

# RED BOOK

Revised February 2006

## *Introduction*

This second edition of the Vascular Red Book was written to help you meet the educational goals and objectives for the Vascular Service. Collectively the vascular attendings have reviewed a broad range of topics in peripheral vascular disease. For each topic we list the learning objectives, summarize the most critical information, and provide a list of key references.

Residents are expected to have read the Red Book by the end of each and every Vascular Surgery rotation. The intention of the Red Book is to help prioritize your study and provide a basic knowledge base. It does not take the place of traditional surgical texts and journals. As adult learners, PGY1-5 residents will be expected to be able to access information from specialty texts (such as Rutherford's *Vascular Surgery*, 6th ed.), journals (*Journal of Vascular Surgery*, etc.), and on-line sources to compare practice patterns and outcomes to benchmarks in the literature. This fund of information will give you the tools to succeed on written and oral examinations and to take better care of patients with vascular disease. Medicine is constantly changing and future editions of this book will be necessary for it to remain relevant; we welcome any suggestions that could make it a better source of information.

### **Table of Contents**

Cardiac risk assessment.....	2
Carotid stenosis.....	8
Abdominal aortic aneurysms.....	12
Peripheral arterial aneurysms.....	15
Lower extremity occlusive disease.....	18
Leg ulcers.....	25
Renovascular hypertension.....	27
Mesenteric occlusive disease.....	30
Peripheral vascular trauma.....	36
The mangled extremity.....	39
Thrombolytic therapy.....	41
Venous disease.....	43
Thoracic outlet syndrome.....	52
The noninvasive vascular lab.....	54
Arterial imaging modalities.....	60

# Cardiac Risk Assessment

---

by Daniel J Bertges, MD

## Learning Objectives

### I. Scope of the Problem

1. Recognize the common association of coronary artery disease (CAD) and peripheral vascular disease (PVD).
2. Know the statistics of the frequency of severe CAD in patients with symptomatic PVD.
3. Understand the risk factors predictive of perioperative myocardial infarction (MI).
4. Be familiar with the early and late cardiac mortality rates following major vascular surgery.

### II. Anatomy and Pathophysiology

1. Describe normal coronary artery anatomy.
2. Understand the clinical importance of stable/unstable angina, recent/past MI and congestive heart failure (CHF).
3. Understand the clinical and pathological difference between a subendocardial and transmural MI.
4. Understand the most important factors intraoperatively and post-operatively that contribute to myocardial ischemia. (i.e., increased demand for myocardial oxygen, catecholamine release).
5. Realize that *nonhemodynamically* significant coronary lesions can cause MI.

### III. Diagnosis

1. Know the risks of operation in a patient with a recent MI, unstable angina, or decompensated CHF.
2. Begin to develop an understanding of which patients should undergo a preoperative test for CAD.
3. Be familiar with provocative cardiac tests and their limits (exercise testing, dipyridamole thallium testing, dobutamine stress echo).
4. Know how to interpret the results of thallium scans.
5. Understand the need for further evaluation in a patient with a positive stress test.
6. Understand that the type of procedure should be tailored to the severity of the patient's cardiac risk. Understand options for non-operative treatment, percutaneous intervention, extra-anatomic bypass, or a limited procedure instead of an intra-abdominal operation.
9. Understand when vascular patients are most likely to suffer myocardial ischemia.
10. Be aware that MI is more difficult to diagnose in postoperative patients and understand the use of the EKG and cardiac enzymes in making the diagnosis.

### IV. Treatment

1. Recognize that most patients with even severe CAD can survive a major vascular operation, but need cardiology consultation, careful anesthesia technique, and maximum medical therapy.

2. Acknowledge the importance of antiplatelet therapy and beta-blockade in reducing cardiac events after vascular surgery.
3. Know how to detect and treat myocardial ischemia postoperatively.
4. Know how to diagnose and treat common complications of MI (monitoring for arrhythmia).

**Scope of the problem.** Coronary artery disease (CAD) is extremely common in patients with peripheral vascular disease. In a landmark study by Hertzner, coronary angiography was performed in 1000 patients (roughly divided into thirds by carotid, aortic, and infrainguinal disease) under consideration for elective vascular reconstruction. In this study, hemodynamically significant CAD was found in 36% of patients with AAA, 28% of patients with lower extremity ischemia, and 32% of patients with carotid stenosis. Severe, correctable CAD was identified in 25% of the entire series while only 8% had normal coronary anatomy. The reportedly high prevalence of CAD in vascular patients has led to numerous algorithms (i.e., Goldman, Eagle criteria) for its evaluation and management. While much controversy still remains, practice guidelines exist for the clinical risk stratification, preoperative cardiac testing, and decision making for coronary revascularization.

While numerous untoward cardiac events (arrhythmia, CHF, etc.) may occur after surgery, nonfatal and fatal MIs are the most important “hard” bad outcomes that can be prevented. Myocardial ischemia occurs in 20% - 40% of vascular surgery patients, and more than half of these patients develop clinically significant events. Table 1 shows the incidence of MI for the 3 most common procedures. Surprisingly the incidence of MI is highest in the LE bypass group. The same is true of late cardiac events, which are twice as common in patients with LE PVD compared to aortic procedures 2 years after surgery.

**Table 1.** Incidence of perioperative nonfatal and fatal myocardial infarction after common vascular procedures.

Procedure	Nonfatal MI	Fatal MI
Elective AAA	2.2%	1.4%
LE revascularization	4%	1.8%
CEA	1%	0.4%

*From Rutherford. Vascular Surgery, 6<sup>th</sup> ed.*

**Diagnosis of perioperative MI.** The traditional criteria for diagnosis of acute MI require 2 of the following 3: (1) prolonged chest pain, (2) EKG changes, and (3) positive serial cardiac enzymes. The typical symptoms of MI may not be present in 75% of postoperative patients due to numerous factors (for example, incisional pain, narcotics, or diabetes). Knowing this, the diagnosis often rests on cardiac enzymes. Creatine phosphokinase (CPK) is released from skeletal muscle after surgery and can hide the CK-MB fraction released from dying heart muscle. The troponins (C, T, and I) are normal muscle proteins involved in calcium-regulated, actin-myosin interaction. Troponins I and T are distinct cardiac-subtypes. Troponin I only exists in cardiac tissue and is not present in the blood of healthy people or patients with renal failure. Andrews et al. found that the presence of troponin I is accurate in detecting cardiac ischemia after vascular surgery. Antman summarized the use of troponin testing in

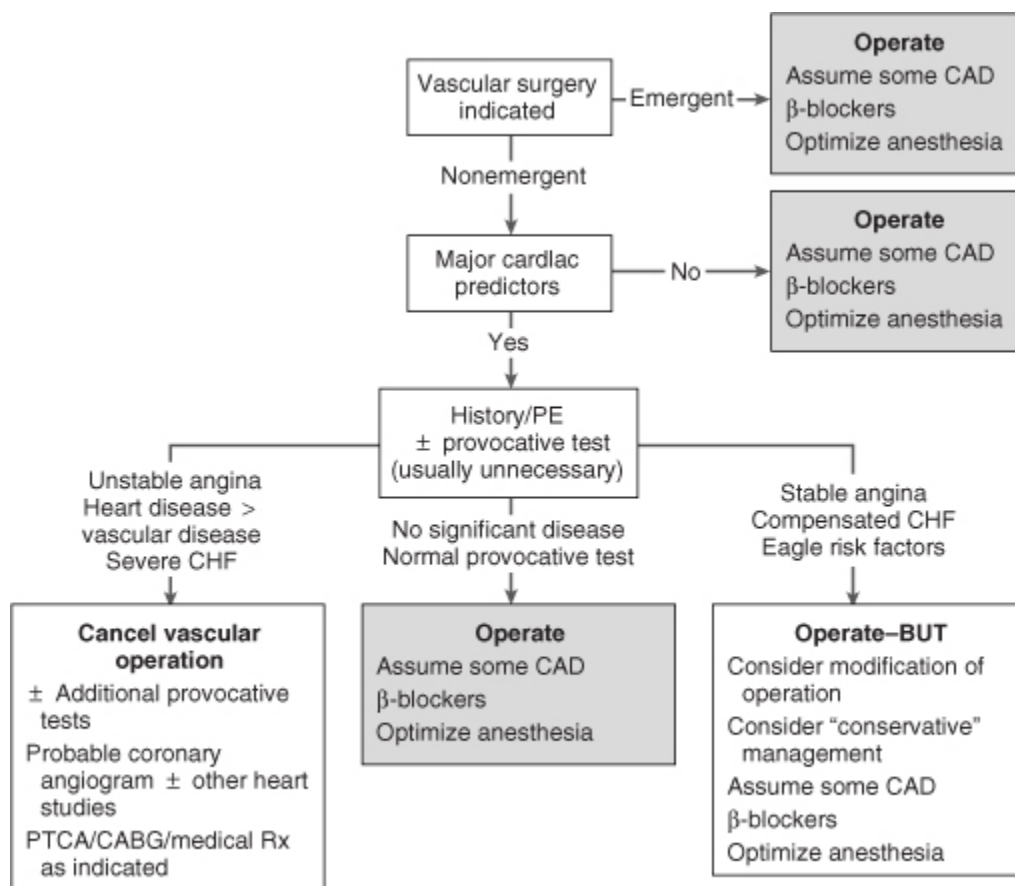
a useful review. Some clinicians question the importance of “chemical MIs” or “troponin leaks”. The medical literature reports higher rates of early and late ischemic complications, such as reinfarction and postinfarction angina, in these patients. For this reason even non-Q-wave MIs have significance for your patients.

*The diagnosis of perioperative MI requires a high clinical suspicion with an understanding of the atypical presentation and liberal use of EKGs and troponin testing.*

**Pathophysiology of MI.** It is now accepted that primary MIs in ambulatory patients are often caused by non-hemodynamically significant stenosis (less than 50% lesions). Cardiac events may result from disrupted atherosclerotic plaques without severe stenosis. Because there are no tests to identify the exact lesion that will cause an MI, we rely on noninvasive tests to screen for hemodynamically significant disease. Detection of severe coronary stenosis is still important but may be more of a marker for the degree of “atherosclerotic burden” than predictive of the location of potential perioperative myocardial ischemia.

**Guidelines for cardiac work-up.** In 2002 the American Heart Association issued updated guidelines for perioperative cardiovascular evaluation for noncardiac surgery. While comprehensive, these guidelines are complex and difficult to apply to clinical practice. A more practical algorithm suggested by Krupski appears in Figure 1. Your evaluation starts with the history and exam to identify Eagle criteria. Eagle criteria include: (1) age > 70, (2) MI by history or Q wave on EKG, (3) angina, (4) history of CHF, (5) diabetes, and (6) ventricular ectopy requiring therapy. Provocative cardiac testing then follows. Options include exercise treadmill testing (many vascular patients cannot do this), dipyridamole-thallium scintigraphy, dobutamine stress echocardiography, or sestamibi stress echocardiography. A meta-analysis comparing dipyridamole-thallium imaging and dobutamine echocardiography for risk stratification before vascular surgery showed the following: low risk of MI in patients without a history of CAD (1%), intermediate risk in patients with CAD and a normal or fixed-deficit pattern (5%), and high risk with one or more areas of ischemia (19%).

**If severe, reconstructible CAD is found how should you proceed?** The CARP (Coronary Artery Revascularization Prophylaxis) Study performed by VA hospitals questioned the effectiveness of routine coronary revascularization prior to elective vascular surgery. CARP randomized 510 patients (1/3 with expanding AAA and 2/3 with symptomatic LE occlusive disease) with stable CAD to coronary revascularization by PTCA or CABG in one group; the other group had no preop revascularization. Important exclusions included left main disease, LVEF <20% and severe aortic stenosis. The primary endpoint was long-term mortality. During the 30-day period after vascular surgery, there was no difference in death or MI rate between groups. At a median of 2.7 years, there was no difference in mortality rate between the revascularization and no-revascularization groups (22% vs. 23%). Vascular surgery was delayed significantly in the revascularization group. CARP concluded that preoperative coronary-artery revascularization *does not improve long-term survival* after elective vascular surgery in patients with stable CHD and risk factors for cardiac complications and should not be recommended for this patient population. At a minimum, caution is advised in the widespread use of coronary revascularization before elective vascular surgery. Patients with PVD are at higher risk for complications after CABG or PTCA. In 2005, the trend is toward maximizing medical management and going away from reflex revascularization of the heart.



Copyright ©2005, 2000, 1995, 1989, 1976 by Elsevier, Inc.

**Figure 1.** Cardiac risk assessment algorithm. *From: Vascular Surgery 6/e (on 16 November 2005) © 2005 Elsevier.*

**Perioperative medical management.** A large body of literature suggests that perioperative beta-blockade can reduce cardiac events after non-cardiac vascular surgery (Table 2). The stress response to surgery involves adrenal cortex stimulation with an increase in catecholamines. Catecholamines increase myocardial oxygen consumption by affecting heart rate, preload, afterload, and contractility. It is not enough to simply place patients on beta-blockers. You must pay attention to the heart rate and titrate the drug accordingly.

**Table 2.** Controlled, Randomized Trials of Perioperative Beta Blockade.

AUTHOR	YEAR	No.	DRUG	FOLLOW-UP	CONTROL	BETA BLOCKERS
Stone	1988	128	Control = 39; labetalol =29; atenolol= 30; oxprenolol = 30	Intraoperative	28% Ischemia	2% Ischemia <i>P</i> <.001
Mangano	1996	200	Control = 101; atenolol= 99	2 years	21% Mortality	10% Mortality <i>P</i> <.019
Poldermans	1999	112	Control = 53; bisoprolol = 59	30 days	17% Cardiac death	3.4% Cardiac death <i>P</i> = .02
Raby	1999	26	Control = 11; esmolol = 15	48 hours	73% Persist ischemia	33% Persist ischemia <i>P</i> <.05
Urban	2000	107	Control = 55; esmolol = 52	48 hours	15% Ischemia; 6% MI; 16% cardiac morbidity	6% Ischemia; 2% MI; 11% cardiac morbidity; <i>P</i> = NS
Poldermans	2001	101	Control = 44; bisoprolol = 57	2 years	32% Cardiac events	12% Cardiac events <i>P</i> = .025

From Rutherford. *Vascular Surgery, 6<sup>th</sup> edition*

**Summary.** Cardiac risk assessment for vascular surgery is complex. To keep it simple it is safe to assume that the patient has some degree of CAD and will benefit from medical therapy. Judicious and thoughtful use of preoperative tests and selective application of coronary revascularization may further reduce the incidence of perioperative MI and early and late cardiac death.

## References

- Hertzer NR, Beven EG, Young JR, et al. Coronary artery disease in peripheral vascular patients: A classification of 1000 coronary angiograms and results of surgical management. *Ann Surg* 1984;199:223-33.
- Goldman L. Cardiac risks and complications of noncardiac surgery. *Ann Intern Med* 1983;98:504-13.
- Andrews N, Jenkins J, Andrews G, Walker P: Using postoperative cardiac Troponin-I (cTi) levels to detect myocardial ischaemia in patients undergoing vascular surgery. *Cardiovasc Surg* 2001;9:254-265.
- Antman EM: Decision making with cardiac troponin tests. *N Engl J Med* 2002;346:2079-2082.
- Eagle KA, Berger PB, Calkins H, et al: ACC/AHA guideline update for 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;105:1257-1267.
- Mangano DT, Goldman L. Preoperative assessment of patients with known or suspected coronary disease. *N Engl J Med* 1995 ;333:1750-56.
- Shaw LJ, Eagle KA, Gersh BJ, Miller DD: Meta-analysis of intravenous dipyridamole-thallium-201 imaging (1985 to 1994) and dobutamine echocardiography (1991 to 1994) for risk stratification before vascular surgery. *J Am Coll Cardiol* 1996;27:787-798.
- McFalls EO, Ward HB, Moritz TE et al: Coronary-artery revascularization before elective major vascular surgery. *N Engl J Med* 2004;351:2795-2804.

9. Stone JG, Foex P, Sear JW, et al: Myocardial ischemia in untreated hypertensive patients: Effect of a single small oral dose of a beta-adrenergic blocking agent. *Anesthesiology* 1988;68:495-500.
10. Mangano DT, Layug EL, Wallace A, Tateo I: Effect of atenolol on mortality and cardiovascular morbidity after noncardiac surgery: Multicenter Study of Perioperative Ischemia Research Group. *N Engl J Med* 1996;335:1713-1720.
11. Poldermans D, Boersma E, Bax JJ, et al: The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography Study Group. *N Engl J Med* 1999;341:1789-1794.
12. Raby KE, Brull SJ, Timimi F, et al: The effect of heart rate control on myocardial ischemia among high-risk patients after vascular surgery. *Anesth Analg* 1999;88:477-482.
13. Urban MK, Markowitz SM, Gordon MA, et al: Postoperative prophylactic administration of beta-adrenergic blockers in patients at risk for myocardial ischemia. *Anesth Analg* 2000;90:1257-1261.
14. Poldermans D, Boersma E, Bax JJ, et al: Bisoprolol reduces cardiac death and myocardial infarction in high-risk patients as long as 2 years after successful major vascular surgery. *Eur Heart J* 2001;22:1353-1358.
15. Ryan TJ, Anderson JL, Antman EM, et al: ACC/AHA guidelines for the management of patients with acute myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol* 1996;28:1328-1428.
16. 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;308:1540.
17. Parker WR, Leeper NJ, Kirkpatrick JN, Lang RM, Sorrentino MJ, Williams KA. The effect of preoperative statin therapy on cardiovascular outcomes in patients undergoing infrainguinal vascular surgery. *Int J Cardiol.* 2005;104:264-8.

# ***Carotid Stenosis***

---

by Daniel J Bertges, MD

## **Learning Objectives**

### **I. Anatomy and Pathophysiology**

1. Know the anatomy of the arch, great vessels, and intracranial arteries.
2. Know the collateral circulation of the extracranial and intracranial arteries.
3. Understand the different etiologies of carotid artery disease.
  - a. Atherosclerosis
  - b. Fibromuscular dysplasia
  - c. Traumatic occlusion
  - d. Acute dissection
  - e. Vasculitis
4. Discuss the mechanism of cerebral events in terms of occlusion and/or embolism vs. cerebral hypoperfusion.

### **II . Diagnosis**

1. Understand difference between hemispheric, non-hemispheric, and non-specific symptoms.
2. Differentiate among transient ischemic attack (TIA) and stroke/cerebrovascular accident (CVA).
3. Realize the pros and cons of carotid duplex examination
4. Be aware of alternative imaging modalities (MRA, CTA, angiography) including the risk of cerebral angiography.

### **III . Treatment**

1. Know the results of key trials of carotid endarterectomy (CEA) in asymptomatic (ACAS) and symptomatic patients (NASCET).
2. Describe the relationship between the severity of carotid stenosis and decision to treat asymptomatic and symptomatic patients.

3. Describe the standard approach to carotid endarterectomy, including anesthetic techniques and monitoring, intraoperative shunting, patching, and methods of completion evaluation.
4. Understand and manage the complications of CEA, including: neck hematoma, infection, post-operative hyper- and hypotension, cranial nerve palsies, TIA and stroke, reperfusion injury with intracranial hemorrhage or post-operative seizure, and myocardial ischemia.
5. Discuss the potential role of carotid angioplasty and stenting under cerebral protection.

