undergoing endoluminal graft repair of abdominal aortic aneurysm: a single-institution experience with 365 patients. J Vasc Surg 2003;38:56-60.
1162. Cuypers PW, Gardien M, Buth J, et al. Cardiac response and complications during endovascular repair of abdominal aortic aneurysms: a concurrent comparison with open surgery. J Vasc Surg 2001;33:353-60.
1163. Buth J, Laheij RJ. Early complications and endoleaks after endovascular abdominal aortic aneurysm repair: report of a multicenter study. J Vasc Surg 2000;31(1 pt 1):134-46.
1164. Chuter TA, Reilly LM, Faruqi RM, et al. Endovascular aneurysm repair in high-risk patients. J Vasc Surg 2000;31(1 pt 1):122-33.
1165. May J, White GH, Waugh R, et al. Improved survival after endoluminal repair with second-generation prostheses compared with open repair in the treatment of abdominal aortic aneurysms: a 5-year concurrent comparison using life table method. J Vasc Surg 2001;33(2 suppl):S21-6.
1166. Buth J, van Marrewijk CJ, Harris PL, et al. Outcome of endovascular abdominal aortic aneurysm repair in patients with conditions considered unfit for an open procedure: a report on the EUROSTAR experience. J Vasc Surg 2002;35:211-21.
1167. Blum U, Voshage G, Beyersdorf F, et al. Two-center German experience with aortic endografting. J Endovasc Surg 1997; 4:137-46.
1168. May J, White GH, Yu W, et al. Endovascular grafting for abdominal aortic aneurysms: changing incidence and indication for conversion to open operation. Cardiovasc Surg 1998;6:194-7.
1169. Amesur NB, Zajko AB, Orons PD, et al. Embolotherapy of persistent endoleaks after endovascular repair of abdominal aortic aneurysm with the Ancure-endovascular technologies endograft system. J Vasc Interv Radiol 1999;10:1175-82.
1170. Becquemin J, Bourriez A, D'Audiffret A, et al. Mid-term results of endovascular versus open repair for abdominal aortic aneurysm in patients anatomically suitable for endovascular