**Pathophysiology.** Stroke is the third most common cause of death in the United States each year. Extracranial carotid artery stenosis accounts for an estimated 10%-25% of strokes in the US, depending on the population studied. Carotid stenosis is due to atherosclerotic disease of the carotid bifurcation in 90% of cases. The mechanism of cerebral ischemia from carotid stenosis is believed to be due to emboli originating from the carotid plaque. These emboli may consist of atheromatous debris, platelet aggregates, or thrombus. Other causes such as cerebral hypoperfusion from a flow-limiting carotid lesion, while possible, are less common given the brain's capacity to autoregulate.

**Diagnosis.** The condition of patients with carotid stenosis is classified by the presence or absence of symptoms and degree of stenosis. Typical presentation of carotid distribution cerebral ischemia results in lateralizing, anterior circulation symptoms which include:

1. ipsilateral monocular blindness (amaurosis fugax)
2. contralateral motor weakness
3. contralateral paresthesias
4. speech abnormality (aphasia)

Nonlateralizing ischemic attacks, such as dizziness, vertigo, ataxia, or syncope, usually represent symptoms associated with brain stem or posterior circulation ischemia and are usually not due to carotid stenosis.

Symptoms are categorized as a TIA if they completely resolve within 24 hours of onset or as a CVA if they persist beyond 24 hours.

The degree of stenosis can be measured by one of several imaging modalities including ultrasound, CT or MR angiography, or cerebral arteriography. In our practice a technically good duplex performed by an accredited laboratory is sufficient to make decisions for intervention for most patients. Cerebral angiography carries up to 1% risk of stroke.

**Treatment of carotid stenosis.** Management options range from maximizing medical therapy, to carotid artery angioplasty and stenting (CAS), to CEA. The first strategy is to stabilize or halt the progression of carotid stenosis through modification of risk factors such as hypertension, hypercholesterolemia, and smoking. Antiplatelet therapy has been proven to reduce the risk of stroke from carotid stenosis, while the benefit of oral anticoagulation has not been proven.

Level 1 evidence exists to guide decision making for surgery. The benefit of CEA for symptomatic carotid stenosis was established by 3 randomized trials: North American Symptomatic Carotid Endarterectomy Trial (NASCET), European Carotid Surgery Trial (ECST) and the VA Cooperative Studies Program. The Asymptomatic Carotid Atherosclerosis Study (ACAS) showed the benefit of CEA in preventing ipsilateral CVA at 5 years for asymptomatic patient with >60% stenosis. The most recent Asymptomatic Carotid Surgery Trial (ACST) reported similar results but used a different endpoint of death and any stroke out to 5 years.

The relevant studies are summarized in Table 1. The number needed to treat (NNT) represents the number of CEAs that must be done to prevent 1 CVA and is a useful way of judging the potential benefit of CEA.

**Table 1.** Risk of ipsilateral stroke with best medical therapy vs. CEA for symptomatic and asymptomatic carotid stenosis. \* = statistically significant, NS= not statistically significant

Study	Medical Rx	CEA	NNT
NASCET >70%	26	9*	6
NASCET 50-69%	22	16*	15
NASCET <50%	19	15 (NS)	--
ECST >70%	17	10 (NS)	7-8
VA #309	19	8 (NS)	--
ACAS >60%	11	5*	17
ACST >60%	12	6*	--

**Carotid Endarterectomy.** Successful CEA requires knowledge of normal extracranial cerebrovascular anatomy: branches of external carotid, relationships between vascular and nervous structures, vagus, hypoglossal, internal jugular, transverse facial vein, omohyoid, sternocleidomastoid, and carotid sinus. Because adverse events are uncommon it is difficult to have a study that is sufficiently powered to prove superiority of one technique over another. Local/regional anesthesia, general anesthesia with non-selective shunting, or selective shunting with EEG monitoring are all reasonable options. A meta-analysis has suggested a benefit of CEA under local anesthesia. A body of literatures suggests that patch closure of the endarterectomy site results in lower 30-d stroke rates and recurrent stenosis. Traditional CEA and eversion endarterectomy appear to be equally effective as shown by EVEREST (EVERsion carotid Endarterectomy versus Standard Trial). Cranial nerve injuries occur in 5%-10% of patients, with the majority being temporary. The benefit of a completion study such as intraoperative duplex or carotid angiography, while commonly practiced, has not been proven.

**Carotid stenting.** Carotid artery angioplasty and stenting is an emerging treatment modality for selected patients with carotid stenosis. At the time of this writing, a single clinical trial (SAPPHIRE) prompted the approval of CAS by the FDA. The Stenting and Angioplasty with Protection in Patients with High Risk for Endarterectomy (SAPPHIRE) trial reported the combined endpoint of major cardiac adverse events (MACE) which included death, stroke, or MI within 30 days and ipsilateral stroke or death within 1 year. The risk of MACE for CAS was 12%, vs. 20% for CEA. Cerebral protection devices (filters, balloon occlusion catheters) are recommended to reduce the incidence of embolic events during the procedure. Future pivotal, randomized studies such as Carotid Revascularization Endarterectomy versus Stent Trial (CREST) are expected to provide more data on the utility of CAS.

## References

1. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial Effect of Carotid Endarterectomy in Symptomatic Patients with High - Grade Stenosis . N Engl J Med 1991;325:445.
  2. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 1998;339:1415-25.
  3. European Carotid Surgery Trialists : Collaborative Group. European Carotid Surgery Trial: Interim Results for Symptomatic Patients with Severe or with Mild Carotid Stenosis. Lancet 1991;337:1235.
  4. Hobson RW II, Weiss DG, Field WS, et al. Efficacy of Carotid Endarterectomy for Asymptomatic Carotid Stenosis: The Veterans Affairs Cooperative Study Group. N Engl J Med 1993;328:221.
  5. Asymptomatic Carotid Atherosclerosis Study (ACAS) Endarterectomy for asymptomatic stenoses JAMA 1995;273:14211-1428.
  6. MRC Asymptomatic Carotid Surgery Trial (ACST) collaborative Group. Prevention of disabling and fatal strokes by successful carotid endarterectomy in patients without recent neurological symptoms: randomized controlled trial. Lancet 2004;363:1491-1502.
  7. Dawson DL, Strandness DE, et al: The role of duplex scanning before carotid endarterectomy: A prospective study. J Vasc Surg 1993;18:673.
  8. Tangkanakul C, Counsell CE, Warlow CP. Local versus general anaesthesia in carotid endarterectomy: a systematic review of the evidence. Eur J Vasc Endovasc Surg. 1997;13:491-9.
  9. Prospective Randomized Trial of CEA with Primary closure and Patch Angioplasty with Saphenous Vein, PTFE patch. J Vasc Surg 1996;24:998.
  10. Cao P, Giordano G, De Rango P, Zannetti S, Chiesa R, Coppi G, Palombo D, Peinetti F, Spartera C, Stancanelli V, Vecchiati E. Eversion versus conventional carotid endarterectomy: Late results of a prospective multicenter randomized trial. J Vasc Surg 2000;31:19-30.
  11. Schaubert MD, Fontenelle LJ, Solomon JW, Hanson TL. Cranial/cervical nerve dysfunction after carotid endarterectomy. J Vasc Surg 1997;25:481-487.
  12. Mackey WC, Belkin M, Sindhi R et al: Routine postendarterectomy duplex surveillance: Does it prevent late stroke? J Vasc Surg 1992;16:34.
  13. Protected Carotid Artery Stenting versus Endarterectomy in High Risk Patients. For the Stenting and Angioplasty with Protection in Patients with High Risk for Endarterectomy (SAPPHIRE) Investigators. N Engl J Med 2004;351:1493-501.
  14. Hobson RW. Carotid artery stenting is associated with increased complications in octogenarians: 30-day stroke and death rates in the CREST lead-in phase. J Vasc Surg 2004;40:1106-11.
- Historic reference** 1. Eastcott HHG, Pickering GW, Robb CG: Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. Lancet 1954;267:994.

# ***Abdominal Aortic Aneurysms***

---

By Andrew C Stanley, MD

## **Learning Objectives**

### **1. Scope of Problem:**

1. To describe the role of aging and atherosclerosis in aortic enlargement.
2. To describe the role of inflammation and proteases in aneurysm formation.
3. To understand the incidence and prevalence of aneurysmal disease according to age.
4. To understand the natural history of abdominal aortic aneurysms.
5. To understand the genetic distribution of the disease.

### **2. Anatomy and Physiology:**

1. To describe aortic architecture and functions.
2. To describe hemodynamic changes at major bifurcation and Laplace's Law.

### **3. Diagnosis:**

1. To understand the roles of the following in screening and in planning surgery: ultrasound, angiography, CT, and MRI/MRA.

### **4. Treatment/Management:**

1. To understand the indications for surgical repair and the factors which contribute to surgical decision making.
2. To understand the technical aspects of aortic aneurysm repair and surgical options and alternatives.
3. To have knowledge of both the immediate and long-term outcomes of surgery for aortic aneurysmal disease (including symptomatic, asymptomatic, thoracoabdominal, juxtarenal, infrarenal, and recurrent).
4. To describe the management and prevention of surgical complications, including spinal cord ischemia, distal embolization, myocardial infarction, graft infection.

## **Pathophysiology:**

Aortic aneurysms are a widening of the normal blood vessel. Aneurysms can be “true” or “false”. False aneurysms are the result of arterial trauma or suture line breakdown and do not involve all layers of the arterial wall. True aneurysms involve all layers of the artery. By definition an aneurysm is the expansion of the vessel diameter to >50% of the artery's normal size. The pathophysiology of true aneurysms is still under investigation but is multifactorial; the following are possible causes:

1. Genetic influences
  - Syndromes with aneurysms include
    - Turner
    - Marfan
    - Ehlers-Danlos
  - Familial clustering

-Patients with AAA have 25% chance of having 1<sup>st</sup> degree relative with AAA (general population 2%-3%)

2. Smoking-associated degeneration
3. Infection/inflammation
  - Observational studies noting reduced elastin content.
  - Syphilis
  - Bacterial

### **Scope of Problem:**

AAA are much more common in males than females (2- to 6-fold difference). Once discovered, small AAAs have an average rate of growth of about 0.5 cm/year. The rupture risk of small aneurysms (<5.0 cm) is about 1% per year. The rupture risk of large aneurysms is higher: 6.5% per year for aneurysms that are 5.0 to 5.9 cm, and 10% per year for aneurysms that are 6.0 to 6.5 cm. Currently, the best way to evaluate the risk of rupture is by size. However, shape, location, and relationship to normal aortic size are thought to also play a role.

### **Diagnosis**

Most patients present with asymptomatic AAA. If symptomatic, these AAA can cause back, abdominal, or groin pain. Abdominal pain with a large or enlarging aorta with no other source of abdominal pain should be attributed to acute expansion or impending rupture of the aneurysm. Rarely, inflammatory aneurysms can present with obstruction of the ureter or bowel. Asymptomatic aneurysms can be found incidentally on physical exam or incidentally on an imaging test.

#### *Imaging*

Screening - exclusively ultrasound

Therapeutic planning - CT scan or occasionally arteriography

Once identified, small aneurysms (<5.0 cm) usually followed with ultrasound 2x/yr.

#### *Risk Stratification*

Risk stratification involves physiologic evaluation of the heart, lungs, and renal function. Various strategies exist for coronary evaluation but in general some type of stress testing is indicated before elective repair. Assessment of high-risk patients involves both overall function (ejection fraction <40%) as well as potential for intra-operative ischemia (positive stress testing).

Pulmonary function is often tested with spirometry. FEV1<1.0 or reduced to below 50% identifies a high-risk patient. Serum creatinine >2.0 is a risk factor for renal failure and a marker for increased mortality.

### **Treatment**

Once a decision is made to repair an AAA, the options are either open or endovascular repair, depending on the aneurysm, and also the history of the patient, co-morbidities, and operative risk. You should be familiar with the following:

Open AAA: mortality, MI, renal failure, long-term graft complications.

Endovascular AAA Repair (EVAR): Mortality, renal failure, technical nuance, endoleaks - type and treatment, long-term graft complications

**References:**

1. Hollier LH, Taylor LM, Oschner J: Recommended indications for operative treatment of AAA. *J Vasc Surg* 1992;15:1046-1056.
2. Ernst CB: Current Concepts: Abdominal Aortic Aneurysms *N Engl J Med* 1993;328:1167-1172.
3. Nevitt MP, Ballard DJ, Hallett JW, Jr: Prognosis of AAA; A population based study. *N Engl J Med* 1989;321:1009-1014.
4. 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;11:260-269.
5. Bernstein EF, Chan EL: AAA in high risk patients; Outcome of selective management based on size and expansion rate. *Ann Surg* 1984;200:255-263.
6. Rehm JP, Grange JJ, Baxter BT: The formation of aneurysms. *Sem in Vasc Surg* 1998;11:193-202.
7. Dobrin PB: Pathophysiology and pathogenesis of abdominal aortic aneurysms. *Current Concepts. Surg Clin North Am* 1989;69:687-703.
9. White GH, Yu W, May J, et al: Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: Classification, incidence, diagnosis, and management. *J Endovasc Surg* 1997;4:152-168.
10. Johnston KW: Multicenter prospective study of nonruptured AAA. Variables predicting morbidity and mortality. *J Vasc Surg* 1989;9:437.
11. Cambria RP, Brewster DC, Abbott WM, et al: Transperitoneal versus retroperitoneal approach for aortic reconstruction: A randomized prospective study. *J Vasc Surg* 11:314,1990
12. 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.
13. Mortality results for randomized controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. The UK Small Aneurysm Trial Participants. *Lancet* 1998;352:1649.
14. 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.
15. EVAR Trial Participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. *Lancet*. 2005;365:2179-86.
16. EVAR Trial Participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): randomised controlled trial. *Lancet* 2005;365:2187-92.
17. Dutch Randomized Endovascular Aneurysm Management Trial Group. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. *N Engl J Med* 2005;352:2398-405.

18. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. *Lancet* 2004;364:843-8.

### **Historic References:**

1. Dubost C, Allary M, Oeconomos N: Resection of an aneurysm of the abdominal aorta: Reestablishment of the continuity by a preserved arterial graft, with result after five months. *Arch Surg* 1952;64:405.
2. Szilagyi DE, Elliott JP, Smith RF: Clinical fate of the patient with asymptomatic AAA and unfit for surgical treatment. *Arch Surg* 1972;104:600-606.
3. Parodi JC, Palmaz JC, Barone HD: Transfemoral intraluminal graft implantation for abdominal aortic aneurysm. *Ann Vasc Surg* 1991;5:491-499.

## **Peripheral Arterial Aneurysm**

By Georg Steinthorsson, MD

### **Learning Objectives:**

#### **I. Scope of Problem:**

- A. Arteriosclerotic femoral artery aneurysms (FAA)
  1. Demographics:
    - a. more common in men; average age at diagnosis is 70 years
    - b. Generally found in pts with diffuse aneurysmal disease of the abdominal aorta and the femoral and popliteal arteries
    - c. 3% of patients with AAA have femoral aneurysm
  2. Complications:
    - a. acute thrombosis (approximately 30% of cases present as surgical emergencies)
    - b. chronic thrombosis
    - c. no obvious correlation between size of aneurysm and likelihood of complication
    - d. elective surgical repair is warranted for most cases
    - e. Femoral aneurysm though less than 2 cm in size can be followed with serial ultrasounds
- B. Popliteal Artery Aneurysms (PAA)

1. Demographics:
  - a. most common peripheral arterial aneurysm
  - b. occurs almost exclusively in men, 60 – 70 years old
2. Complications:
  - a. thrombosis of the aneurysm or embolization from it to the distal tibial vessels threatens limb survival
  - b. In over 50% of patients with PAAs, either acute or chronic ischemia will be the primary threat to the involved limb
3. Presenting symptoms:
  - a. painful mass behind the knee
  - b. occlusion of the popliteal vein
  - c. compression of the posterior tibial nerve.

## II. Anatomy and Physiology:

Distribution of FAA:

- 60% in the aorto-iliac segment
- 37% popliteal aneurysm
- 50% bilateral

## III. Diagnosis:

### A. Femoral Artery Aneurysms

1. palpation of abdominal aorta and popliteal arteries for associated AAA or popliteal artery aneurysm
2. Ultrasound of the abdominal aorta and the popliteal space to substantiate physical findings
3. arteriogram of the involved femoral artery and distal runoff to delineate involvement of the profunda femoris and superficial femoral arteries

### B. Popliteal Artery Aneurysms

1. examine for other aneurysms, especially in the aortoiliac location
2. Preoperative angiography, CTA, or MRA should show not only the location of the PAA but also delineate the below-the-knee runoff
3. The presence of a patent distal popliteal artery or an adequate posterior or anterior tibial artery is the most important determinant of successful repair for PAAs

## IV. Treatment/Management:

### A. Femoral Artery Aneurysms

Three factors govern the choice of operation for common FAAs:

- location of the origin of the profunda femoris artery
- patency of the superficial femoral artery
- patency of the aneurysm itself

1. **Type 1:** FAAs in which the profunda femoris orifice is distal to the aneurysm and all

vessels are patent, the simplest and most successful repair is a vein or synthetic graft interposition for the resected aneurysm.

2. **Type 2:** FAAs in which the profunda femoris arises from the aneurysmal sac. It is extremely important to maintain patency of the profunda femoris when such aneurysms are repaired. In most instances, such aneurysms are resected and an interposition graft is carried out to the superficial femoral artery. A small side-arm graft then is constructed to the profunda femoris artery.
3. When the superficial femoral artery is chronically occluded, it is ligated and an end-to-end anastomosis of the graft to the profunda femoris artery is performed.

**Postoperative follow-up.** After FAA repair, patients must have follow-up at least yearly. Duplex ultrasound is an ideal method to monitor grafts and to discover other aneurysms. Newly discovered aneurysms of the abdominal aorta or popliteal arteries should be corrected as indicated.

## B. Popliteal Artery Aneurysms

All symptomatic popliteal aneurysms should be treated. All asymptomatic aneurysms greater than 2 cm should be treated. There should be consideration of treatment if the aneurysm is smaller but has mural thrombus, since revascularization at the time of severe ischemia is associated with high incidence of limb loss (35% needed primary amputation and another 15% needed secondary amputation after graft failure with mortality of 5.4%). Graft patency is 80%-90% after elective repair, but significantly worse if done for severe ischemia (40%-70%).

1. PAA < 3 cm  
preferred surgical treatment for a smaller (3 cm) PAA is proximal and distal ligation of the aneurysm combined with a reverse saphenous vein bypass. The aneurysm is exposed through a medial knee incision. When possible, the aneurysm should not be resected, since this dissection can injure the popliteal vein.
2. PAA >3 cm  
Larger aneurysms should be opened, the thrombus removed, and a portion of the sac resected to prevent compressive symptoms from the mass effect of the aneurysm. Posterior approach can be helpful for large aneurysm.

When thromboembolism has acutely occluded the infra popliteal outflow, 2 methods may help reestablish distal flow. One is regional thrombolytic therapy, used preoperatively, or in smaller regional doses intra-operatively. The second is retrieval of the thrombus by a Fogarty thromboembolectomy catheter.

Endoluminal management of popliteal aneurysm is still under investigation but early results are promising and should be at least considered in patients who are surgically high-risk.