repair. Eur J Vasc Endovasc Surg 2000;19:656-61.
1171. Zarins CK, White RA, Fogarty TJ. Aneurysm rupture after endovascular repair using the AneuRx stent graft. J Vasc Surg 2000;31:960-70.
1172. Blum U, Hauer M, Pfammatter T, et al. Percutaneous endoprosthesis for treatment of aortic aneurysms. World J Surg 2001;25:347-52; discussion 353-4.
1173. Fairman RM, Velazquez O, Baum R, et al. Endovascular repair of aortic aneurysms: critical events and adjunctive procedures. J Vasc Surg 2001;33:1226-32.
1174. Holzenbein TJ, Kretschmer G, Thurnher S, et al. Midterm durability of abdominal aortic aneurysm endograft repair: a word of caution. J Vasc Surg 2001;33(2 suppl):S46-54.
1175. Howell MH, Strickman N, Mortazavi A, et al. Preliminary results of endovascular abdominal aortic aneurysm exclusion with the AneuRx stent-graft. J Am Coll Cardiol 2001;38:1040-6.
1176. Sicard GA, Rubin BG, Sanchez LA, et al. Endoluminal graft repair for abdominal aortic aneurysms in high-risk patients and octogenarians: is it better than open repair? Ann Surg 2001; 234:427-35; discussion 435-7.
1177. Dattilo JB, Brewster DC, Fan CM, et al. Clinical failures of endovascular abdominal aortic aneurysm repair: incidence, causes, and management. J Vasc Surg 2002;35:1137-44.
1178. Sampram ES, Karafa MT, Mascha EJ, et al. Nature, frequency, and predictors of secondary procedures after endovascular repair of abdominal aortic aneurysm. J Vasc Surg 2003;37:930-7.
1179. Ouriel K, Greenberg RK, Clair DG, et al. Endovascular aneurysm repair: gender-specific results. J Vasc Surg 2003; 38:93-8.
1180. Shames ML, Sanchez LA, Rubin BG, et al. Delayed complications after endovascular AAA repair in women. J Endovasc Ther 2003;10:10-5.
1181. Moore WS, Rutherford RB. Transfemoral endovascular repair of abdominal aortic aneurysm: results of the North American EVT phase 1 trial. EVT Investigators. J Vasc Surg 1996;23:543-53.
1182. Coppi G, Pacchioni R, Moratto R, et al. Experience with the Stentor endograft at four Italian centers. J Endovasc Surg 1998;5:206-15.
1183. Becquemin JP, Lapie V, Favre JP, et al. Mid-term results of a second generation bifurcated endovascular graft for abdominal aortic aneurysm repair: the French Vanguard trial. J Vasc Surg 1999;30:209-18.
1184. Zarins CK, White RA, Schwarten D, et al. AneuRx stent graft versus open surgical repair of abdominal aortic aneurysms: multicenter prospective clinical trial. J Vasc Surg 1999;29:292-305; discussion 306-8.
1185. Zarins CK, White RA, Hodgson KJ, et al. Endoleak as a predictor of outcome after endovascular aneurysm repair: AneuRx multicenter clinical trial. J Vasc Surg 2000;32:90-107.
1186. Beebe HG, Cronenwett JL, Katzen BT, et al. Results of an aortic endograft trial: impact of device failure beyond 12 months. J Vasc Surg 2001;33(2 suppl):S55-63.
1187. Faries PL, Brener BJ, Connelly TL, et al. A multicenter experience with the Talent endovascular graft for the treatment of abdominal aortic aneurysms. J Vasc Surg 2002;35:1123-8.
1188. Matsumura JS, Brewster DC, Makaroun MS, et al. A multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysm. J Vasc Surg 2003;37:262-71.
1189. Harris PL, Vallabhaneni SR, Desgranges P, et al. Incidence and risk factors of late rupture, conversion, and death after endovascular repair of infrarenal aortic aneurysms: the EUROSTAR experience. European Collaborators on Stent/graft techniques for aortic aneurysm repair. J Vasc Surg 2000;32:739-49.
1190. Vallabhaneni SR, Harris PL. Lessons learnt from the EUROSTAR registry on endovascular repair of abdominal aortic aneurysm repair. Eur J Radiol 2001;39:34-41.
1191. Peppelenbosch N, Buth J, Harris PL, et al. Diameter of abdominal aortic aneurysm and outcome of endovascular aneurysm repair: does size matter? A report from EUROSTAR. J Vasc Surg 2004;39:288-97.
1192. Riambau V, Laheij RJ, Garcia-Madrid C, et al. The association between co-morbidity and mortality after abdominal aortic aneurysm endografting in patients ineligible for elective open surgery. Eur J Vasc Endovasc Surg 2001;22:265-70.
1193. Walker SR, Macierewicz J, MacSweeney ST, et al. Mortality rates following endovascular repair of abdominal aortic aneurysms. J Endovasc Surg 1999;6:233-8.
1194. Birch SE, Stary DR, Scott AR. Cost of endovascular versus open surgical repair of abdominal aortic aneurysms. Aust N Z J Surg 2000;70:660-6.
1195. Clair DG, Gray B, O'hara PJ, et al. An evaluation of the costs to health care institutions of endovascular aortic aneurysm repair. J Vasc Surg 2000;32:148-52.
1196. Bosch JL, Lester JS, McMahon PM, et al. Hospital costs for elective endovascular and surgical repairs of infrarenal abdominal aortic aneurysms. Radiology 2001;220:492-7.
1197. Sternbergh WC 3rd, Money SR. Hospital cost of endovascular versus open repair of abdominal aortic aneurysms: a multicenter study. J Vasc Surg 2000;31:237-44.
1198. Carpenter JP, Baum RA, Barker CF, et al. Durability of benefits of endovascular versus conventional abdominal aortic aneurysm repair. J Vasc Surg 2002;35:222-8.
1199. Bertges DJ, Zwolak RM, Deaton DH, et al. Current hospital costs and medicare reimbursement for endovascular abdominal aortic aneurysm repair. J Vasc Surg 2003;37:272-9.
1200. Arko FR, Hill BB, Reeves TR, et al. Early and late functional outcome assessments following endovascular and open aneurysm repair. J Endovasc Ther 2003;10:2-9.
1201. Schermerhorn ML, Finlayson SR, Fillinger MF, et al. Life expectancy after endovascular versus open abdominal aortic aneurysm repair: results of a decision analysis model on the basis of data from EUROSTAR. J Vasc Surg 2002;36:1112-20.
1202. Cuypers P, Buth J, Harris PL, et al. Realistic expectations for patients with stent-graft treatment of abdominal aortic aneurysms: results of a European multicentre registry. Eur J Vasc Endovasc Surg 1999;17:507-16.
1203. Ohki T, Veith FJ, Shaw P, et al. Increasing incidence of midterm and long-term complications after endovascular graft repair of abdominal aortic aneurysms: a note of caution based on a 9-year experience. Ann Surg 2001;234:323-34; discussion 334-5.
1204. Ouriel K, Clair DG, Greenberg RK, et al. Endovascular repair of abdominal aortic aneurysms: device-specific outcome. J Vasc Surg 2003;37:991-8.
1205. Cuypers PW, Laheij RJ, Buth J. Which factors increase the risk of conversion to open surgery following endovascular abdominal aortic aneurysm repair? The EUROSTAR collaborators. Eur J Vasc Endovasc Surg 2000;20:183-9.
1206. Laheij RJ, Buth J, Harris PL, et al. Need for secondary interventions after endovascular repair of abdominal aortic aneurysms: intermediate-term follow-up results of a European collaborative registry (EUROSTAR). Br J Surg 2000;87:1666-73.
1207. Conner MS 3rd, Sternbergh WC 3rd, Carter G, et al. Secondary procedures after endovascular aortic aneurysm repair. J Vasc Surg 2002;36:992-6.

1208. Ayerdi J, McLafferty RB, Markwell SJ, et al. Indications and outcomes of AneuRx Phase III trial versus use of commercial AneuRx stent graft. J Vasc Surg 2003;37:739-43.
1209. Sternbergh WC, Nordness PJ, York JW, et al. Endo-exuberance to endo-reality: trends in the management of 431 AAA repairs between 1996 and 2002. J Endovasc Ther 2003;10:418-23.
1210. Zarins CK, Bloch DA, Crabtree T, et al. Aneurysm enlargement following endovascular aneurysm repair: AneuRx clinical trial. J Vasc Surg 2004;39:109-17.
1211. Laheij RJ, van Marrewijk CJ; EUROSTAR Group. The evolving technique of endovascular stenting of abdominal aortic aneurysm; time for reappraisal. Eur J Vasc Endovasc Surg 2001; 22:436-42.
1212. Ouriel K, Srivastava SD, Sarac TP, et al. Disparate outcome after endovascular treatment of small versus large abdominal aortic aneurysm. J Vasc Surg 2003;37:1206-12.
1213. Scheinert D, Schroder M, Steinkamp H, et al. Treatment of iliac artery aneurysms by percutaneous implantation of stent grafts. Circulation 2000;102(19 suppl 3):III253-8.
1214. Hausegger KA, Mendel H, Tiessenhausen K, et al. Endoluminal treatment of infrarenal aortic aneurysms: clinical experience with the Talent stent-graft system. J Vasc Interv Radiol 1999; 10:267-74.
1215. Howell MH, Zaqqa M, Villareal RP, et al. Endovascular exclusion of abdominal aortic aneurysms: initial experience with stent-grafts in cardiology practice. Tex Heart Inst J 2000; 27:136-45.
1216. Criado FJ, Wilson EP, Fairman RM, et al. Update on the Talent aortic stent-graft: a preliminary report from United States phase I and II trials. J Vasc Surg 2001;33(2 suppl):S146-9.
1217. Valentine RJ, Decaprio JD, Castillo JM, et al. Watchful waiting in cases of small abdominal aortic aneurysms: appropriate for all patients? J Vasc Surg 2000;32:441-8; discussion 448-50.
1218. Zarins CK, Shaver DM, Arko FR, et al. Introduction of endovascular aneurysm repair into community practice: initial results with a new Food and Drug Administration-approved device. J Vasc Surg 2002;36:226-32; discussion 232-3.
1219. Trastek VF, Pairolero PC, Joyce JW, et al. Splenic artery aneurysms. Surgery 1982;91:694-9.
1220. Cohen JR, Shamash FS. Ruptured renal artery aneurysms during pregnancy. J Vasc Surg 1987;6:51-9.
1221. Ohta M, Hashizume M, Tanoue K, et al. Splenic hyperkinetic state and splenic artery aneurysm in portal hypertension. Hepatogastroenterology 1992;39:529-32.
1222. Kobori L, van der Kolk MJ, de Jong KP, et al. Splenic artery aneurysms in liver transplant patients. Liver Transplant Group. J Hepatol 1997;27:890-3.
1223. Lee PC, Rhee RY, Gordon RY, et al. Management of splenic artery aneurysms: the significance of portal and essential hypertension. J Am Coll Surg 1999;189:483-90.
1224. Carmeci C, McClenathan J. Visceral artery aneurysms as seen in a community hospital. Surg Gynecol Obstet 2000;179:486-9.
1225. Carr SC, Mahvi DM, Hoch JR, et al. Visceral artery aneurysm rupture. J Vasc Surg 2001;33:806-11.
1226. Stone WM, Abbas M, Cherry KJ, et al. Superior mesenteric artery aneurysms: is presence an indication for intervention? J Vasc Surg 2002;36:234-7; discussion 237.
1227. Tham G, Ekelund L, Herrlin K, et al. Renal artery aneurysms: natural history and prognosis. Ann Surg 1983;197:348-52.