**Postoperative follow-up.** Patients surgically treated for PAAs should be reevaluated at least yearly. Ultrasound is the ideal test to monitor any popliteal graft and to check for associated aneurysms of the aortic, femoral, and opposite popliteal artery.

## References:

1. Hallett JW Jr, Brewster DC, Darling RC. Handbook of Patient care in Vascular Surgery, 3rd ed. Boston:Little Brown; 1995:224-226.
2. Ernst CB, Stanley JC. Current Therapy in Vascular Surgery, 4th ed. St Louis, Mo: Mosby; 2001:331-345.
3. Ascher E, Markevitch N, Schutzer RW, et al. Small popliteal artery aneurysm: Are they clinically significant? J Vasc Surg 2003;37:755-60.
4. Dawson I, von Bockel JH, Brand R, Terpstra JL. Popliteal artery aneurysm: Long-term follow-up of aneurysmal disease and results of surgical treatment. J Vasc Surg 1991;13:398-407.
5. Marty B, Wicky S, Ris HB. Success of thrombolysis as a predictor of outcome in acute thrombosis of popliteal aneurysm. J Vasc Surg 2002;35:487-93.
6. Antonelli M, Frigatti P, Battocchio P. Open repair versus endovascular treatment for asymptomatic popliteal artery aneurysm: Results of a prospective randomized study. J Vasc Surg 2005;42:185-193.

# ***Lower Extremity Arterial Occlusive Disease***

---

By Julie E Adams, MD

## **Learning Objectives**

### **Scope of Problem:**

1. Assess vascular system by appropriate skills in history taking and physical examination.
  - a. History
    - i. Pain: Characterize by pattern of presentation, severity, location, frequency, duration, precipitating factors, ameliorating factors ---should help determine acuity and severity of lower extremity ischemia
    - ii. Ulcer formation: Characterize onset, associated trauma, history of previous ulcers, associated pain/drainage/odor, presence of diabetes.
    - iii. Vascular risk factors: diabetes, hypertension, hyperlipidemia, h/o peripheral or coronary artery disease
  - b. Physical Examination of Extremities
    - i. Appearance of limbs: note color (pale, violaceous, red), temperature, presence of edema, hair pattern, location of ulcers or calluses, if any; neurologic exam, prior incisions

- ii. Palpation of pulses: femoral, popliteal, posterior tibial, and dorsalis pedis; if absent pulses → presence of Doppler signals and the ankle-brachial index should be recorded.
2. Explain the concept of critical arterial stenosis.
 

This is defined as the degree of stenosis required to produce a measurable drop in blood flow or a pressure gradient across the stenosis. This is not seen until 75% of the cross-sectional area of the artery is blocked. This area change is associated with a 50% stenosis. Because energy losses across a gradient are related inversely to the 4<sup>th</sup> power of the radius, significant decreases in blood flow and pressure occur after this critical radius has been reached.
3. Understand the natural history of lower extremity claudication.
  - a. Rates of major and minor amputations performed less than 10% over 10 years. Revascularizations less than 20% at 10 years, and most for ongoing claudication.
  - b. Only 10% of lower extremity revascularizations are performed for claudication. The remaining 90% are done for limb salvage (rest pain, ulcer, gangrene).
  - c. Of 100 claudicants who stop smoking and participate in an exercise program, how many will
    - Stay the same or get better?
    - Get worse?
    - Require amputation?
4. Describe the hemodynamics and pathophysiology of claudication.
  - a. What arterial lesions could cause calf claudication alone, calf and thigh claudication, and calf, thigh, hip/buttock claudication?
 

Claudication occurs when blood flow to a muscle group is unable to meet metabolic requirements at a given level of exercise. The pain is reproducible with a given level of activity, and is relieved by cessation of the activity.
5. Know the principles of care for both chronically and acutely ischemic limbs.
  - a. Chronic limb ischemia
    - i. Evaluate degree of ischemia with history and physical, e.g., intermittent claudication v. rest pain; presence of ulceration or gangrene.
    - ii. If limb-threatening ischemia (rest pain, ulceration, or gangrene), angiography for evaluation of arterial anatomy.
    - iii. Vein mapping for surgical bypass options.
    - iv. Cardiac risk stratification.
    - v. Intervention → either endovascular or open surgical depending on all of above factors.
  - b. Acute limb ischemia
    - i. Immediate systemic anticoagulation with heparin (goal → decrease risk of thrombus propagation, or if embolic, of another embolism)
    - ii. Evaluate severity of acute ischemia (based on history and physical exam) → this guides urgency of revascularization.
    - iii. Urgent treatment to restore perfusion and salvage limb –either angiography with possible thrombolytic therapy or open surgical revascularization.

## Anatomy and Physiology:

Describe arterial anatomy of lower extremity.

- a. Femoral arteries
  - i. Superficial femoral artery
  - ii. Profunda (deep) femoral artery
- b. Popliteal artery
- c. Infrapopliteal arteries
  - iii. Anterior tibial artery
  - iv. Posterior tibial artery
  - v. Peroneal artery

## Diagnosis:

1. Discuss the role of angiography.
  - a. Used as a diagnostic tool when intervention is indicated or planned.
  - b. Can be performed urgently in the operating room in conjunction with revascularization or electively/semi-electively in angio suite for preoperative planning.
  - c. See section on imaging.
2. Outline the manifestation of the failing peripheral vascular graft.
  - a. Definition: hemodynamic deterioration within an arterial reconstruction secondary to intimal hyperplasia, progression of proximal or distal disease, or lesions within the graft. If not corrected, graft will likely go on to thrombose.
  - b. Diagnosis of failing graft: recurrence of symptoms, change in peripheral pulse exam, evidence of peripheral ischemia, surveillance imaging.
  - c. Findings on graft duplex/PVR: Shift in velocity within graft or at an anastomosis; drop in ABI.
3. Outline the diagnosis of prosthetic graft infections in the lower extremity.
  - a. Presentation: mild cellulitis, sinus tract, anastomotic pseudoaneurysm, frank hemorrhage. Infected grafts in the abdomen may present with fever/malaise.
  - b. Imaging: CT scan – useful for identifying perigraft fluid, gas, inflammation, abscess formation, or pseudoaneurysm; US also useful in extremity grafts; angiography may be necessary for planning operative repair.
4. Describe signs of limb-threatening acute lower extremity ischemia—the “5 Ps”
  - a. Paresthesia
  - b. Pain – usually acute onset; can be acute on chronic
  - c. Pallor
  - d. Pulselessness
  - e. Paralysis
  - f. Poikilothermia (bonus P)
5. Know the Rutherford Criteria for acute limb-threatening ischemia.
  - a. Class 1: the limb is viable and remains so even without therapeutic intervention.
  - b. Class 2A: the limb is threatened and requires revascularization, but not immediately.
  - c. Class 2B: the limb is severely threatened and requires urgent revascularization for salvage.
  - d. Class 3: the limb is irreversibly ischemic; salvage is not possible.

**Treatment/Modification:**

1. What does “risk factor modification” mean in regard to peripheral vascular disease and why is it important?
  - a. Antiplatelet therapy, smoking cessation, exercise rehabilitation, control of hyperlipidemia (statins), control of diabetes mellitus, treatment of hypertension, treatment of elevated homocysteine.
  - b. Risk factor modification can not only improve outcomes with regard to peripheral arterial disease but plays an important role in reducing cardiac morbidity, a major cause of death in patients with PAD.
    - the 5-, 10-, and 15-year mortality rates for patients with intermittent claudication are approximately 30%, 50%, and 70%, respectively. Most deaths are due to CAD, which is nearly universal.
  
2. Outline pharmacologic interventions available for patients with lower extremity vascular disease.
  - a. Antiplatelet therapy
    - i. Aspirin – irreversibly blocks thromboxane A<sub>2</sub>, leading to decreased platelet aggregation; has been shown to decrease the incidence of peripheral arterial surgery in men
    - ii. Clopidogrel (Plavix) – blocks adherence of fibrinogen to platelets; CAPRIE study found that compared to aspirin, in patients with PAD, clopidogrel was more effective in reducing risk of ischemic stroke, MI, and vascular death.
  - b. Pentoxifylline (Trental) – rheologic agent, increases RBC flexibility and decreases viscosity; early reports found it to increase walking distance in claudicants, but most recent data has found it ineffective over placebo. No longer widely used.
  - c. Cilostazol (Pletal) – inhibits phosphodiesterase type 3, increasing cAMP and resulting in vasodilation and antiplatelet effects; approved for claudication in 1999. Studies have shown an increase of 50% in maximal walking distance compared with placebo. Contraindicated in patients with CHF.
  - d. Prostaglandins – relax vascular smooth muscle and inhibit platelet aggregation; Mixed results with oral agents, though recent randomized trial revealed no reduction in rates of amputation, rest pain, or gangrene.
  - e. Statins – 3 trials have found beneficial results for claudicants with atorvastatin and simvastatin; increased pain-free walking time demonstrated; mechanism? possibly changes in metabolism and/or endothelial function
  
3. Outline the indications for operation/intervention for claudication.
  - a. Lifestyle is significantly impaired or patient’s livelihood is jeopardized.
  - b. Patient should have had a trial of risk factor modification, smoking cessation included.
  - c. If disease is proximal (iliac), treatment may involve iliac angioplasty and/or stenting, and a more modest degree of claudication may be found acceptable for treatment.
  
4. Outline the treatment of prosthetic graft infections in the lower extremity.
  - a. COMPLETE graft removal is the surgical tenet, though some data shows that leaving incorporated graft with use of long-term antibiotics and good tissue coverage may be safe; only true with less virulent organisms (Staph epi). Extraanatomic bypass or reconstruction with autogenous tissue necessary.

5. Explain the treatment options for the failing peripheral vascular graft.
  - a. Thrombolysis with revision
  - b. Surgical thrombectomy with revision
    - i. For early graft failures (less than 30 days), return to OR
    - ii. should occur with attention to distal anastomosis—thrombectomy and evaluation of distal disease.
    - ii. For late graft failures (more than 30 days), full repeat arteriography is indicated.
  - c. Secondary reconstruction
    - factors influencing which type of secondary bypass to use include type of primary bypass (native or PTFE), location and extent of initial lesion, distal arterial anatomy, location of primary bypass, level and severity of ischemia.
  - d. No intervention
6. Review the management workup for lower extremity arterial occlusion.
  - a. Patient selection – only about 10% of bypasses are for claudication; Discuss use of noninvasive testing for evaluation of severity. Know alternative causes of claudication (e.g., popliteal entrapment, spinal stenosis, popliteal cysts, etc).
  - b. Imaging – arteriography gold standard for lower extremity arterial evaluation.
  - c. Endovascular treatment – understand role for angioplasty/stenting in management of iliac and femoropopliteal disease.
  - d. Planning for open surgery – be familiar with indications and techniques for aortofemoral bypass, common femoral endarterectomy, profundaplasty, and femoropopliteal and distal bypasses.
  - e. Review patency rates with use of autogenous and prosthetic grafts in different anastomotic locations.
7. Describe the TASC classification for iliac and femoropopliteal disease and its impact on treatment strategy. See below:

*Morphological stratification of iliac lesions*

TASC Type A iliac lesions

Single stenosis < 3 cm of the CIA or EIA (unilateral/bilateral)

TASC Type B iliac lesions:

Single stenosis 3-10 cm in length, not extending into the CFA

Total of two stenosis < 5 cm long in the CIA and/or EIA and not extending into the CFA

Unilateral CIA occlusion

TASC Type C iliac lesions:

Bilateral 5-10 cm long stenosis of the CIA and/or EIA, not extending into the CFA

Unilateral EIA occlusion not extending into the CFA

Unilateral EIA stenosis extending into the CFA

Bilateral CIA occlusion

TASC Type D iliac lesions:

Diffuse, multiple unilateral stenosis involving the CIA, EIA, and CFA (usually >10 cm)

Unilateral occlusion involving both the CIA and EIA

Bilateral EIA occlusions

Diffuse disease involving the aorta and both iliac arteries

Iliac stenosis in a patient with an abdominal aortic aneurysm or other lesion requiring aortic or iliac surgery

*Recommendation for treatment of iliac lesions:*

TASC type A iliac lesions: Endovascular procedure is the treatment of choice

TASC type B and C iliac lesions: Endovascular treatment is currently more commonly used, but scientific evidence of any superiority over vascular surgery is lacking.

TASC type D iliac lesions: Surgery is the procedure of choice.

*Morphological stratification of femoropopliteal lesions:*

TASC Type A femoropopliteal lesions:

Single stenosis < 3 cm of the SFA

TASC Type B femoropopliteal lesions:

Single stenosis 3-10 cm in length, not involving the distal popliteal artery (some advocate for an occlusion of this length as well)

Heavily calcified stenosis up to 3 cm in length

Multiple lesions, each less than 3 cm (stenosis or occlusions)

Single or multiple lesions in the absence of continuous tibial runoff to improve inflow for distal surgical bypass

TASC Type C femoropopliteal lesions:

Single stenosis or occlusion longer than 5 cm (some advocate for change to >10 cm)

Multiple stenosis or occlusions, each 3-5 cm, with or without heavy calcification

TASC Type D femoropopliteal lesions:

Complete common femoral artery or superficial femoral artery occlusions or complete popliteal and proximal trifurcation occlusions

*Recommendation for treatment of femoropopliteal lesions*

Treatment for TASC Type A femoropopliteal lesions: Endovascular procedure is treatment of choice.

Treatment for TASC Type B femoropopliteal lesions: Endovascular treatment is more commonly used, but more evidence is needed to make firm recommendations.

Treatment for TASC Type C femoropopliteal lesions: Endovascular treatment is more commonly used, although scientific evidence of any superiority over vascular surgery is lacking.

Treatment for TASC Type D femoropopliteal lesions: Surgery is treatment of choice.

## References:

1. Colburn, GL, Mattar SG, Taylor B, et al: The surgical anatomy of the deep femoral artery. *Am Surg* 1995;61:336.
2. May AG, Van de Berg L, DeWeese, JA, Rob CG: Critical arterial stenosis. *Surgery* 1963; 54:250-259.
3. Dormandy JA, Murray GD: The fate of the claudicate—a prospective study of 1969 claudicates. *Eur J Vasc Surg* 1991;5:131-133.
4. Rutherford RB, Baker JD, Ernst C, et al: Recommended standards for reports dealing with lower extremity ischemia: Revised Version. *J Vasc Surg* 1988;26:517-538.
5. 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;33:251-257.
6. Goldhaber SZ, Manson JE, Stampfer MJ, et al: Low-dose aspirin and subsequent peripheral arterial surgery in the Physicians' Health Study. *Lancet* 1992;340:143-145.
7. CAPRIE Steering Committee. A randomized, blinded, trial of clopidogrel versus aspirin in patients at risk of ischemic events (CAPRIE). *Lancet* 1996;348:1329-1339.
8. Hood Sc, Moher D, Barber GG: Management of intermittent claudication with pentoxifylline: Meta-analysis of randomized controlled trials. *Can Med Assoc J* 1996;155:1053-1059.
9. Dawson DL, Cutler BS, Hiatt WR, et al: A comparison of cilostazol and pentoxifylline for treating intermittent claudication. *Am J Med* 2000;109:523-530.
10. Mohler ER, Hiatt, WR, Creager MA: Cholesterol reduction with atorvastatin improves walking distance in patients with peripheral arterial disease. *Circulation* 2003;108:1481-1486.
11. Hiatt WR: The US experience with cilostazol in treating intermittent claudication. *Atheroscler Suppl* 2005 Nov 3, 2005 [epub ahead of print].
12. Rutherford DB, Baker JD, Ernst C, et al: Recommended standards for reports dealing with lower extremity ischemia: Revised version. *J Vasc Surg* 1997;26:517-538.
13. Eliason JL, Reid MW, Proctor MC, et al: A national and single institutional experience in the contemporary treatment of acute lower extremity ischemia. *Ann of Surg* 2003;238:382-390.
14. Machledre HI: Prognosis of the failed infrainguinal graft. *Sem in Vasc Surg* 1990;3:43.
15. Management of peripheral arterial disease (PAD). TASC Working Group. TransAtlantic Inter-Society Consensus (TASC). *J Vasc Surg* 2000;31:S1-S296.
16. Pentecost MJ, Criqui MH, Dorros G, et al: Guidelines for peripheral percutaneous transluminal angioplasty of the abdominal aorta and lower extremity vessels. *Circulation* 1994;89:511-531.
17. Mertens RA, O'Hara PG, Hertzner NR, et al: Surgical management of infrainguinal arterial prosthetic graft infections: review of a thirty-five year experience. *J Vasc Surg* 1995;21:782-790.
18. ACC/AHA Guidelines for the Management of Patients With Peripheral Arterial Disease(Lower Extremity, Renal, Mesenteric, and Abdominal Aortic): A Collaborative Report From the AAVS/SVS, SCAI, SVMB, SIR, and the ACC/AHA Task Force on Practice Guidelines. *JACC Vol. xx, No. x. 2006. 1-75.*

# ***Leg Ulcers***

---

By Andrew Stanley, MD

## **Etiologic Classification of Foot and Leg Ulcers**

Venous obstruction and insufficiency

Arterial etiologies

- Larger arteries
- Atherosclerotic lower extremity PAD
- Thromboemboli, atheroemboli
- Thromboangiitis obliterans
- Microcirculatory
- Diabetic microangiopathy
- Vasculitis

Collagen vascular diseases

Neuropathic

- Diabetes mellitus

Infectious

- Leprosy
- Mycotic

Hematologic

- Sickle cell anemia
- Polycythemia
- Leukemia
- Thalassemia
- Thrombocytosis

Malignancy

- Squamous cell carcinoma
- Kaposi's sarcoma
- Secondary metastases
- Lymphosarcoma, mycosis fungoides

Miscellaneous

- Gout
- Pyoderma gangrenosum
- Necrobiosis lipoidica
- Vitamin B12 deficiency
- Drugs

The above list represents a comprehensive list of the possible etiologies for leg ulcers. In the general vascular practice it is important to be aware of ALL of the potential etiologies of leg ulcers but by far, the most common etiologies for chronic leg ulcers seen in a vascular practice include:

1. Ulcers related to arterial insufficiency
2. Ulcers related to venous insufficiency
3. Ulcers related to neuropathy (diabetic foot)

At this point differentiation between chronic and acute leg ulcers must be made. A widely accepted definition of a “chronic “ leg ulcer is one that has existed for 3 weeks.

It is important to determine the causative factors behind the ulcer so that appropriate therapeutic action can be taken to reverse or correct the physiology that contributed to the ulcerative process. Information can be obtained from a good history, physical exam and non-invasive testing.

	<b>Arterial</b>	<b>Venous</b>	<b>Neurotrophic</b>
Ulcer Location	Toes/Heel	“gaiter” of Leg (ankle to knee NOT FOOT)	Pressure points -1 <sup>st</sup> M-T joint -Lat part of foot
Pain	Yes, esp When elevated	Mild, relieve by elevation	Painless (neuro- trophic)
PE	No palp Pulse ABI<0.4  No bleeding	Normal pulses, swollen leg Normal foot bleeding tissue	Normal pulses Ulcer over “press- ure point”. Good bleeding tissue

As above, arterial ulcers are located on the toes/heel of the foot and are typically painful. They may be accompanied by dependent rubor. The pain is exacerbated by foot/ulcer elevation. On exam, pulses are not palpable and typically, ABI<0.4. Treatment revolves around revascularizing the limb and local wound care (control infection, debride necrotic tissue). Good healing results can be expected in well-vascularized limbs where the ischemic process was identified and treated in a timely fashion.