1228. Henriksson C, Bjorkerud S, Nilson AE, et al. Natural history of renal artery aneurysm elucidated by repeated angiography and pathoanatomical studies. Eur Urol 1985;11:244-8.
1229. Hallett JW Jr. Splenic artery aneurysms. Semin Vasc Surg 1995; 8:321-6.
1230. Kasirajan K, Greenberg RK, Clair D, et al. Endovascular management of visceral artery aneurysm. J Endovasc Ther 2001; 8:150-5.
1231. Angelakis EJ, Bair WE, Barone JE, et al. Splenic artery aneurysm rupture during pregnancy. Obstet Gynecol Surv 1993;48:145-8.
1232. Salam TA, Lumsden AB, Martin LG, et al. Nonoperative management of visceral aneurysms and pseudoaneurysms. Surg Gynecol Obstet 1992;164:215-9.
1233. Carr SC, Pearce WH, Vogelzang RL, et al. Current management of visceral artery aneurysms. Surgery 1996;120:627-33; discussion 633-4.
1234. Sandgren T, Sonesson B, Ahlgren R, et al. The diameter of the common femoral artery in healthy human: influence of sex, age, and body size. J Vasc Surg 1999;29:503-10.
1235. Graham LM, Zelenock GB, Whitehouse WM Jr, et al. Clinical significance of arteriosclerotic femoral artery aneurysms. Arch Surg 1980;115:502-7.
1236. Whitehouse WM Jr, Wakefield TW, Graham LM, et al. Limb-threatening potential of arteriosclerotic popliteal artery aneurysms. Surgery 1983;93:694-9.
1237. MacSweeney ST, Skidmore C, Turner RJ, et al. Unravelling the familial tendency to aneurysmal disease: popliteal aneurysm, hypertension and fibrillin genotype. Eur J Vasc Endovasc Surg 1996;12:162-6.
1238. Lawrence PF, Lorenzo-Rivero S, Lyon JL. The incidence of iliac, femoral, and popliteal artery aneurysms in hospitalized patients. J Vasc Surg 1995;22:409-15; discussion 415-6.
1239. van Keulen CJ, van de Akker E, Pals G, et al. The role of type III collagen in the development of familial abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 1999;18:65-70.
1240. Lanne T, Hansen F, Mangell P, et al. Differences in mechanical properties of the common carotid artery and abdominal aorta in healthy males. J Vasc Surg 1994;20:218-25.
1241. Makita S, Ohira A, Tachieda R, et al. Dilation and reduced distensibility of carotid artery in patients with abdominal aortic aneurysms. Am Heart J 2000;140:297-302.
1242. Tilson MD, Dang C. Generalized arteriomegaly: a possible predisposition to the formation of abdominal aortic aneurysms. Arch Surg 1981;116:1030-2.
1243. Ward AS. Aortic aneurysmal disease: a generalized dilating diathesis. Arch Surg 1992;127:990-1.
1244. Callum KG, Lea Thomas M, Browse NL. A definition of arteriomegaly and the size of arteries supplying the lower limbs. Br J Surg 1983;70:524-9.
1245. Duffy ST, Colgan MP, Sultan S, et al. Popliteal aneurysms: a 10-year experience. Eur J Vasc Endovasc Surg 1998;16:218-22.
1246. Taurino M, Calisti A, Grossi R, et al. Outcome after early treatment of popliteal artery aneurysms. Int Angiol 1998;17:28-33.
1247. Baxter BT, McGee GS, Flinn WR, et al. Distal embolization as a presenting symptom of aortic aneurysms. Surg Gynecol Obstet 1990;160:197-201.
1248. Gifford RW Jr, Hines EA Jr, Janes JM. An analysis and follow-up study of one hundred popliteal aneurysms. Surgery 1953;33:284-93. 9
1249. Dawson I, Sie RB, van Bockel JH. Atherosclerotic popliteal aneurysm. Br J Surg 1997;84:293-9.
1250. Dawson I, van Bockel JH, Brand R, et al. Popliteal artery aneurysms. Long-term follow-up of aneurysmal disease and results of surgical treatment. J Vasc Surg 1991;13:398-407.

1251. Dawson I, Sie R, van Baalen JM, et al. Asymptomatic popliteal aneurysm: elective operation versus conservative follow-up. Br J Surg 1994;81:1504-7.
1252. Lowell RC, Gloviczki P, Hallett JW Jr, et al. Popliteal artery aneurysms: the risk of nonoperative management. Ann Vasc Surg 1994;8:14-23.
1253. Schroder A, Gohlke J, Gross-Fengels W, et al. [Popliteal aneurysms—surgical management versus conservative procedure] Langenbecks Arch Chir Suppl Kongressbd 1996;113:857-63.
1254. Roggo A, Brunner U, Ottinger LW, et al. The continuing challenge of aneurysms of the popliteal artery. Surg Gynecol Obstet 1993;177:565-72.
1255. Szilagyi DE, Schwartz RL, Reddy DJ. Popliteal arterial aneurysms: their natural history and management. Arch Surg 1981;116:724-8.
1256. Poirier NC, Verdant A, Page A. [Popliteal aneurysm: surgical treatment is mandatory before complications occur] Ann Chir 1996;50:613-8.
1257. Stiegler H, Mendler G, Baumann G. Prospective study of 36 patients with 46 popliteal artery aneurysms with non-surgical treatment. Vasa 2002;31:43-6.
1258. Jivegard L, Holm J, Bergqvist D, et al. Acute lower limb ischemia: failure of anticoagulant treatment to improve one-month results of arterial thromboembolectomy: a prospective randomized multi-center study. Surgery 1991;109:610-6.
1259. Jarrett F, Makaroun MS, Rhee RY, et al. Superficial femoral artery aneurysms: an unusual entity? J Vasc Surg 2002;36:571-4.
1260. Cutler BS, Darling RC. Surgical management of arteriosclerotic femoral aneurysms. Surgery 1973;74:764-73.
1261. Roseman JM, Wyche D. True aneurysm of the profunda femoris artery. Literature review, differential diagnosis, management. J Cardiovasc Surg (Torino) 1987;28:701-5.
1262. Levi N, Schroeder TV. Blood transfusion requirement in surgery for femoral artery aneurysms. J Cardiovasc Surg (Torino) 1997; 38:661-3.
1263. Levi N, Schroeder TV. Arteriosclerotic femoral artery aneurysms: a short review. J Cardiovasc Surg (Torino) 1997; 38:335-8.
1264. Defraigne JO, Limet R. [An unusual presentation of an aortic abdominal aneurysm, source of diagnostic errors: chronic rupture] Rev Med Liege 1997;52:535-40.
1265. Farina C, Cavallaro A, Schultz RD, et al. Popliteal aneurysms. Surg Gynecol Obstet 1989;169:7-13.
1266. Anton GE, Hertzer NR, Beven EG, et al. Surgical management of popliteal aneurysms. Trends in presentation, treatment, and results from 1952 to 1984. J Vasc Surg 1986;3:125-34.
1267. Cole CW, Thijssen AM, Barber GG, et al. Popliteal aneurysms: an index of generalized vascular disease. Can J Surg 1989;32:65-8.
1268. Inahara T, Toledo AC. Complications and treatment of popliteal aneurysms. Surgery 1978;84:775-83.
1268a. Lilly MP, Flinn WR, McCarthy WJ, III, et al. The effect of distal arterial anatomy on the success of popliteal aneurysm repair. J Vasc Surg 1988;7:653-60.
1269. Reilly MK, Abbott WM, Darling RC. Aggressive surgical management of popliteal artery aneurysms. Surg Gynecol Obstet 1983;145:498-502.
1270. Schellack J, Smith RB 3rd, Perdue GD. Nonoperative management of selected popliteal aneurysms. Arch Surg 1987;122:372-5.
1271. Towne JB, Thompson JE, Patman DD, et al. Progression of popliteal aneurysmal disease following popliteal aneurysm resection with graft: a twenty year experience. Surgery 1976; 80:426-32.