Venous ulcers are typically located above the ankle and below the knee on the medial aspect of the leg (gaiter portion of the leg). Venous ulcers are not often painful, when pain is part of the clinical picture it is often a dull ache relieved by leg elevation. Hyperpigmentation and lipodermatosclerosis are skin changes associated with chronic venous insufficiency and often precede frank ulceration. Often the foot is spared of all skin changes and swelling. The leg itself is often swollen and weeping fluid. Arterial exam is normal with palpable pulses though swelling often makes pulses difficult to feel. Mainstay of ulcer care here is external compression (Unna’s boot, graded compression stockings). Typical venous ulcers take months to heal (70% of venous ulcers will heal after 3 months of compression therapy)

Neurotrophic ulcers occur most often in diabetic patients. These ulcers are painless and most frequently affect the pressure-bearing plantar aspect of the foot. Common locations include the tissue covering the 1<sup>st</sup> metatarsal head and the tissue lateral to the 5<sup>th</sup> metatarsal head. These patients have normal vascular exams with palpable pulses. Care for these ulcers involves relieving sources of pressure (diabetic orthotics, metatarsal head resection) as well as routine ulcer care (controlling infection, local wound care). Constant surveillance of neurotrophic ulcers is necessary until healing. Clinical conditions to watch for include osteomyelitis and deep space infections.

### References:

1. Sumpio B. Foot ulcers. N Engl J Med 2000;343:787-793.
2. Boulton J, Kirsner R, Vileikyte L. Neuropathic diabetic foot ulcers. N Engl J Med 2004;351:48-55.
3. Singh N, Armstrong D, Lipsky B. Preventing foot ulcers in patients with diabetes. JAMA 2005;293:217-228.
4. Brem H, Sheehan P, Boulton A. Protocol for treatment of diabetic foot ulcers Am J Surg 2004;187 (Supl):1S–10S.
5. Mayfield JA, Reiber GE, Sanders LJ, Janisse D, et al. Preventive Foot Care in Diabetes. Diabetes Care 2004; 27:S63-4.

## ***Renovascular Hypertension***

---

By Julie E Adams, MD

### **Learning Objectives**

#### **Scope of Problem:**

1. Understand which patients might be evaluated for renovascular disease.
  - a. Patients with previously controlled mild hypertension who develop progressive difficulty in control of BP or progressive deterioration in renal function.
  - b. Childhood or adolescent hypertension
  - c. Sudden onset of diastolic blood pressure >115 mm Hg in women < 45 years of age.
  - d. Hypertension refractory to medical management, especially if diastolic >105.
  - e. Hypertension treated with ACE inhibitors with resultant decline in renal function.
  - f. Abdominal bruit
  - g. Hypertensive retinal arteriopathy
  - h. Flash pulmonary edema

2. Describe the natural history of renal artery stenosis.
  - a. About 50% of renal arteries with stenosis will develop worsening stenosis over 5 years.
  - b. About 10% will become totally occluded.
  - c. Renal atrophy will develop in 20% of patients with stenoses greater than 60%.
3. Describe characteristics of pediatric renovascular disease.
  - a. Renovascular hypertension is the 2<sup>nd</sup> most common cause of correctable hypertension in children (2<sup>nd</sup> to coarctation).
  - b. Hypertension in children is defined as BP > 95<sup>th</sup> percentile for age
  - c. 3-10% of children referred for evaluation of hypertension will be found to have significant renovascular lesions
  - d. Causes include FMD, midaortic syndrome, Takayasu's arteritis, neurofibromatosis, Marfan, Klippel-Trenaunay-Weber, Kawasaki disease and Crohn's disease.
  - e. Bilateral disease more common than unilateral disease.
  - f. Renal arteriography is the gold standard for diagnosis, though US, CT, MR, captopril tests all play an initial role in selecting which patients should have further invasive workup.
  - g. Renal vein renin assays are an important component of angiography – ratios of >1.5:1 b/w affected and nonaffected side are significant (not useful for bilateral disease).
  - h. For renal artery lesions, PTA, stenting, and surgical revascularization are all potential treatments.

### **Anatomy and Physiology:**

1. Describe the hemodynamics and pathophysiology of renovascular hypertension.
  - a. Blood pressure elevation is a result of renal hypoperfusion. This stimulates activation of the renin-angiotensin system. Renin release results in increased angiotensin II levels and direct vasoconstriction.
  - b. Angiotensin II also causes volume expansion through sodium retention (via aldosterone). If only one kidney is affected, the contralateral kidney will partially counteract this volume expansion effect through pressure-induced natriuresis. If the contralateral kidney is absent or also affected by renal artery stenosis, the hypertension will be more severe (vasoconstriction + volume expansion).
2. Describe the 2 types of lesions most often responsible for renovascular hypertension, atherosclerotic disease and fibromuscular dysplasia.
  - a. Atherosclerosis—responsible for 90%; usually ostial and proximal lesions.
  - b. Fibromuscular Dysplasia—responsible for 10%, primarily women; frequently involves distal 2/3 of artery; most common type is medial fibrodysplasia (85%); often occurs as a “string-of-beads” series of stenoses; bilateral in 60-70%.

### **Diagnosis:**

1. Describe screening tests for renovascular hypertension.
  - a. Duplex (most reliable)
  - b. captopril-perfusion tests,
  - c. renal vein renin assays,
  - d. split renal function tests, and
  - e. angiography.

## Treatment/Management:

1. Describe the management of renovascular hypertension.
  - a. Medical therapy
    - i. 3 randomized trials comparing medical therapy with angioplasty (not stenting) found only minimal differences in control of blood pressure between the groups; exception is patients with bilateral disease. Overall number of meds decreased with PTA.
    - ii. Patients who have acute decline in renal function after treatment with ACE inhibitor are a subset who benefit from intervention.
  - b. Interventional or surgical therapy
    - iii. Randomized trial comparing angioplasty alone with angioplasty and stenting found significantly improved primary success and primary patency for stented arteries with lower incidence of restenosis.
    - iv. Surgical options include aortorenal bypass, renal artery thromboendarterectomy, renal artery reimplantation, splanchnorenal bypass, and ex vivo reconstruction.
  
2. Describe which patients are most likely to benefit from angioplasty and stenting for renal artery stenosis.
  - c. Progressive renal failure of short duration (<6 months)
  - d. Pulmonary edema and refractory CHF
  - e. Severe renal failure precipitated by ACE inhibitors
  - f. Refractory hypertension
  - g. Severe renal artery stenosis (>80%)
  - h. Renal length must not be < 8 cm

## References:

1. Tollefson DF, Ernst CB: Natural history of atherosclerotic renal artery stenosis associated with aortic disease. *J Vasc Surg* 1991;14:327-331.
2. Caps MT, Perissinotto C, Zierler RE, et al: Prospective study of atherosclerotic disease progression in the renal artery. *Circulation* 1998;98:2866-2872.
3. Zerler RE, Bergelin RO, Isaacson JA, Strandness DE Jr: Natural history of atherosclerotic renal artery stenosis: a prospective study with duplex ultrasonography. *J Vasc Surg* 1994;19:250-258.
4. Goldblatt H: Studies on experimental hypertension. *J Exp Med* 1934;59:347.
5. Safian RD, Textor SC: Renal-artery stenosis. *N Engl J Med* 2001;344:431-442.
6. Garovic V, Textor SC: Renovascular hypertension: current concepts. *Semin Nephrol* 2005;25:261-271.
7. Olin JW, Piedmonte MA, Young JR, et al: The utility of duplex ultrasound scanning of the renal arteries for diagnosing significant renal artery stenosis. *Ann Intern Med* 1995;122:833-838.
8. Gray BH: Intervention for renal artery stenosis: endovascular and surgical roles. *J Hypertens* 2005;Suppl 3:S23-29.
9. Cherr GS, Hansen KJ, Craven TE, et al: Surgical management of atherosclerotic renovascular disease. *J Vasc Surg* 2002;35:235-245.

10. Yutan E, Glickerman DJ, Caps MT, et al: Percutaneous transluminal revascularization for renal artery stenosis: Veterans Affairs Puget Sound Health Care System experience. *J Vasc Surg* 2001;34:685-693.
11. 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 Group. *N Engl J Med* 2000;342:1007-1014.
12. van de Ven PJG, Kaatee R, Beutler JJ, et al: Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: a randomized trial. *Lancet* 1999;353:282-286.
13. Dean RH, Krueger TC, Whiteneck JM, et al: Operative management of renovascular hypertension: results after 15-23 years of follow-up. *J Vasc Surg* 1984;1:234.
14. Novick AC: Long-term results of surgical revascularization for renal artery disease. *Urol Clin North Am* 2001;28:827-831.
15. Delias KT, Gloviczki P: Middle aortic syndrome: from presentation to contemporary open surgical and endovascular treatment. *Perspect Vasc Surg Endovasc Ther* 2005;17:187-203.
16. Stanley JC: Renal vascular disease and renovascular hypertension in children. *Urol Clin North Amer* 1984;11:451-463.
17. Stanley JC, Zelenock GB, Messina LM, Wakefield TW: Pediatric renovascular hypertension: a thirty-year experience of operative treatment. *J Vasc Surg* 1995;21:212-226.
18. Tyagi S, Kaul UA, Satsangi DK, Arora R: Percutaneous transluminal angioplasty for renovascular hypertension in children: initial and long-term results. *Pediatrics* 1997;99:44-49.

## ***Mesenteric occlusive disease***

---

By Georg Steinthorsson, MD

### **Learning Objectives**

#### **I. Anatomy and Pathophysiology**

1. Define normal arterial and venous anatomy of the mesenteric circulation and be familiar with common variations.
2. Recognize the physiologic and pathophysiologic collateral circulation to the gastrointestinal tract that may develop in response to occlusive disease of the main mesenteric vessels.
3. Understand the high flow, low resistance physiology of normal mesenteric blood flow, recognize the neural, humoral (hormonal) and enteric (intraluminal ) mechanisms of autoregulation, and understand the high degree of vasoreactivity of this arterial bed.
4. Understand the multiple etiologies of acute mesenteric ischemia including embolism, thrombosis, dissection, venous occlusion, trauma, and gut ischemia following aortic reconstruction
5. Understand the multiple possible etiologies of syndromes of chronic mesenteric ischemia including atherosclerosis, aneurysm, extrinsic compression syndromes, and other nonatherosclerotic arteriopathies.
6. Understand the clinical correlation of multiple visceral vessel involvement with the development of symptoms of chronic intestinal ischemia based upon an understanding of the compensatory collateral perfusion of the gut.

#### **II . Diagnostic Evaluation**

### ***Acute Mesenteric Ischemia***

1. Identify the characteristic initial signs and symptoms; and distinguish from other causes of the acute abdomen.
2. Define predisposing clinical conditions: e.g., atrial fibrillation, previous myocardial infarction (mesenteric embolism), severe cardiopulmonary dysfunction (non-occlusive ischemia), history of post- prandial pain and weight loss, known aortic dissection (mesenteric thrombosis), hypercoagulable states (mesenteric venous thrombosis).
3. Diagnostic testing:
  - a) initial serologic testing
  - b) mesenteric arteriography (or other forms of visceral arterial imaging):
    - 1) when indicated in patients with suspected acute mesenteric ischemia and understand the technical aspects of the conduct of arteriography necessary to make an accurate diagnosis.
    - 2) define findings diagnostic of mesenteric thrombosis, mesenteric embolism, and non-occlusive mesenteric ischemia.
  - c) appropriate diagnostic evaluation for suspected intestinal ischemia following aortic surgery
  - d) CT and MRI as alternative diagnostic imaging in the diagnosis of acute mesenteric venous thrombosis.

### ***Chronic Mesenteric Ischemia***

1. Identify characteristic signs, symptoms, and predisposing conditions
2. Diagnostic testing:
  - a) standard GI diagnostic testing (e.g.,endoscopy, contrast studies, CT)
  - b) porto-mesenteric duplex ultrasound scanning for elective noninvasive evaluation of the major visceral vessels.
  - c) arteriography (or alternative vascular imaging studies). To recognize the characteristic arteriographic findings in atypical causes of mesenteric arterial compromise including extrinsic compression and nonatherosclerotic visceral arterial disease.

## **III . Treatment**

### ***Acute Mesenteric Ischemia***

1. Surgery:
  - a) To be familiar with techniques for surgical exposure of the main mesenteric vessels
  - b) To understand standard surgical options for revascularization
  - c) to understand surgical options for the manage intestinal necrosis
  - d) understand the relative usefulness of intraoperative techniques to assess intestinal viability
  - e) recognize relationship of different anatomic patterns of gut infarction to the different causes of acute mesenteric ischemia when intestinal infarction is encountered unexpectedly at the time of laparotomy.
2. Interventional non-surgical treatments
3. Prognosis:

- a) Impact of relationship between the extent of viable bowel (before and/or after successful revascularization) and the extent of resection of nonviable intestine
- b) pathophysiologic effects of intestinal reperfusion
- c) role of early empiric re-exploration following surgical treatment

### ***Chronic Mesenteric Ischemia***

#### 1. Surgery:

- a) To be familiar with all standard surgical techniques for direct, elective visceral revascularization and understand the importance of comprehensive revascularization in the surgical treatment of chronic intestinal ischemia.
- b) surgical alternatives for atypical or non-atherosclerotic visceral arterial occlusive lesions.

2. Percutaneous treatment. To understand the possible application of interventional, nonsurgical treatments for chronic visceral arterial occlusive lesions.

3. Follow-up: To understand the usefulness of noninvasive vascular testing after visceral revascularization procedures.

### **Mesenteric Artery Anatomy**

Three major mesenteric arteries supply blood to specific territories, communicating by collateral channels. These collateral channels enlarge when a proximal mesenteric artery is stenotic or occluded. The primary collateral pathways between the celiac and superior mesenteric arteries are through the gastroduodenal artery to the pancreaticoduodenal arteries that connect with the superior mesenteric artery. The inferior mesenteric artery has 2 main sources of collateral flow when it is obstructed. The middle colic branch of the superior mesenteric artery connects around the transverse colon to the marginal artery of Drummond, a continuation of the left colic branch of the inferior mesenteric artery.

The inferior mesenteric artery also receives collateral flow through the middle hemorrhoidal artery, a branch of the internal iliac artery. These abundant collateral channels for mesenteric circulation explain the clinical observation that intestinal angina usually does not occur until at least 2 of the 3 main mesenteric arteries have severe occlusive disease.

### **Clinical Presentation**

Intestinal ischemia is classified as either chronic or acute. However, the presentations may overlap, since chronic intestinal stenosis can progress to acute thrombosis and intestinal infarction.

Chronic intestinal ischemia often eludes early diagnosis because the chronic abdominal pain is attributed to some more common gastrointestinal (GI) disorder. Frequently, the patient has undergone a negative diagnostic evaluation of the gallbladder, liver, and entire GI tract. Because of progressive weight loss, some patients are mistakenly thought to have cancer. Certain clinical features, however, should raise suspicion of chronic intestinal ischemia. Classically, the chronic abdominal pain is intermittent and postprandial. It usually is localized to the epigastrium and is a dull ache or colic that begins 30-60 minutes after eating and may persist for a few hours. Patients may have associated abdominal bloating or diarrhea. Involuntary weight loss eventually occurs because the patient associates eating with pain. Consequently, a "food fear" develops. Oral intake often is

modified until liquids become the primary nutrient.

Physical findings of chronic intestinal ischemia are limited primarily to weight loss and an abdominal bruit. Since the weight loss usually is insidious over several months and many patients with atherosclerosis have abdominal bruits, the significance of these nonspecific findings often is overlooked when the patient initially presents.

Chronic intestinal ischemia should be suspected in any adult who has chronic abdominal pain, progressive weight loss, other signs of generalized cardiovascular disease, and a negative work-up for more common GI disorders.

Acute intestinal ischemia has 3 main etiologies: thrombosis of an arterial stenosis, embolism, and nonocclusive small vessel insufficiency.

Although the initial symptom for all these etiologies is abdominal pain, the clinical setting often suggests the most likely underlying cause. Incidence may vary among medical centers, but generally acute intestinal ischemia is caused by thrombosis in 40% of cases, embolism in another 40%, and intestinal hypoperfusion in 20% of cases.

Severe generalized abdominal pain that is disproportionate to the physical findings remains the classic presentation of acute intestinal ischemia. Nausea, vomiting, or diarrhea may follow shortly after the onset of symptoms. Although the abdomen may have diffuse tenderness, bowel sounds may be heard and peritoneal signs usually are absent. The only early laboratory abnormality may be an elevated white blood cell count. When these findings are made, the clinician must suspect acute mesenteric ischemia and undertake steps to alleviate it. Aggressive radiologic and surgical intervention at this point can salvage about 50% of such patients. If intestinal ischemia remains unrecognized, physical findings will change as intestinal necrosis develops. Bloody diarrhea may occur, although often it is not present. Hypovolemia becomes evident as fluids are sequestered in the ischemic intestinal wall and surrounding tissues. Fever, peritoneal signs, and shock occur as sepsis becomes established. When intestinal ischemia has advanced to this point, the likelihood of salvaging the ischemic intestine and the critically ill patient is less than 15%-20%.

The clinical setting and the patient's past medical history usually suggest the etiology of acute intestinal ischemia. If chronic intestinal angina preceded acute symptoms, mesenteric artery thrombosis is the most likely etiology. Emboli should be suspected when atrial fibrillation is present or if the patient has had previous cerebral or lower extremity thromboembolism. Nonocclusive, mesenteric ischemia occurs in the setting of low cardiac output. The most common predisposing conditions for nonocclusive, mesenteric ischemia are myocardial infarction, congestive heart failure, renal or hepatic disease, or any major operation that leads to hypovolemia or hypotension in a patient with atherosclerosis. This type of nonocclusive, acute mesenteric ischemia is being recognized more commonly.

## **Diagnostic Tests**

Arteriography is the most reliable way to diagnose acute or chronic intestinal ischemia. Early angiographic diagnosis is the most important principle of successful management of acute intestinal ischemia. An arteriogram with lateral views of the aorta to show the mesenteric artery origins is the definitive method. The angiographic catheter also provides an important route for delivering vasodilating drugs and thrombolytic agents to the mesenteric circulation.

The evaluation of possible chronic mesenteric ischemia usually includes other diagnostic tests before angiography is done, such as barium studies of the upper and lower GI tract. Abdominal ultrasound and computed tomography (CT) scanning may also reveal hepatobiliary disease or occult tumors such as cancer of the pancreas or lymphoma of the retroperitoneum. Duplex scanning can scan mesenteric blood flow patterns, allowing noninvasive determination of stenosis and changes in flow before and after a test meal. The use of velocity wave form parameters that can discriminate between normal subjects and those with visceral artery stenosis should reduce both the incidence of missed diagnosis and unnecessary angiography. The extent of diagnostic work-up for patients presenting with chronic abdominal pain obviously must be individualized.

## Management

Treatment of intestinal ischemia also can be organized into the 2 broad categories of chronic and acute ischemia.