1272. Graham L. Femoral and popliteal aneurysms. In: Rotherford RB, ed. Vascular Surgery. 5th ed. Philadelphia, Pa: WB Saunders; 2000:1345-56.
1273. Adiseshiah M, Bailey DA. Aneurysms of the femoral artery. Br J Surg 1977;64:174-6.
1274. Baird RJ, Gurry JF, Kellam J, et al. Arteriosclerotic femoral artery aneurysms. Can Med Assoc J 1977;117:1306-7.
1275. Sapienza P, Mingoli A, Feldhaus RJ, et al. Femoral artery aneurysms: long-term follow-up and results of surgical treatment. Cardiovasc Surg 1996;4:181-84.
1276. Feld R, Patton GM, Carabasi RA, et al. Treatment of iatrogenic femoral artery injuries with ultrasound-guided compression. J Vasc Surg 1992;16:832-40.
1277. Fellmeth BD, Roberts AC, Bookstein JJ, et al. Postangiographic femoral artery injuries: nonsurgical repair with US-guided compression. Radiology 1991;178:671-5.
1278. Johns JP, Pupa LE Jr, Bailey SR. Spontaneous thrombosis of iatrogenic femoral artery pseudoaneurysms: documentation with color Doppler and two-dimensional ultrasonography. J Vasc Surg 1991;14:24-9.
1279. Kazmers A, Meeker C, Nofz K, et al. Nonoperative therapy for postcatheterization femoral artery pseudoaneurysms. Am Surg 1997;63:199-204.
1280. Kresowik TF, Khoury MD, Miller BV, et al. A prospective study of the incidence and natural history of femoral vascular complications after percutaneous transluminal coronary angioplasty. J Vasc Surg 1991;13:328-33; discussion 333-5.
1281. Samuels D, Orron DE, Kessler A, et al. Femoral artery pseudoaneurysm: Doppler sonographic features predictive for spontaneous thrombosis. J Clin Ultrasound 1997;25:497-500.
1282. Schaub F, Theiss W, Busch R, et al. Management of 219 consecutive cases of postcatheterization pseudoaneurysm. J Am Coll Cardiol 1997;30:670-5.
1283. Toursarkissian B, Allen BT, Petrinec D, et al. Spontaneous closure of selected iatrogenic pseudoaneurysms and arteriovenous fistulae. J Vasc Surg 1997;25:803-8; discussion 808-9.
1284. Weatherford DA, Taylor SM, Langan EM, et al. Ultrasound-guided compression for the treatment of iatrogenic femoral pseudoaneurysms. South Med J 1997;90:223-6.
1285. Chatterjee T, Do DD, Mahler F, et al. A prospective, randomized evaluation of nonsurgical closure of femoral pseudoaneurysm by compression device with or without ultrasound guidance. Catheter Cardiovasc Interv 1999;47:304-9.
1286. Coghlan JG, Cowell R, Jepson N, et al. Simplified method for compression of femoral false aneurysms. Eur Heart J 1995; 16:1589-92.
1287. Cox GS, Young JR, Gray BR, et al. Ultrasound-guided compression repair of postcatheterization pseudoaneurysms: results of treatment in one hundred cases. J Vasc Surg 1994;19:683-6.
1288. Dean SM, Olin JW, Piedmonte M, et al. Ultrasound-guided compression closure of postcatheterization pseudoaneurysms during concurrent anticoagulation: a review of seventy-seven patients. J Vasc Surg 1996;23:28-34, discussion 34-5.
1289. Hajarizadeh H, LaRosa CR, Cardullo P, et al. Ultrasound-guided compression of iatrogenic femoral pseudoaneurysm failure, recurrence, and long-term results. J Vasc Surg 1995;22:425-30; discussion 430-3.
1290. Hertz SM, Brener BJ. Ultrasound-guided pseudoaneurysm compression: efficacy after coronary stenting and angioplasty. J Vasc Surg 1997;26:913-6; discussion 916-8.
1291. Kumins NH, Landau DS, Montalvo J, et al. Expanded indications for the treatment of postcatheterization femoral pseudoa-