Chronic intestinal ischemia can be relieved only by correction of the occlusive lesions. There is no effective medical therapy. Surgical correction has been the most common technique. Although balloon angioplasty has been shown to be successful, it is associated with a higher incidence of recurrent symptoms. Operative management is preferable but endoluminal treatment should be considered if the patient is high surgical risk.

Nutritional repletion is one aspect of preoperative preparation that deserves special emphasis. Since chronic intestinal ischemia leads to progressive weight loss, some patients are chronically malnourished and have no nutritional reserves for a major abdominal operation. We strongly recommend that such catabolic patients undergo a period of total parenteral nutrition before elective surgery. This nutritional repletion may prolong hospitalization but should enhance perioperative wound healing.

There are 2 basic surgical options for mesenteric revascularization: bypass grafting or endarterectomy. Either supraceliac or infra-renal aortomesenteric bypass grafting is the procedure of choice for most patients. The optimum method of mesenteric revascularization, however, depends highly on the number of vessel occlusions and the condition of the abdominal aorta in each patient. Transaortic endarterectomy has also been successful for multiple visceral occlusive lesions at the origins of the mesenteric arteries. Combined infrarenal aortic replacement and Dacron bypasses to mesenteric arteries appears to be the technique of choice when severely symptomatic infrarenal aortic occlusive or aneurysmal disease coexists with chronic intestinal ischemia. However, combining mesenteric revascularization and aortic replacement carries a high mortality rate of 10%-20% in these cachectic patients. In contrast, limiting the operation to some type of aortomesenteric grafting or endarterectomy alone brings mortality to 3%-5%.

Regardless of which method of revascularization is selected, early relief of intestinal angina is achieved in about 90% of patients. Some reports indicate that results are superior when at least 2 occluded or stenotic vessels are revascularized. In most patients this combination has included both the celiac and superior mesenteric arteries. However, good results have been reported from single-vessel retrograde ileac-mesenteric bypasses.

Symptoms recurred in 10% of patients who underwent complete revascularization, in 25% who had 2 of 3 occlusive lesions corrected, and in 50% who had a single vessel revascularized. Since

revascularization of a single mesenteric occlusion offers relief to most patients, complete revascularization must be weighed against the patient's overall condition, prognosis, and other technical factors.

Chronic or acute intestinal ischemia also may be the iatrogenic result of sacrificing the inferior mesenteric artery at the time of infra renal aortic grafting. Revascularization of the inferior mesenteric artery should be performed when the superior mesenteric artery is occluded and a large inferior mesenteric artery is present.

Successful management of acute intestinal ischemia must begin with arteriography to define the mesenteric anatomy. Optimal therapy for acute mesenteric ischemia cannot be determined unless the clinician knows whether the problem is thrombotic, embolic, or hypoperfusion-related.

**Thrombosis** usually is apparent on the arteriogram by obstruction of the superior mesenteric artery at its origin from the aorta. Systemic heparin is indicated to prevent clot propagation. Emergency abdominal exploration should be undertaken to assess bowel viability and to revascularize the obstructed artery. Resection of a nonviable intestine without revascularization of the remaining small bowel is associated with a high incidence of further intestinal infarction and death. Generally the bowel should be revascularized before intestinal resection. Resection of a nonviable intestine without revascularization of the remaining small bowel is associated with a high incidence of further intestinal infarction and death.

An exception to this rule is resection before revascularization when a segment of intestine is grossly gangrenous or perforated. A single aortomesenteric bypass is sufficient in these seriously ill patients. When bowel contamination is present, a vein graft is preferable to a synthetic material. Clinical judgment of intestinal viability may be enhanced by fluorescein examination of the GI tract. The test is performed by injection of 2 ampules (1,000 mg) of sodium fluorescein through a peripheral vein and immediate examination of the bowel under an ultraviolet Wood's light in a darkened operating room. A viable bowel has a smooth or uniform fluorescence. A nonviable bowel has decreased, patchy, or no fluorescence. When it appears that the bowel may survive, it may be left alone and a second-look operation should be performed within 24 hours to reassess the intestinal viability. Doppler flow analysis has been used also to check intestinal perfusion, but it appears to be less reliable than fluorescein examination. Open surgical technique, resection of nonviable bowel, and liberal use of second-look procedures are associated with improved survival.

**Emboli** usually lodge a few centimeters beyond the origin of the mesenteric artery at the level of the first jejunal branches. Standard therapy remains identical to the management of thrombosis, except the embolus usually is removed by a mesenteric artery arteriotomy and Fogarty catheter thromboembolectomy. Sometimes a vein patch or a bypass graft is needed if significant arterial stenosis is present also.

**Nonocclusive mesenteric ischemia** generally is seen in critically ill patients who have low cardiac output. They often are poor risks for any surgery and many times are so labile that transport to the angiographic suite is a major undertaking in itself. Their arteriograms show peripheral mesenteric vasoconstrictions but no large-vessel occlusions. Noninvasive ischemic colitis appears to be mediated primarily by a remarkable sensitivity of the colonic vasculature to the renin-angiotensin axis. The best results with this group of patients have been achieved by measures to improve cardiovascular hemodynamics and to vasodilate the peripherally constricted mesenteric vasculature. The recommended splanchnic vasodilator therapy is a papaverine infusion of 30-60 mg/hour through an angiographic catheter positioned in the superior mesenteric

artery. If a vasopressor is needed to increase blood pressure, dopamine is the drug of choice since it reduces renal and mesenteric vascular resistance.

### References:

1. Hallett JW Jr, Brewster DC, Darling RC. Handbook of Patient care in Vascular Surgery, 3rd ed. Boston:Little Brown; 1995:236-243.
2. Kasirajank K, O'Hara PJ, Gary BH, et al. Chronic mesenteric ischemia: Open surgery versus percutaneous angioplasty and stenting. J Vasc Surg 2001;33:63-71.
3. Cho JS, Carr JA, Jacobsen G, Shepard AD, Nypaver TJ, Reddy DJ. Long term outcome after mesentery artery reconstruction: A 37- year experience. J Vasc Surg 2002;35: 453-60.
4. Park WM, Gloviczki P, Cherry KJ Jr. Contemporary management of acute mesenteric ischemia: Factors associated with survival. J Vasc Surg 2002;35:445-52.

## ***Peripheral Vascular Trauma***

---

By Steven R Shackford, MD

### Learning Objectives:

#### I. Scope of Problem:

Vascular injury can be either blunt or penetrating. In rural environments, such as Vermont, most vascular trauma is due to blunt injury, whereas in urban environments it is mostly penetrating. Since the advent of endoluminal therapies, the number of iatrogenic injuries has increased.

#### II. Resuscitation:

Peripheral vascular trauma, for the most part, is *limb threatening*; however, vascular injury can lead to a life-threatening situation, such as hypovolemic hemorrhagic shock. Thus, the initial management consists of airway, breathing, and circulation. Circulation means not only controlling hemorrhage, but also initiating intravenous access and beginning volume resuscitation. Generally, low-pressure resuscitation (not to exceed a systolic blood pressure of 100) is advocated so that one does not "pop the clot" on injured vessels.

#### III. Diagnosis

##### A. Physical examination

A complete history and physical examination should be done to rule out other injuries and sources of blood loss. With respect to the injured extremity, one should look for "hard" signs of hemorrhage, which would mandate the need for operative exploration. These hard signs are (1) pulsatile bleeding, (2) expanding hematoma, (3) palpable thrill, and (4) evidence of

regional ischemia as manifested by pallor, parathesias, paralysis, pain, pulselessness, and poikilothermia. One should also be cognizant of the “soft” signs which would indicate the need for further evaluation (i.e., imaging or ultrasound). These “soft” signs include a (1) history of moderate hemorrhage, (2) injury in the proximity of a major vessel, (3) diminished pulse, and a (4) peripheral nerve deficit. One should do a complete neurologic examination assessing sensation to light touch and motor strength. All findings should be carefully documented in the medical record.

#### B. *Non-invasive Imaging*

A continuous wave Doppler ankle brachial or wrist brachial index should be obtained in the injured extremity. If the pulse is absent (not palpable) or diminished and there is evidence of reasonable perfusion, an ankle brachial or wrist brachial index of less than 0.9 (compared to the uninjured extremity) would indicate the need for further imaging.

#### C. *Imaging*

Imaging is reserved for those patients with evidence of regional ischemia who have multiple potential sites of injury (such as a shotgun wound with multiple pellet wounds and no distal pulses) and those patients in which a vascular injury is suspected but the access is difficult (i.e., wounds at the base of the neck or in the apex of the neck).

##### 1. CTA

CT angiography using multi-detector CT has been shown to be as accurate as arteriography in the diagnosis of arterial abnormalities. Post processing using either multidetector reconstruction or volume rendering can detect peripheral vascular injuries with the sensitivity and specificity similar to standard digital subtraction digital angiography.

##### 2. Angiography

Angiography can be done either in the operating room, or in the angiography suite.

### IV. **Treatment**

#### A. *Observation*

Some arterial injuries can be observed. These are the ones that are detected in nutrient vessels (such as a tertiary branch of the profunda femoris artery) or ones that produce small intraluminal defects or small pseudoaneurysms in conduit vessels. Long follow-up of these injuries has shown them to be benign.

#### B. *Endoluminal*

Endoluminal therapy has been used preferentially for injuries to the carotid and vertebral system. Either covered or uncovered stent grafts can be used for intimal dissections and pseudoaneurysms.

#### C. *Ultrasound guided*

Iatrogenic pseudoaneurysms that occur following percutaneous procedures can be treated by ultrasound guided thrombin injection.

#### D. *Operative*

Most arterial injuries that produce either regional ischemia or hemorrhage are managed operatively. The basic principles are heparinization (which can be done preoperatively if there is an isolated arterial injury), wide exposure using incisions for elective exposure, proximal control, distal control, catheter thrombectomy, and completion arteriography.

Significant injuries require interposition grafting with autologous saphenous vein. Prosthetic material can be used but it is not the first choice.

#### E. *Intraluminal shunts*

Intraluminal shunts were first used in the Israeli war in 1967 to transport patients with significant vascular injuries from the field to an advanced care facility. The placement of these shunts allowed almost immediate restoration of blood flow and may have saved many limbs during this conflict. The concept was adopted in modern trauma day care when prehospital transport times were long or when patients had complex injuries involving a fracture and an arterial injury. When patients present with a fracture and an arterial injury there has been much debate as to which procedure should be done first: bone stabilization or vascular repair. Advocates for initial bone stabilization argue that the limb will be brought to the appropriate length and orthopedic manipulation is done prior to the vascular repair and, thus, avoids trauma to the vascular repair during fracture alignment. Advocates for initial vascular repair argue that the limb needs to be revascularized as quickly as possible. Intraluminal shunts placed immediately after the patient arrives in the operating room remove the sense of urgency and allow the orthopedic surgeons to stabilize the fracture.

Shunts can be premade or can be made from intravenous tubing at the time of surgery. The placement requires a quick exploration of the wound and proximal and distal control of the injured artery. The artery can be debrided and the shunt inserted and kept in place with vessel loops or a silk tie. Patency of the shunt must be determined by continuous wave Doppler interrogation of the distal artery. Shunts can be placed in both the artery and the vein. Shunt dwell times have been as long as 72 hours. Heparin is not needed.

#### **References**

1. Hiatt MD, Fleischman D, Hellinger JC, Rubin GD: Angiographic imaging of the lower extremities with multidetector CT. *Radiol Clin North Am* 2005;43:1119-27.
2. Frykberg ER, Dennis JW, Bishop K, et al: The reliability of physical examination in the evaluation of penetrating extremity trauma for vascular injury. Results at one year. *J Trauma* 1991;31:502.
3. Johansen J, Lynch K, Paun M, Copass M: Non-invasive vascular tests reliably exclude occult arterial trauma in injured extremities. *J Trauma* 1991;31:515.
4. Lynch K, Johansen K: Can Doppler pressure measurement replace "exclusion" arteriography in the diagnosis of occult extremity arterial trauma? *Ann Surg* 1991;214:737.

# ***THE MANGLED EXTREMITY***

By Steven R Shackford, MD

## **I. Definition:**

A mangled extremity is a limb (upper or lower) that has been exposed to high energy transfer (usually a crushing or degloving injury), is open and contaminated, and has injury to integument, muscle, and bone. There is often injury to neural elements and the vascular supply. It can be an isolated injury or occur with life threatening associated injuries. It should be considered a limb-threatening injury either immediately (due to compromise of the blood supply) or remotely (due either to sepsis or poor neurologic function or pain).

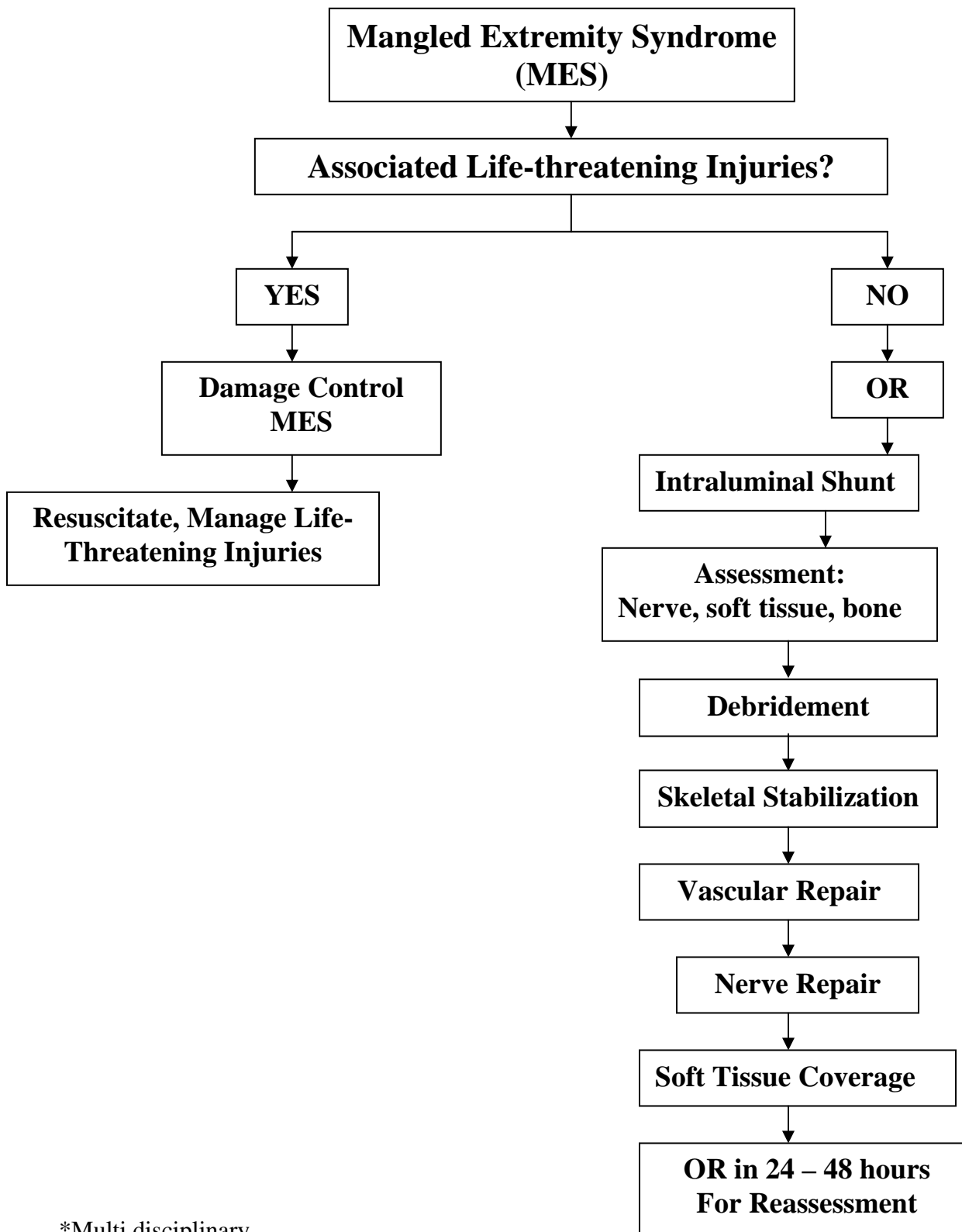
## **II. Management:**

Even with maximal medical and surgical therapy the amputation rate remains 40% - 70%. The surgical dilemma, therefore, is whether to immediately amputate the limb or to proceed with major reconstruction of bone and soft tissue. A variety of injury scoring systems have been developed to assist surgeons in decision making. A recent prospective evaluation of these scoring tools demonstrated that they have little or no clinical utility in predicting the need for amputation. However, it is generally accepted that very low scores have high specificity (meaning no need for amputation) while high scores have low sensitivity (may result in unnecessary amputation).

One of the most important factors in determining long-term outcome of these injuries is the condition of the nerve. Significant neural injury can lead to loss of protective sensation or severe post injury pain syndromes that lead to late amputation. A recent large prospective study evaluating outcomes of reconstruction or amputation of severe leg injuries found no difference in the eventual amputation rates between patients undergoing immediate amputation and patients having reconstruction. Functional status of the limb at 24 months showed similar lower extremity function in the 2 groups. The group undergoing reconstruction required significantly more operations, had a significantly higher rate of osteomyelitis, and had significantly more complications.

Management of these injuries is always difficult because the patient usually is young and vigorous. The best approach is (see diagram):

1. Initial multidisciplinary evaluation including orthopedic surgery, plastic surgery (if necessary), vascular surgery (if necessary) and neurosurgery (if necessary)
2. Skeletal stabilization
3. Soft tissue coverage
4. Return to the operating room in 24-48 hours for reassessment. This "contemplative" approach allows adequate multidisciplinary input and avoids precipitous amputation. It also allows time for the family to be involved in the progress of the case. An algorithm is included.



\*Multi disciplinary  
+If necessary

**REFERENCES:**

1. Bosse MJ, Mackenzie EJ, Kellam JF et al: A prospective evaluation of the clinical utility of the lower extremity injury severity scores. J Bone Joint Surg 2001;83-A:3-14.
2. Bosse MJ, Mackenzie EJ, Kellam JF et al: An analysis of outcomes of reconstruction or amputation of leg-threatening injuries. N Engl J Med 2002; 347:1924-1931.