neurysms with ultrasound-guided compression. Surg Gynecol Obstet 1998;176:131-6.

1292. Langella RL, Schneider JR, Golan JF. Color duplex-guided compression therapy for postcatheterization pseudoaneurysms in a community hospital. Ann Vasc Surg 1996;10:27-35.

1293. Paulson EK, Kliewer MA, Hertzberg BS, et al. Ultrasonographically guided manual compression of femoral artery injuries. J Ultrasound Med 1995;14:653-9.

1294. Perkins JM, Gordon AC, Magee TR, et al. Duplex-guided compression of femoral artery false aneurysms reduces the need for surgery. Ann R Coll Surg Engl 1996;78:473-5.

1295. Sorrell KA, Feinberg RL, Wheeler JR, et al. Color-flow duplex-directed manual occlusion of femoral false aneurysms. J Vasc Surg 1993;17:571-7.

1296. Steinkamp HJ, Werk M, Felix R. Treatment of postinterventional pseudoaneurysms by ultrasound-guided compression. Invest Radiol 2000;35:186-92.

1297. Edgerton JR, Moore DO, Nichols D, et al. Obliteration of femoral artery pseudoaneurysm by thrombin injection. Ann Thorac Surg 2002;74:S1413-5.

1298. Olsen DM, Rodriguez JA, Vranic M, et al. A prospective study of ultrasound scan-guided thrombin injection of femoral pseudoaneurysm: a trend toward minimal medication. J Vasc Surg 2002;36:779-82.

1299. La Perna L, Olin JW, Goines D, et al. Ultrasound-guided thrombin injection for the treatment of postcatheterization pseudoaneurysms. Circulation 2000;102:2391-5.

1300. Mohler ER III, Mitchell ME, Carpenter JP, et al. Therapeutic thrombin injection of pseudoaneurysms: a multicenter experience. Vasc Med 2001;6:241-4.

1301. Friedman SG, Pellerito JS, Scher L, et al. Ultrasound-guided thrombin injection is the treatment of choice for femoral pseudoaneurysms. Arch Surg 2002;137:462-4.

1302. Lonn L, Olmarker A, Geterud K, et al. Treatment of femoral pseudoaneurysms. Percutaneous US-guided thrombin injection versus US-guided compression. Acta Radiol 2002;43:396-400.

1303. Hughes MJ, McCall JM, Nott DM, et al. Treatment of iatrogenic femoral artery pseudoaneurysms using ultrasound-guided injection of thrombin. Clin Radiol 2000;55:749-51.

1304. Kang SS, Labropoulos N, Mansour MA, et al. Expanded indications for ultrasound-guided thrombin injection of pseudoaneurysms. J Vasc Surg 2000;31:289-98.

1305. Liau CS, Ho FM, Chen MF, et al. Treatment of iatrogenic femoral artery pseudoaneurysm with percutaneous thrombin injection. J Vasc Surg 1997;26:18-23.

1306. Reeder SB, Widlus DM, Lazinger M. Low-dose thrombin injection to treat iatrogenic femoral artery pseudoaneurysms. AJR Am J Roentgenol 2001;177:595-8.

1307. Sackett WR, Taylor SM, Coffey CB, et al. Ultrasound-guided thrombin injection of iatrogenic femoral pseudoaneurysms: a prospective analysis. Am Surg 2000;66:937-40; discussion 940-2.

1308. Taylor BS, Rhee RY, Muluk S, et al. Thrombin injection versus compression of femoral artery pseudoaneurysms. J Vasc Surg 1999;30:1052-9.