## ***Thrombolytic Therapy***

---

By Julie E Adams, MD

### **Learning Objectives**

#### **Scope of Therapy:**

1. Discuss various thrombolytic agents and their mechanism of action
  - a. Streptokinase – indirectly activates plasminogen by complexing with human plasminogen, which then catalyzes plasminogen to plasmin → dissolves thrombi
  - b. Urokinase – directly activates plasminogen to active plasmin, allowing fibrinolysis to occur
  - c. Recombinant Tissue Plasminogen Activator (rt-PA or Alteplase) – t-PA is an endogenous plasminogen activator made by endothelial cells. t-PA binds to fibrin and locally converts fibrin-bound plasminogen to plasmin, activating the endogenous fibrinolytic system
  - d. TNK-t-PA – plasminogen activator like t-PA but with longer half-life, increased specificity for fibrin and increased resistance to inactivation
  - e. Altimeprase – a new direct acting thrombolytic that does not require plasminogen; has direct fibrinolytic activity against the  $\alpha$ -chain of fibrinogen/fibrin; still in clinical trials
2. Recognize the common situations in which these agents are used
  - a. Acute embolic or thrombotic occlusion of arteries inaccessible or difficult to access surgically for operative thromboembolectomy
  - b. Acute thrombosis of a popliteal aneurysm with limb-threatening ischemia
  - c. Acute arterial thrombosis, especially of proximal arteries
  - d. Thrombosed saphenous vein graft (that had been functional for a year or more)
  - e. Venous thromboses
  - f. Pulmonary embolus
  - g. Acute MI
  - h. Acute ischemic stroke
3. Discuss the contraindications for thrombolytic therapy
  - a. Absolute contraindications
    - i. Established cerebrovascular event (including TIAs) within last 2 months
    - ii. Active bleeding diathesis
    - iii. Recent gastrointestinal bleeding (<10 days)
    - iv. Neurosurgery (intracranial, spinal) within last 3 months
    - v. Intracranial trauma within last 3 months

- b. Relative major contraindications
    - i. Cardiopulmonary resuscitation within last 10 days
    - ii. Major nonvascular surgery or trauma within last 10 days
    - iii. Uncontrolled hypertension: >180 mmHg systolic or >110 mmHg diastolic
    - iv. Puncture of noncompressible vessel
    - v. Intracranial tumor
    - vi. Recent eye surgery
  - c. Minor contraindications
    - i. Hepatic failure, especially with coagulopathy
    - ii. Bacterial endocarditis
    - iii. Pregnancy
    - iv. Diabetic hemorrhagic retinopathy
4. Know dosages and rates of administration for thrombolytic t-PA and urokinase. Understand why streptokinase is not clinically used.
- a. rt-PA
    - i. dose: 0.5 to 2.0 mg/hr
    - ii. half-life: 3.5 minutes
  - b. urokinase
    - i. dose: 250,000 IU bolus, 4000 IU/min for 4 hours, then 2000 IU/min
    - ii. half-life: 14 minutes
  - c. TNK rt-PA
    - i. Dose: 5 mg bolus and 0.25 mg/hour infusion
    - ii. half life: 15 minutes
  - d. Streptokinase not clinically used because of high antigenicity and resultant allergic reactions; also with unpredictable and severe bleeding complications.
5. Discuss important clinical trials that establish treatment guidelines for used of thrombolytic therapy.
- a. Rochester Trial
    - i. 114 patients with acute LE ischemia ( $\leq 7$  days)  $\rightarrow$  urokinase versus surgical revascularization. Lysis successful in 70% of urokinase group.
    - ii. Limb salvage: 82% in each group at 12 months
    - iii. Survival: 84% (lysis) vs. 58% (OR) at 12 months ( $p=0.01$ )
  - b. STILE Trial (Surgery versus Thrombolysis for the Ischemic Lower Extremity)
    - i. Randomized patients with acute and chronic (>6 mos) limb ischemic to rt-PA, urokinase, or surgical revascularization
    - ii. Acute limb ischemia (<2 wks)  $\rightarrow$  limb salvage at 6 months better with lysis (AMP rate-30% surgery vs. 11% lysis,  $p=0.02$ )
    - iii. Chronic limb ischemia (>6 mos)  $\rightarrow$  limb salvage at 6 months better with surgery (AMP rate 3% surg vs. 12% lysis,  $p=0.01$ )
    - iv. Subset of bypass patients: Patients with acute graft occlusion (<14 days) did better with lysis  $\rightarrow$  lower 1-year amp rate compared with surgery (20% vs. 48%,  $p=0.026$ ). Patients with chronic graft occlusion (>14 days) did better with surgery,  $p=0.003$ )
    - v. No difference in efficacy or bleeding complications with rt-PA versus urokinase
  - c. TOPAS Trial – Thrombolysis Or Peripheral Arterial Surgery for acute limb ischemia

- i. Compared urokinase with surgical revascularization for acute (<14 days) of ischemia secondary to arterial or graft occlusion
- ii. 1 year amputation rates were similar (15% and 13%) and death rates were similar (20% and 17%). Patients in lytic group had higher risk of major hemorrhage (13% v 6%,  $p=0.005$ ) and a 1.6 % risk of intracranial bleed

### References:

1. NIH Consensus Development Conference. Thrombotic therapy in thrombosis. Br Med J 1980;280:1585-1587.
2. Fletcher AP: Pharmacology of thrombolysis: urokinase. J Clin Pathol 1997;25:633.
3. Hoylaerts M, Rijken DC, Lijnen HR, Collen D: Kinetics of the activation of plasminogen by human tissue plasminogen activator: role of fibrin. J Biol Chem 1982;257:2912.
4. Cannon CP, Gibson CM, McCabe CH, et al: TNK-tissue plasminogen activator compared with front-loaded alteplase in acute myocardial infarction: results of the TIMI 10B trial. Circulation 1998;98:2805-2814.
5. Ouriel K, Cynamon J, Weaver FA, et al: A phase I trial of alfimeprase for peripheral arterial thrombolysis. J Vasc Interv Radiol 2005;16:1075-1083.
6. The STILE Investigators. Results of a prospective randomized trial evaluating surgery versus thrombolysis for ischemia of the lower extremity. The STILE trial. Ann Surg 1994;220:251-268.
7. Ouriel K, Shortell CK, DeWeese JA, et al. A comparison of thrombolytic therapy with operative vascularization in the initial treatment of acute peripheral arterial ischemia. J Vasc Surg 1994;19:1021-1070.
8. Ouriel K, Veith FJ, Sasahara AA: Thrombolysis or peripheral arterial surgery (TOPAS): phase I results. J Vasc Surg 1996;23:64-75.
9. Thrombolysis in the management of lower limb peripheral arterial occlusion—A consensus document. J Vasc Interv Radiol 2003;7:S337-S349.
10. Earnshaw JJ, Whitman B, Foy C: National audit of thrombolysis for acute leg ischemia (NATALI): clinical factors associated with early outcome. J Vasc Surg 2004;39:1018-1025.

## ***Venous Diseases***

---

By Michael A Ricci, MD

### Learning Objectives

#### Scope of Problem:

#### Specific Diseases

1. venous thrombosis
2. deep venous thrombosis
3. pulmonary embolism
4. venous stasis ulceration
5. post-thrombotic venous insufficiency
6. lymphedema

#### Pathophysiology:

Understand the pathophysiology behind the development of venous thrombosis

- Virchow's triad
- Clinical risk factors for thromboembolic disease

Understand the pathophysiology of chronic venous insufficiency

- Venous hemodynamics
- Pathophysiology of venous stasis dermatitis and lipodematofibrosis
- Venous ulceration

### Diagnosis:

Understand the complexities of diagnosis of DVT

- Clinical signs and symptoms
- Reliability of clinical exam

Ultrasound diagnosis

- Accuracy in absence of physical signs
- Role of venography

Understand the risk for pulmonary embolus, diagnostic pitfalls, and treatment options

### Treatment:

Compression therapy

Inferior vena cava filters

## PATHOPHYSIOLOGY OF SPECIFIC DISEASES

### Venous Thrombosis:

Thrombosis begins in the valve cusps or at sites of direct venous injury with the accumulation of platelets. Activation of the coagulation cascade occurs as factor XII (Hageman Factor) contacts any surface other than endothelium, such as the aggregated platelets. Ultimately, fibrin stabilizes the platelet clump. If natural fibrinolytic mechanisms, including antithrombin III (which opposes the activity of factors IX, X, XI, XII), are unable to reverse this process, platelets and fibrin continue to accumulate, eventually obstructing the venous channel and propagating clot in both directions from the site of origin.

The factors that predispose to venous thrombosis were first described by Virchow in 1856, and his categorization is still useful today: stasis, hypercoagulability, and endothelial injury.

*Venous stasis* is enhanced by immobility or venous obstruction and contributes to thrombosis by preventing dilution and washout of activated clotting factors. Blood pools in the venous lakes of the calf during bed rest, promoting the development of venous thrombosis during and after surgery or trauma.

*Hypercoagulability* occurs after almost any surgical procedure, trauma, or burn, as well as with widespread malignancies. Thrombosis can be predisposed to by polycythemia vera, which produces thrombocytosis and erythrocytosis, and paroxysmal nocturnal hemoglobinuria. Systemic lupus erythematosus may produce circulating immune complexes (the lupus anticoagulant) which injure

endothelial cells; this paradoxically elevates the activated partial thromboplastin time while predisposing the patient to venous thrombosis. Inherited hypercoagulable states are produced by a deficiency of antithrombin III, protein C (which inactivates factors V and VIII), protein S (a cofactor for protein C activity), or fibrinolytic proteins.

Finally, *direct injury* to the veins is a less frequent contributor to thrombosis but may follow venous trauma, venography, or even thermal injury from curing methyl methacrylate from total joint replacement. Clinical factors which place a patient at risk for thromboembolism are listed below.

Earliest observations suggested that deep thrombi originate in the deep muscular veins of the calf. A study of surgical patients found that most venous thrombi arise in the calf and 20% of these propagate into the popliteal and femoral veins. An autopsy study by Sevitt and Gallagher, however, showed 6 sites where thrombi may form primarily: intramuscular veins of the calf, posterior tibial veins, deep femoral veins, common femoral veins, iliac veins, and popliteal veins.

Thrombi may undergo lysis, organize and recanalize, or embolize to the lungs. While pulmonary emboli may arise from thrombi at any level in the venous system, the most serious threat is from thrombi above the popliteal vein. Large pulmonary emboli may cause sudden death, especially if more than one-third of the pulmonary arterial flow is obstructed. Large emboli produce a failure of oxygenation and acute right ventricular strain as well as bronchospasm. Smaller emboli may cause pulmonary infarction, and over the long term, subacute or occult emboli may cause pulmonary hypertension.

### **Deep Venous Thrombosis:**

Deep venous thrombosis (DVT) is one of the most difficult conditions for the surgeon to diagnose. It frequently occurs without any signs or symptoms; therefore, a high index of suspicion is essential to make the diagnosis. Although the disease may occur in any patient, certain patients have a greater likelihood of developing DVT. Table 1 lists the myriad of risk factors for DVT together with the recommended prophylaxis recommended at FAHC.

*History:* Symptoms usually arise within 7-10 days after surgery or an injury, when the patient is doing well and may even have been discharged. However, DVT may also occur spontaneously in ambulatory patients without a readily apparent cause. Patients may complain of an ache or tightness in the calf, but may also present with complaints of limb swelling. On occasion, the first symptoms may be shortness of breath or hemoptysis from a pulmonary embolism!

*Physical Examination:* There may be a low-grade fever (38°C) days before DVT becomes evident, especially in the post-operative patient. Careful examination of the extremities is mandatory. Dorsiflexion of the foot may cause posterior calf pain (positive Homan's sign) and edema at the calf or ankle may be noted. Measuring the circumference of the calves may help strengthen the clinical impression. In spite of this, however, *the reliability of the clinical examination is no better than 50%*. The diagnosis of DVT should be objectively confirmed before committing the patient to long-term anticoagulant treatment.

Severe iliofemoral thrombosis with massive swelling of the leg is termed *phlegmasia alba dolens*. The term *alba* is used because the leg usually turns white. Simple thrombosis of the iliofemoral system alone probably will not cause this and the perivenous lymphatics need to be involved as well. The perivenous lymphatics are involved in an inflammatory reaction in the presence of venous thrombosis, but why this disease occurs in some patients and not in others is unknown. About 25% of

these patients will develop pulmonary emboli, probably because there are no valves in the iliac veins and because the venous clot is large. Interestingly, this condition also occurs on the left side about 4 times more frequently than on the right. Compression of the left iliac vein by the right iliac artery may account for this difference.

An even more serious condition, probably representing progression of *phlegmasia alba dolens*, is *phlegmasia cerulea dolens*. In this instance, the massively swollen limb is blue, hence the term *cerulea*. In these cases massive edema has increased tissue pressure to the point where capillary and arterial flow ceases, leading to microvascular arterial thrombosis, gangrene, and, if not corrected, limb loss. At this stage, urgent surgical therapy is necessary to prevent amputation.

<b>TABLE – RISK FACTORS</b>						
<b>VTE RISK</b>	<b>SURGERY TYPE<sup>(1)</sup></b>	<b>AGE</b>	<b>ADD'L RISK FACTORS</b>	<b>WITHOUT PROXIMAL DVT %</b>	<b>PROPHYLAXIS CLINICAL PE %</b>	<b>RECOMMENDED<sup>(2)</sup> PROPHYLAXIS</b>
Low	Minor	<40	0	0.4	0.2	None*
Moderate	Minor/Major	any	≥1	2-4	1-2	LMWH or IPC
	Major	40-60	0	2-4	1-2	LMWH or IPC
HIGH	Major	>60	0	4-8	2-4	LMWH or IPC
	Major	40-60	≥1	4-8	2-4	LMWH or IPC
HIGHEST	Major	>40	≥2	10-20	4-10	LMWH <u>and</u> IPC

<sup>(1)</sup>Minor = non-cavitary surgery    Major = cavitary surgery or prolonged procedure

<sup>(2)</sup>Geerts WH, Pineo GF, Heit JA, Bergqvist D, Lassen MR, Colwell CW, Ray JG. Prevention of Venous Thromboembolism: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004;126:338-400.

\*Aggressive ambulation/mobilization encouraged.

### **Risk Factors for VTE**

Surgery  
 Trauma (major or lower extremity)  
 Immobility, paresis  
 Malignancy  
 Cancer therapy (hormonal, chemotherapy, or radiotherapy)  
 Previous VTE  
 Increasing age  
 Pregnancy and the postpartum period  
 Estrogen-containing oral contraception or hormone replacement therapy  
 Selective estrogen receptor modulators  
 Acute medical illness  
 Heart or respiratory failure  
 Inflammatory bowel disease  
 Nephrotic syndrome  
 Myeloproliferative disorders

Paroxysmal nocturnal hemoglobinuria  
 Obesity  
 Smoking  
 Varicose veins  
 Central venous catheterization  
 Inherited or acquired thrombophilia

### **Pulmonary Embolism:**

Pulmonary embolism probably occurs more frequently than is recognized, but it can be difficult to diagnose because of the often subtle nature of the symptoms. The clinical diagnosis of pulmonary embolism is extremely inaccurate because the classic signs and symptoms only occur in 23%-35% of patients. In addition, the signs and symptoms are similar to those of many cardiorespiratory disorders and include dyspnea, pleuritic pain, cough, hemoptysis, and syncope.

Although the exact incidence of this problem is unknown, autopsy studies have determined that 2%-14% of major pulmonary emboli preceded death, although diagnosis before death occurred in only 16%-38%. However, approximately 630,000 cases of pulmonary emboli occur each year in the United States with approximately 200,000 deaths. Eleven percent of these patients die within the first hour after onset, but 89% survive more than 1 hour, or long enough for treatment to be instituted. If adequate therapy is instituted, only about an additional 8% will die, whereas without treatment, about 30% will die.

*History:* With pulmonary embolism, the patient may have no warning that anything is wrong until the advent of sudden chest pain, shortness of breath, and possibly hemoptysis. Apprehension that the patient cannot explain, but is able to express is sometimes a prodrome. Some patients have multiple small pulmonary emboli with essentially no symptoms. They may not mention a slight shortness of breath because they think that the feeling is part of recuperation from surgery. Others will note that there is a "catch" in the chest with deep breathing. In a large study of treatment for pulmonary embolism, the most common symptoms were chest pain (88%), and dyspnea (84%), while apprehension (59%), cough (53%), and hemoptysis (30%) were less frequent. The "classic triad" of chest pain, hemoptysis, and dyspnea is distinctly uncommon and the clinician must be alert for subtle symptoms.

*Physical Examination:* The signs of pulmonary embolism are also relatively non-specific in most patients. In the study mentioned, tachypnea (more than 16 breaths per minute) was the most common sign, occurring in 92%. Other signs include rales (58%), accentuated second heart sound (53%), tachycardia (44%), and fever (43%). *Thus, the signs of pulmonary emboli are non-specific, so that a high index of suspicion, especially in the post-operative patient, is required to appropriately diagnose and treat the problem.*

If a diagnosis of DVT can be made, the occurrence of the above symptoms and signs make a strong clinical case for a diagnosis of pulmonary embolism, justifying the start of treatment. Unfortunately, this occurs in only 32% of patients.

There is no sure way to prevent pulmonary embolism. Early ambulation or in-bed exercise after surgery are of theoretical help and are easily overlooked. The use of heparin or pneumatic compression is a cost-effective approach.

## Venous Stasis Ulceration

Venous stasis ulceration may follow DVT years after the primary event. Often, at the time the patient presents with ulceration, there is no history of prior venous thrombosis. Overall, approximately 20% of patients will develop stasis ulceration.

*Pathophysiology:* Although it is acknowledged that edema plays a role in post-thrombotic venous insufficiency, explaining all the changes by edema alone is difficult for several reasons:

1. Ulceration almost never occurs in patients with lymphedema, even if it is massive.
2. Edema is common in patients with congestive heart failure, renal failure, or severe liver disease, but ulceration is not a part of these conditions.
3. Edema occurs in patients with varicose veins, but ulceration is uncommon.

In anatomic studies, the location of an ulcer was almost always over the 3 lower perforating veins which communicate with the posterior arch vein; these must be incompetent for ulceration to occur. With an incompetent deep system, calf contraction generates high pressure in the distal veins and transmits it to the superficial system via the perforating veins, causing them to become incompetent. Venous hypertension develops, causing edema. This venous hypertension has several additional effects. It may reduce the arteriovenous pressure gradient and lead to tissue hypoxia. The small venules and capillaries dilate with transudation of proteins and red cells into the surrounding tissues, causing the deposition of hemosiderin. Actual rupture of these small vessels may occur and cause extravascular thrombosis, hemosiderin deposition, and fibrosis. Finally, the skin in the area of ulceration drains into the posterior arch vein. This arch vein communicates with the perforating veins and thus receives the high pressure that occurs when the calf contracts.

The key factor in the pathogenesis of these ulcers is the state of the deep venous system. If the veins are open and the valves are intact, ulceration will not occur. On the other hand, if they are occluded or the valves are incompetent, high pressure develops along with bidirectional flow and perforating vein incompetence, leading to edema, fibrosis, and hypoxia of the local tissue, and eventually ulceration.

### Post-thrombotic venous insufficiency:

*History:* Patients with post-thrombotic venous insufficiency usually seek medical attention because of chronic swelling of the lower extremity. The patient may have an open, painful, indolent ulcer that is indurated and has failed to heal after all types of medications have been used, from systemic antibiotics to topical applications. The skin is often sensitive, and itching may be a major problem. Some patients keep the area bandaged to keep from scratching, especially during the night. A history of having DVT may be present, having most often occurred after surgery, trauma, or childbirth. Patients may also complain of leg heaviness and a wooden feeling, particularly after prolonged standing.

*Physical Examination:* Examination always reveals a swollen leg from roughly the level of the malleoli to just below the knee. The edema is firm, but reduces when the legs are elevated each night. The skin is brownish because of hemosiderin deposition and is often scaly, dry, and cracked. Most patients demonstrate chronic changes just above and posterior to the medial malleolus. When ulceration occurs, it varies from being very superficial to relatively deep, penetrating to the subcutaneous tissues.

The ulcer is often large and has a dirty gray base covered with a gelatinous exudate. There is little

granulation in the base of the ulcer and often no evidence of healing, even though most patients have normal arterial inflow and good pulses. The foot is warm (and sometimes warmer than the opposite foot).

## **TREATMENT:**

### **Compression:**

The mainstay of treatment is compression. The goal of treatment is to promote healing of ulcers and prevent recurrence while allowing the patient to ambulate. Compression therapy is highly effective in controlling symptoms of chronic venous insufficiency and promoting healing of ulcers

In the 1950s Conrad Jobst observed that his leg symptoms due to venous ulceration improved when he stood upright in a swimming pool. He designed the first ambulatory gradient-compression stockings to mimic the hydrostatic forces exerted by water.

The effectiveness of compression is presumed to be due to the reduction of ambulatory venous hypertension and a favorable effect on subcutaneous interstitial pressures. Compliance with compression is the main problem in treatment.

Unna developed a paste gauze compression in 1896. The current "Unna boot" includes calamine, zinc oxide, glycerin, sorbitol, gelatin, and magnesium aluminum silicate. The dressing provides both compression and topical therapy. The Unna boot consists of a 3-layer dressing: (1) Dome paste rolled-gauze bandage with graded compression from the forefoot to just below the knee, (2) 4-inch-wide continuous gauze dressing and (3) elastic wrap with graded compression. The dressing becomes stiff after drying. The Unna boot has better patient compliance than compression stockings.

### **Vena cava filters:**

Filters are placed in the vena cava to trap potential emboli while maintaining the flow of blood. Although there are a variety of filters available, the greatest experience has been obtained with the Greenfield vena cava filter. This filter may be placed operatively or percutaneously via the jugular or femoral veins. The filter is positioned below the renal veins centered by a guide-wire and, when released, the legs spring open so that the small hooks on the end catch and hold the filter in the vena cava. The Greenfield filter is remarkably successful, trapping emboli as small as 3 mm and maintaining long-term patency in 97%.

The current indications for Greenfield placement are a contraindication to anticoagulation or pulmonary emboli while on adequate anticoagulation treatment. In certain high-risk groups, such as the multiply injured patient with paralysis, prophylactic filter insertion may be appropriate. Recently, retrievable IVC filters have become available expanding their use.

### **LYMPHEDEMA:**

Another common cause of "the swollen leg" that is frequently referred to is the patient with lymphedema. In addition to arteries and veins, the lymphatics compose a third part of the vascular system. Fluid, electrolytes, and protein move through the wall of the capillary into the interstitium. Only the fluid and

electrolytes can be reabsorbed through these vessels; the protein is absorbed through the lymphatic system. The lymphatics can be viewed as a drainage system that collects protein, water, bacteria, and other small particulate matter from the interstitium.

*Anatomy:* A superficial layer of lymphatic capillaries lies within the dermis of the skin and another layer in the subcutaneous tissue. These join the lymphatics in the deeper layers that follow the course of the major blood vessels. Eventually, the lymphatics empty into the venous circulation at the junction of the internal jugular and subclavian veins. Somewhat similar to the veins, the lymphatics contain 1-way valves that direct the flow of the lymph toward the neck. The lymph channels course through the lymph nodes on their way to the neck, and these act as filters to remove certain particulate matter and to add lymphocytes to the circulation. Respiratory movement, muscle contraction, arterial pulsation, and massage aid the lymphatic "circulation." Lymph nodes consist of collections of lymphatic tissue and range in size from 1 mm to 2 cm. Humans have approximately 1,000 nodes, which lie along the course of the lymphatic vessels. The afferent lymph vessels enter the node over its surface, but drainage from the node is through a single efferent lymphatic that exits the node at its hilum along the artery and vein of the node.

*Physiology:* As a circulatory system, the lymphatics protect tissue pressure and volume. During hypovolemia, it supports the circulation by immediate repletion of the intravascular volume from its own stores. Blood passing through a capillary gives up water, electrolytes, and protein into the interstitium. Some of this is reabsorbed at the venous end of the capillary. However, this system cannot reabsorb all the fluid and protein, so the lymphatics serve as a third element of the circulatory system and return fluid, electrolytes, and protein to the venous circulation via the thoracic duct. Absence or obstruction of the lymphatic system produces chronic edema.

*Classification:* Lymphedema may be primary, due to an idiopathic abnormality, or secondary, following lymphatic destruction by surgery, radiation, or infection. Worldwide, filariasis is the most common cause of secondary lymphedema, while the former 2 secondary etiologies are more common in this country.

Primary lymphedema may be *congenital*, presenting early after birth, *lymphedema precox*, presenting in second or third decades, or *lymphedema tarda*, developing after age 35. Each type may be due to aplasia, hypoplasia, or dysplasia of the lymphatic vessels.

*Pathophysiology:* The lymphatics play a major role in removing protein from the interstitium. If they are absent or hypoplastic, protein collects and holds water in the tissues causing the edema. The protein content of fluid has been shown to exceed 2 g/dL of fluid, which is abnormal. In secondary lymphedema, the problem is almost always caused by obstruction. Surgery, infection, tumor invasion, or radiation may all cause the obstruction. Filariæ transmitted by a mosquito bite will enter the lymphatics where the microfilariae develop into the adult worms and clog the lymphatic channels. This clogging produces massive edema (elephantiasis).

*History:* Patients with *lymphedema precox* usually seek medical attention because of chronic swelling of an extremity, usually a lower extremity. Before seeking medical help, many have had the condition for some time, often years. Some diminution in edema usually occurs at night, but with time, this becomes negligible. The involved extremity feels heavier and may have a "wooden" feeling or diminished sensation. The cosmetic appearance is troublesome and occasionally actual physical impairment may occur.

Patients with primary lymphedema will usually not give a history of injury or surgery. It is not

infrequent that they have had a diagnosis of DVT which may not have been documented objectively. Past episodes of lymphangitis or cellulitis are also frequent.

*Physical Examination:* Examination reveals a swollen leg with normal skin color and temperature. The edema is firm, rubbery in consistency, and non-pitting. Nodes are not palpable in the groin, and there may be no evidence of trauma, surgery, or infection. A frequent complication of lymphedema, because of impaired local tissue defenses, is *cellulitis*, so that examination may reveal the typical tenderness and erythema indicative of infection.

There is little need to carry out any further studies because the diagnosis of *lymphedema precox* is satisfactorily made on clinical grounds. The absence of any of the causes of secondary lymphedema makes the diagnosis of primary lymphedema certain. Occasionally, it may be necessary to rule out the coexistence of DVT with duplex scanning or venography.

### References:

1. Goldhaber SZ, ed: Pulmonary Embolism and Deep Venous Thrombosis. Philadelphia: W.B. Saunders Co; 1985.
2. Hirsch J, Hull RD: Venous Thromboembolism: Natural History, Diagnosis, and Management. Boca Raton, Fla: CRC Press, Inc; 1987.
3. Ricci, MA, Emmerich J, Callas PW, et al: Evaluating Chronic Venous Disease Using the New Venous Severity Scoring System. J Vasc Surg 2003;38:909-15.
4. Talbot SR, Oliver MA: Techniques of venous Imaging. Pasadena, Calif: Appleton Davies, Inc; 1992.
5. Geerts WH, Pineo GF, Heit JA, et al. Prevention of Venous Thromboembolism: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 2004;126:338-400.

## **Thoracic Outlet Syndrome (TOS)**

---

By Steven R Shackford, MD

## I. Definition and pathophysiology

TOS is produced by compression of one of the neurovascular structures in the thoracic outlet.

TOS is categorized in the following types:

1. Neurogenic TOS is produced by compression of the brachial plexus (usually the lower trunk). It is the most common form.
2. Venous TOS is also called "effort thrombosis" or Paget-Schrotter syndrome and is produced by compression of the subclavian vein.
3. Arterial TOS is the least common. It is always associated with a cervical rib or osseus abnormality of the outlet.

## II. Diagnosis

At the present time there are no generally accepted criteria for the diagnosis of TOS.

### A. General Exam

The first part of an evaluation of a patient suspected of having TOS is a good history and physical examination.

### B. Neurogenic TOS

1. Differential diagnosis
  - a) Degenerative disk disease of the cervical spine
  - b) Carpal tunnel syndrome
  - c) Ulnar nerve entrapment
  - d) Rotator cuff tear

2. Testing

The purpose of doing physiologic testing and imaging is to rule out other causes for neurogenic TOS. If all other causes are ruled out and the history and physical examination suggest TOS, then treatment should be undertaken. Testing involves the following:

- a) Plain films of the cervical spine to rule out a cervical rib.
- b) Electromyogram/nerve conduction velocity testing to rule of carpal tunnel syndrome and ulnar nerve entrapment.
- c) MRI of the cervical spine to rule out degenerative disk disease.
- d) MRI of the shoulder to rule out a rotator cuff tear.

### C. Venous TOS

Venous TOS can be produced by seemingly minimal effort. It usually presents with a painful swollen arm. The diagnosis can be made by venous duplex exam or venography.

### D. Arterial TOS

Arterial TOS can present with limb threatening ischemia or with upper extremity claudication. It usually is associated with some degeneration in the arterial wall producing either an aneurysm or pseudoaneurysm. All patients suspected of arterial TOS should have an arteriogram.

## III. Treatment

### A. Neurogenic TOS

Initially, neurogenic TOS is treated with analgesia, muscle relaxants, dry heat, and physical therapy exercises to strengthen the shoulder girdle. If symptoms persist after a minimum of 6 months of medical therapy, one may consider a radical scalenectomy, a first rib resection, or both.

Surgical treatment of neurogenic TOS requires a minimum of 2-years follow-up, since recurrences and relapses are frequent. At 2 years, most authors report a cure rate of 10%-15%, and an improvement rate of approximately 40%. Fifty percent of patients at 2 years are unimproved. These poor results are probably due to an inadequate workup of the patient (i.e., the patient received a first rib resection because the diagnosis of neurogenic TOS was made when the patient actually had degenerative disk disease of the cervical spine).

#### B. Venous TOS

Venous TOS can be managed with thrombolytic therapy if the diagnosis is made within 72 hours of the onset of symptoms. During this period, the clot is quite vulnerable to thrombolytics and will require approximately a 24-hour infusion. If this is successful, the patient should undergo first-rib resection during the same hospitalization. If the diagnosis is delayed more than 72 hours, the patient should be placed on heparin and then Coumadin for a minimum of 3 months. Initially, the patient should do minimal activity with the arm. As symptoms resolve with anticoagulation (and they will resolve with anticoagulation), the patient is encouraged to increase activity. If symptoms persist with activity (swelling, venous claudication, etc.) then the patient should be considered for first-rib resection. Generally, good results with improvement are reported in up to 85%-90% of patients.

#### C. Arterial TOS

The management of arterial TOS varies. If there is no arterial pathology, a simple removal of the compressive agent (i.e., the first rib, hypertrophic callus from a fractured clavicle, etc.) is all that is needed. On the other hand, if an aneurysm has developed, then arterial resection using the supraclavicular approach is necessary.

Results following resection and decompression of the outlet for arterial thoracic outlet syndrome are good, with 100% limb salvage.

#### References:

1. Schmacht DC, Back MR, Novotney ML, Johnson BL, Bandyk DF. Primary axillary-subclavian venous thrombosis: Is aggressive surgical intervention justified? *Vasc Surg* 2001;35:353-359.
2. Scher LA, Vieth FJ, Haimovici H, Samson RH, Acsher E, Gupta SK, Sprayregen S. Staging of arterial complications of a cervical rib: Guidelines for surgical management. *Surgery* 1984;95:644-649.

3. Urschel HC, Razzuk MA: Paget- Schrotter syndrome: What is the best management? Ann Thoracic Surg 2000;69:1663-1669.
4. Machleder HI: Thrombolytic therapy and surgery for primary axillosubclavian thrombosis. Current Approach. Semin Vasc Surg 1996;9:46-49.
5. Nehler MR, Taylor LM, Moneta GL, Porter JM: Upper extremity ischemia from subclavian artery aneurysm caused by bony abnormalities of the thoracic outlet. Arch Surg 1997;132:527-532.
6. Leffert RD, Porter JM: Thoracic outlet syndrome: Results of 282 first rib resections. Clin Ortho Rel Res 1999;368:66-79.
7. Jamieson WG, Chinnick B: Thoracic outlet syndrome: Fact or fancy? A review of 409 consecutive patients who underwent operation. Can J Surg 1996;39:321-326.

## ***Non-invasive Vascular Lab***

---

Michael A Ricci, MD and David Pilcher, MD

### **Learning Objectives**

#### **I. Understand the basic techniques used in the non-invasive vascular laboratory**

- A. Explain the principles of the Doppler effect and Doppler ultrasound.
- B. Understand the principles and interpretation of segmental pressures and waveforms (PVRs).
- C. Understand the basic principles behind duplex ultrasound (imaging and Doppler spectral analysis) as well as the Strandness criteria for diagnosis of carotid stenosis.
  1. Criteria for > 50% stenosis: PSV > 125cm/sec (or >140cm/sec with contralateral occlusion)
  2. Criteria for > 80% stenosis: above plus EDV > 105cm/sec
- D. Understand the basics of aortic aneurysm measurement with B-mode ultrasound.
- E. Understand the imaging and physiologic basis for testing for DVT.
  1. Lack of compression of vein indicates acute clot.
  2. Normal venous flow is spontaneous and phasic with respiration augmentation.
  3. Normal flow augments with distal compression or release of proximal compression.

#### **II. Understand techniques' applications to specific disease states.**

- A. Carotid Duplex
  1. To describe the normal Doppler signals in the internal, external, and common carotid arteries.
  2. To discuss the sensitivity and specificity of duplex scanning in detecting carotid artery stenosis.
  3. To discuss the risks and benefits of relying on duplex ultrasound and eliminating angiography.

- B. Describe the utility of ultrasound in diagnosing life or limb threatening signs of vascular disease
1. Symptomatic or ruptured AAA: Consider this diagnosis in any patient presenting with severe back or flank pain and an abdominal mass, especially male smokers aged > 50 years. Ultrasound may be useful in the acute setting if the patient is obese and the aortic pulsation cannot be palpated and is hemodynamically stable.
  2. Suspected chronic mesenteric ischemia: duplex is an initial screening test for patients with a clinical suspicion of the disease.
  3. Acutely ischemic lower extremity. Duplex may be useful to diagnose an embolism to the femoral bifurcation or in proving the diagnosis of a thrombosed popliteal artery aneurysm.

### Introduction

Although almost every artery in the body can be interrogated with the duplex scanner, a few of these are readily accessible for the surgeon caring for a variety of common vascular problems. Vascular diagnostic ultrasound adds more complexity to the examination than simple imaging. As always, the surgeon's complete knowledge of anatomy is of the greatest benefit in the performance and interpretation of these studies. Duplex or color-flow ultrasound scanning is a sensitive, noninvasive technology that allows visualization of the flow pattern within a vessel or graft, anatomic localization of stenosis or abnormality, and physiologic measurement of velocity and flow.

### Ultrasound Principles

An ultrasound transducer transmits ultrasound waves that are reflected by an interface (different stiffness/density change) back to a receiver. The more sound that is reflected back, the brighter the display, hence the term "B-mode" or brightness mode. The originally transmitted sound waves undergo a detectable change in frequency when they are reflected by moving red blood cells (Doppler effect).

The change in frequency ( $\Delta f$ ) is transformed by the scanner's computer to produce a spectrum of all the frequencies present at a given point in the vessel (spectral analysis) that are useful in diagnosing stenosis. Combining these 2 components produces the conventional "duplex" scanner. Some equipment adds a third component. Simultaneous receiving from a multitude of sites records  $\Delta f$  for essentially every moving point in the field. A color code is assigned to each point (blue= away from the transducer, red= towards the transducer, white = high velocity suggesting stenosis) and the image is continuously updated so that a dynamic real-time ultrasonic arteriogram-like image is created.

Because stenosis produces an increased velocity ( $v$ ) of flow, the reflected frequency ( $\Delta f$ ) detected is related to velocity by the following equation:

$$v \text{ (cm/sec)} = (\Delta f \cdot c) / (2F_0 \cdot \cos\theta)$$

where  $c=1540$  m/sec,  $F_0$ =transmitted frequency, and  $0 - 60^\circ$  (incident angle). Convention now usually expresses  $\Delta f$  after it is converted (by the scanner) to velocity, a more clinically useful and understandable number.

### Basic Scanning Technique

The patient should be recumbent and supine for most examinations. When examining the popliteal region, the knee should be flexed slightly and the leg externally rotated or the patient put into the prone position. Elevation of the left flank may occasionally help in visualizing the aorta.

Most exams can be done with a 5 MHz probe. A deep abdominal probe (3.5 MHz) will facilitate visualization of the aorta, while a 7.5 MHz probe may be needed for superficial grafts or other peripheral structures in very thin people. Overall, body habitus may affect the choice of probes since the vessels of an obese patient may be better visualized with the deep probe or those of a very thin person with the superficial probe.

A thorough knowledge of the venous and arterial anatomy is mandatory. Arterial structures are easily identified by the pulsatile motion of the walls on real-time grey-scale image, which may be confirmed by placing the Doppler cursor in the center of the structure. Arterial signals and spectral analysis will be found. Pitfalls in identification may include very low-flow states or the presence of an arteriovenous fistula. Each vein is adjacent to its corresponding artery, which may help in identification.

### **PVR (pulse volume recording)**

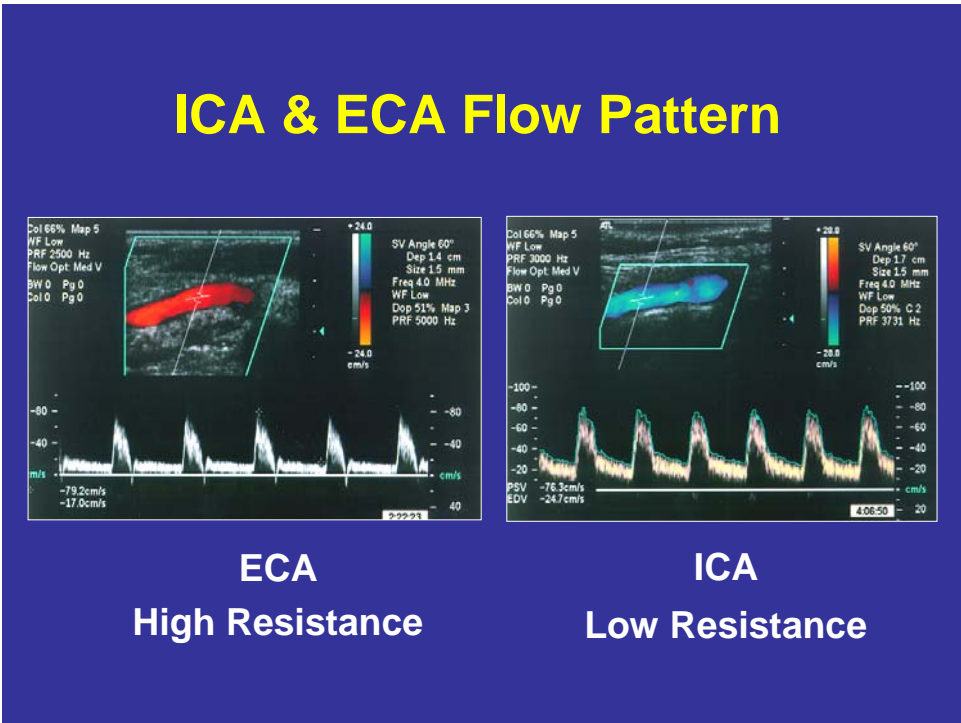
A pressure transducer coupled to the air bladder on a cuff inflated above venous occlusion pressure traces out a waveform which resembles an arterial pressure wave. A rapid upslope and sharp peak and high amplitude are consistent with normal arterial inflow at the level where the measuring cuff is applied. Damped upslope, rounded peak, and low amplitude are suggestive of arterial occlusion/stenosis above the cuff level. A flat waveform is consistent with ischemic changes.

This PVR study is usually coupled with segmental blood pressure obtained by the systolic pressure required to obliterate a distal Doppler signal. These pressures should always be obtained in the supine position to obviate gravity augmentation. Comparison to arm pressure gives an ankle-to-brachial (ABI) pressure ratio. One is normal with no arterial compromise at the ankle compared to the arm. Since arm stenosis is much rarer than leg stenosis, this usually works. An abnormal arm arterial flow would be shown by an abnormal PVR waveform. An ABI of  $<0.5$  is consistent with claudication, and an ABI of  $<0.3$  with ischemia.

In some laboratories the Doppler waveform at various levels is used to detect and stratify stenosis instead of the PVR waveform.

### **SPECIFIC DISEASE STATES:**

**Carotid stenosis.** While each of these aspects is important during the cerebrovascular duplex examination, the most important aspect of diagnosis is the spectral analysis. The B-mode or color image serves to direct the placement of the Doppler for sampling. Obtain multiple samples from the midstream of the vessel being interrogated. The angle of incidence of the ultrasound beam should be kept at  $60^\circ$  or less. Any subsequent scans should be performed at the same angle. Spectral analysis should first be obtained low in the CCA and in its mid and distal portions, in the ECA, through the carotid bulb, and throughout the ICA. Specific parameters to be recorded include: peak systolic velocity (PSV), end diastolic velocity (EDV), the ratio of the PSV in the ICA and CCA (PSV ICA/CCA ratio), and the ratio of the EDV in the ICA and CCA (EDV ICA/CCA ratio). Obviously, any areas suspicious for stenosis on the basis of the color pattern should be thoroughly investigated. The spectral profile in the CCA typically demonstrates a mix of the low resistance pattern seen in the ICA (flow-through diastole) and the high resistance pattern (no-flow during diastole) of the ECA (Figure 1).



**Figure 1.** Normal Doppler spectrum in the external and internal carotid arteries.

A high-resistance profile in the CCA may be the first clue to an ICA occlusion. As noted above, a brief flow-reversal in the carotid bulb is a normal finding. In addition, there is a narrow spectrum of velocities in undiseased vessels known as a *spectral window*. Stenosis within the bulb and ICA produces turbulence that diminishes or obliterates the window with *spectral broadening*, one of the earlier signs of stenosis. Spectral broadening can also be artificially generated if the sample volume is too large or the sample is too close to the vessel wall (as opposed to midstream sample) or in tortuous vessels. In addition, if the gain is too high, the spectral window will be obliterated with artificially elevated velocities. The gain should be set to a minimal but discernable amount of background noise. Published duplex criteria for the degree of carotid stenosis is shown in Table 1.

**Table 1. Criteria for Duplex Diagnosis of ICA Stenosis.**

meta analysis 2005 (3)	FAHC criteria
------------------------	---------------

<b>Normal</b>	PSV < 130 cm/sec No spectral broadening Flow reversal in bulb	<b>Normal</b>	PSV < 125 cm/sec PSV ICA/CCA ratio <2.0 EDV ICA/CCA ratio <2.0 No spectral broadening
		<b>1- 49%</b>	PSV<125 cm/sec PSV ICA/CCA ratio <2.0 EDV ICA/CCA ratio <2.0 Minimal spectral broadening
<b>50 – 79%</b>	PSV > 130 cm/sec	<b>60% – 79%</b>	PSV > 125 cm/sec EDV > 50 cm/sec PSV ICA/CCA ratio 2-4 EDV ICA/CCA ratio 2-4 Marked spectral broadening
<b>80 – 99%</b>	PSV > 200cm/sec	<b>80% – 99%</b>	PSV>125 cm/sec EDV > 105 cm/sec PSV ICA/CCA ratio >4
<b>Occlusion</b>	No flow	<b>Occlusion</b>	No flow in ICA Low velocity CCA to zero in diastole
PSV=peak systolic velocity, EDV=end diastolic velocity			

**Abdominal Aortic Aneurysm.** Ultrasound is the most useful technique to diagnose and follow aortic aneurysms, and remains superior to physical exam which, even with experience, misses as many as 20% of aneurysms. Ultrasound has the great advantage of allowing the sonographer to measure a true transverse diameter, even in the most tortuous aortas. Bowel gas or a recent meal may make the exam difficult. Ultrasound may also be useful in diagnosing associated popliteal aneurysms (10%-25%).

It is important **not** to use ultrasound to diagnose a ruptured AAA since it is inaccurate in this regard. However, emergency ultrasound of the aorta may be useful to rule out (or confirm) an aneurysm in a patient who is in shock without apparent reason, particularly in a large patient in whom an aneurysm might not be palpable.

**Pseudoaneurysm.** In our institution, the overall incidence of arterial injury after cardiac catheterization is 1.4%, but this rises to 3.1% when angioplasty is performed. The most frequent of these complications is pseudoaneurysm formation. These are more frequent in the femoral region, whereas thrombosis is most frequent in the brachial region. Recent evidence suggests that about 60% will spontaneously thrombose without patient morbidity. Ultrasound is useful to confirm the diagnosis and follow the patent aneurysm. Ultrasound-guided thrombin injection is effective in obliterating these pseudoaneurysms.

**Popliteal Artery Aneurysm.** Ultrasound is the test of choice for diagnosing this condition and differentiating the arterial pathology from venous thrombosis or a popliteal synovial cyst. It may also prove useful when patients present with an acutely ischemic limb and normal (or supranormal) contralateral pulses. However, even in the patient who presents with unilateral claudication, the

suggestion of a popliteal aneurysm may be easily and more quickly confirmed by ultrasound. It is important to remember that the chance of bilateral popliteal artery aneurysms when one is found is 50% and the likelihood of an AAA being present may be as high as 50%.

**Graft Surveillance.** With the widespread use of the *in situ* graft, surveillance with duplex ultrasound is quite simple and useful. Early detection of a failing graft will produce better long-term patency. Although color-flow imaging speeds up the exam, it is not an absolute necessity. Criteria that suggest the graft is at risk are: (1) graft velocity < 40 cm/sec, or (2) focal stenosis. A focal stenosis may easily be seen with color-flow as a white "jet". However, when a high velocity is encountered relative to the immediately proximal graft segment ( $\geq 2X$ ), a significant stenosis is present. In our institution, angiography is performed and revision is accomplished by open or percutaneous methods as appropriate.

### References:

1. Ricci MA, Waters MA, Peate D: The role of noninvasive studies in the diagnosis and management of cerebrovascular disease. In; RB Rutherford, ed. Vascular Surgery, 6<sup>th</sup> ed. Philadelphia, Pa: WB Saunders Co; 2005:1957-73.
2. Ricci MA: The changing role of duplex scan in the management of carotid bifurcation disease and endarterectomy. *Sem Vasc Surg* 1998;11:3-11.
3. Jahromi AS, Cina CS, Liu Y, Clase CM. Sensitivity and specificity of color duplex ultrasound measurement in the estimation of internal carotid artery stenosis: A systematic review and meta-analysis. *J Vasc Surg* 2005;41:962-72.
4. Khoury M, Rebecca A, Greene K, et al. Duplex scanning guided thrombin injection for treatment of iatrogenic pseudoaneurysms. *J Vasc Surg* 2002;35:517-521.
5. Zweibel WJ, Pellerito JS. Introduction to Vascular Ultrasonography, 5<sup>th</sup> ed. Philadelphia Pa: WB Saunders; 2004.
6. Taylor BS, Rhee RY, Muluk S, et al. Thrombin injection versus compression of femoral artery pseudoaneurysms. *J Vasc Surg* 1999;30:1052-9.

## Arterial Imaging: CT Angiography, MR Angiography, and

# Conventional Catheter Angiography

By Christopher S. Morris, MD

## Learning Objectives

- I. Basic principles of each imaging modality
  - a. CT angiography (CTA)
  - b. magnetic resonance angiography (MRA)
  - c. conventional catheter angiography or digital subtraction angiography (DSA)
  - d. 3-D rendering techniques
- II. Utility of each imaging modality in specific disease entities
  - a. Vascular trauma
  - b. Peripheral vascular occlusive disease: lower extremity ischemia, mesenteric ischemia, and renovascular ischemia
  - c. Carotid artery occlusive disease
  - d. Abdominal aortic aneurysm (AAA) and peripheral vascular aneurysm disease
  - e. Miscellaneous: thoracic outlet syndrome and effort thrombosis, popliteal artery entrapment, adventitial cystic disease of the popliteal artery, and vasculitis
- III. Patient preparation and complications of each imaging modality
- IV. Contrast agents
  - a. Iodinated contrast media
  - b. Alternative contrast agents for conventional catheter angiography
  - c. MR contrast agents – gadolinium and gadolinium chelates
- V. Adverse reactions to contrast media

Vascular imaging, including CTA, MRA, and conventional catheter angiography, is instrumental in arriving at a diagnosis in vascular disease, and also aids in medical, surgical, or endovascular treatment planning. The choice of modalities may depend on such issues as the patient's comorbidities, the relative likelihood of surgical or endovascular treatment, and the degree of spatial and contrast resolution required of the imaging study. Patients lacking femoral pulses, as seen in aortoiliac occlusive disease, may be better served by CTA or MRA than by catheter angiography, which might require a riskier axillary artery puncture for access. However, in patients with a suspected short segment iliac artery stenosis, a catheter angiogram may be preferred, since endovascular treatment with a stent and angioplasty could be performed at the same sitting. In patients where fine vascular detail is paramount, such as magnified hand angiography of ischemic fingers, a catheter angiogram will be required. Finally, patients with renal insufficiency have the 3 following basic options for imaging: (1) prophylactic medications prior to catheter angiogram with iodinated contrast media, (2) catheter angiogram using a non-renal toxic alternative contrast agent such as CO<sup>2</sup> or gadolinium, or (3) gadolinium-enhanced MRA.

## Digital subtraction angiography (DSA)

Conventional catheter angiography is the gold standard of vascular imaging modality, and continues to serve as the reference study to which all other imaging modalities are compared.

Currently, modern digital subtraction angiography (DSA) imaging chains produce the highest resolution (2K x 2K) images of the vasculature that can be reconstructed in 3-D with the use of rotational angiography protocols. However, catheter angiography is invasive (contrast media is injected into an artery or vein), time-consuming, and expensive. Major complications (which should not exceed 5%) include the following: groin hematoma; hemorrhage; puncture site pseudoaneurysm; vascular injury such as intimal dissection or perforation; catheter or guidewire-induced atheromatous embolization, including stroke; contrast-induced nephrotoxicity; and allergy-like adverse reactions to iodinated contrast media. Therefore, the decision to perform catheter angiography as the first vascular imaging test depends on patient characteristics and specific disease entities.

Specific disease entities that are best interrogated with DSA include vasculitis, upper extremity ischemia, non-occlusive and thromboembolic mesenteric ischemia, cerebral ischemia and stroke, and small vessel atherosclerotic occlusive disease when a distal target for a surgical bypass graft must be characterized. These entities require optimal spatial resolution for diagnosis and treatment planning.

An attractive component to DSA is the therapeutic endovascular option, which can be performed at the same sitting, adding only a small amount of time to the procedure. Carotid, subclavian, renal, mesenteric, aortic, and iliac artery stenting have been recognized as durable revascularization procedures. Femoral, popliteal, and tibioperoneal artery angioplasty can also be performed, with less success. In general, the more favorable and durable results with angioplasty or stenting are found in larger vessels, particularly above the level of the common femoral arteries, short segment stenoses or occlusions less than 3 cm in length, and non-calcified concentric lesions with less elastic recoil. Adjunctive therapies, such as cutting balloon angioplasty, cryoplasty, atherectomy, transcatheter embolization, covered stent (stent-graft) placement, and thrombolysis, can all be performed during the initial diagnostic DSA procedure as well.

There are several strategies for patients with renal insufficiency who need a DSA with iodinated contrast media. Hydration before the procedure is important in patients at risk for all of the following protocols. Currently, a popular protocol for prophylaxis against contrast-induced nephrotoxicity consists of administering 3 cc/kg of sodium bicarbonate (154 mEq/L) for the hour before the procedure, and then 1 cc/kg per hour for 6 hours after the procedure. This solution can be roughly prepared by placing 3 ampules of sodium bicarbonate in 1 L of D5W, or it can be precisely ordered from the pharmacy at 154 mEq of sodium bicarbonate/L. Alternatively, the antioxidant, N-acetyl cysteine, can be administered orally the day before and the day after the procedure at a dose of 600 mg BID. Lastly, alternative contrast agents are often used in patients with renal insufficiency. Carbon dioxide (CO<sup>2</sup>) is commonly used and has not been associated with renal toxicity, even in patients with severe renal dysfunction. CO<sup>2</sup> is readily available, inexpensive, and well-tolerated by patients. However, it produces less image contrast than standard iodinated agents, which may detract from its diagnostic utility. Gadolinium is also radio-opaque, relatively nontoxic to the kidneys, and can also be used as an alternative contrast agent.

Patient preparation before a DSA examination includes restriction to clear liquids after midnight, since intravenous conscious sedation will probably be administered. The patient should be well hydrated, if feasible. In addition, gastrointestinal barium studies, including CT scans, should be avoided for up to 1 week before the angiogram, if possible.

### **CT angiography (CTA)**

CT angiography (CTA) has experienced an explosion in technology over the past several years. Like catheter angiography, it requires an injection of iodinated contrast material, but it is injected in a tight bolus fashion intravenously through a peripheral intravenous cannula, with the CT scanning timed to occur during the arterial and/or venous phases. Multislice (the most advanced current scanners are 64 multislice) helical scanners can image the entire body within seconds and can create sections as thin as 0.4 mm through reformatting techniques. The vascular structures with a high concentration of iodinated contrast media are selected and 3-D data sets are reconstructed. The following rendering techniques are used: (1) maximum pixel intensity (MIP), which are most analogous to DSA images, (2) multiplanar reformat (MPR), which allows vascular structures to be viewed in different planes, (3) radial curved planar reformat (CPR), which will “straighten” out arteries in different planes, and (4) volume rendering, which allows for color shading and semitransparency). In general, CTA produces a higher spatial resolution image than MR angiography (MRA). In addition, CTA is isotropic, which improves 3-D reconstructions.

In patients with normal renal function, CTA is the non-invasive vascular imaging procedure of choice. Specific disease entities that are best imaged with CTA include AAA (both initial diagnosis, follow-up, and pre-operative or pre-endograft planning), peripheral arterial aneurysm disease, vascular trauma, lower extremity peripheral vascular occlusive disease, proximal mesenteric ischemia, and renovascular disease. It may also be very helpful in carotid artery disease, thoracic outlet syndrome, and miscellaneous entities such as popliteal artery entrapment.

If at all possible, patient preparation should include hydration prior to the procedure. Avoid gastrointestinal barium studies for at least 1 week prior to the CTA for best results. In patients with renal insufficiency, MRA should be considered, or prophylactic maneuvers, as described in the DSA section, should be used.

Complications associated with CTA include contrast-induced nephrotoxicity, idiosyncratic “allergic” type adverse reactions to contrast media, and extravasation at the injection site.

## **MRA**

In patients with renal insufficiency or a history of an adverse reaction to iodinated contrast media, MRA should be considered. There are many different MRA techniques, but 2-D and 3-D time-of-flight and 3-D gadolinium-enhanced MRAs are the most common at our institution. Time-of-flight imaging is sometimes used for MR venography and is an older, more cumbersome method of visualizing the vasculature. Gadolinium-enhanced MRA requires the intravenous bolus injection of gadolinium, with imaging within the coronal plane occurring during the arterial, venous, and/or equilibrium phases. Similar to CTA, the bolus of contrast medium (gadolinium) can be followed down the arterial tree, beginning in the abdomen, continuing to the pelvis and thighs, and finally including the knees, calves, and feet. Also, like CTA, 3-D reconstructions can be created for easier viewing of the vascular tree. However, since the smallest imaging units (voxels) are not isotropic, the 3-D capabilities are somewhat limited compared to CTA.

The best use for MRA includes vascular imaging in patients with underlying renal insufficiency. Specific disease entities best depicted with MRA are similar to those for CTA, although the spatial resolution for MRA is somewhat lower than that of CTA.

Patient preparation for MRA is minimal. The patient will be placed into the bore of the magnet for a variable amount of time, so claustrophobia may need to be treated with sedatives. Complications include extravasation at the injection site.

Alternative paramagnetic contrast agents other than gadolinium are under investigation. In addition, a gadolinium chelate blood pool agent, MS-325 (Vasovist, Epix Medical) has the advantage of binding to albumin and remaining in the vessels for a long time.

### References:

1. Merten GJ, Burgess WP, Gray LV, et al. Prevention of contrast-induced nephropathy with sodium bicarbonate. A randomized controlled trial. *JAMA* 2004; 291:2328-2334.
2. Tepel M, van der Giet M, Schwarzfeld C, Laufer U, Liermann D, Zidek W. Prevention of radiographic-contrast-agent-induced reductions in renal function by acetylcysteine. *N Engl J Med* 2000; 343:210-212.
3. Hiatt MD, Fleischmann D, Hellinger JC, Rubin GD. Angiographic imaging of the lower extremities with multidetector CT. *Radiol Clin North Am.* 2005; 43:1119-1127.
4. Kock MC, et al. DSA versus multi-detector row CT angiography in peripheral arterial disease: randomized controlled trial. *Radiology* 2005; 237:727-737.
5. Ouwendijk R, de Vries M, Pattynama PM, et al. Imaging peripheral arterial disease: a randomized controlled trial comparing contrast-enhanced MR angiography and multi-detector row CT angiography. *Radiology* 2005; 236:1094-1103.
6. Leiner T, Kessels AG, Schurink GW, et al. Comparison of contrast-enhanced magnetic resonance angiography and digital subtraction angiography in patients with chronic critical ischemia and tissue loss. *Invest Radiol.* 2004; 39:435-444.
7. Martin LG, Rundback JH, Sacks D, et al. Quality improvement guidelines for angiography, angioplasty, and stent placement in the diagnosis and treatment of renal artery stenosis in adults. *J Vasc Interv Radiol* 2002; 13:1069-1083.
8. Spinosa DJ, Matsumoto AH, Angle JF, Hagspiel KD, McGraw JK, Ayers C. Renal insufficiency: usefulness of gadodiamide-enhanced renal angiography to supplement CO<sub>2</sub>-enhanced renal angiography for diagnosis and percutaneous treatment. *Radiology* 1999; 210:663-672.
9. Oliva VL, Denbow N, Therasse E, et al. Digital subtraction angiography of the abdominal aorta and lower extremities: carbon dioxide versus iodinated contrast material. *J Vasc Interv Radiol* 1999; 10:723-731.
10. Strandness DE Jr, van Breda A. *Vascular Diseases. Surgical and interventional therapy.* New York, NY: Churchill Livingstone; 1994.
11. Kadir S. *Diagnostic Angiography.* Philadelphia, Pa: W B Saunders Company; 1986.