Vascular injuries

1. The progress in the techniques of resuscitation and vascular repair have resulted in the progressive reduction in the amputation rate in major limb injuries. While the amputation rate was
   - nearly 80% in the battle injuries to limbs in World War I (1914/18)
   - it fell to 50% in the World War II (1939/44) and
   - it was further reduced to around 10% in the more recent wars
If not recognized and treated rapidly, injuries to major arteries, veins, and nerves may have disastrous consequences resulting in the loss of life and limb.

   Incidence - between 0.07 and 2.5% from total of trauma

   In orthopedic field – vascular trauma exceed

   5% (Candea) to 6.5% (Bishara).

2. years of productive life lost (YPLL) if the patient would survive

   For each traumatic death there are, on average: 36 YPLL

   compared with 16 for cancer and

   12 for cardiovascular diseases.
3. **CLASSIFICATION**

The following are the types of injuries to arteries.

- **Open injuries** produce
  
  a) Division or laceration of the artery  
  b) Traumatic false aneurysm (Pulsating Haematoma)  
  c) Arterio-Venous Fistula

- **Closed injuries** produce
  
  a) External compression  
  b) Arterial spasm  
  c) Thrombosis following intimal tears

Closed injuries are the commonest cause of acute traumatic ischaemia in a limb.

4. **ETIOLOGY** – Closed and open injuries

The cause of vascular obstruction could be:

a) outside the wall of the vessel  

b) in the wall  

c) inside the lumen of the vessel

- **outside** - External compression of the vessels can be caused by
  
  a) tight plaster, tight bandages, etc.,  
  
  b) subfascial haematoma in places like cubital fossa, popliteal fossa,  
  
  c) increasing traumatic edema of the muscles in the forearm or in calf,  
  
  d) Direct pressure by the fractured bone end.

- **inside** - Internal obstruction may follow injury to the arterial wall (stab wound or closed injury), arterial spasm, thrombosis or embolism
5. At Arrival to the hospital…. SUSPICION OF INJURY

**CAUSES**

**Penetrating(open) wounds**
- Gunshot, stab,
- IV drug abuse

**Blunt trauma (closed)**
- Joint displacement - Adjacent to major artery
- Bone fracture
- Contusion

**Invasive procedures**
- Arteriography
- Cardiac catheterization
- Balloon angioplasty
- Hernia repair
- Saphenectomy
6. HARD SIGNS OF ARTERIAL INJURY

- Immediate surgery
  - External arterial bleeding- Hemorage
  - Rapidly expanding hematoma
  - Palpable thrill, audible bruit
  - Obvious arterial occlusion – acute ischemia (pulseless, pallor, paresthesia, pain, paralysis (especially after reduction or dislocation or realignment of fracture)).

7. SOFT SIGNS OF ARTERIAL INJURY

- History of arterial bleeding at the scene
- Proximity of penetrating wound or blunt trauma to major artery
- Diminished unilateral distal pulse
- Small nonpulsatile hematoma
- Neurologic deficit
- Abnormal flow-velocity waveform on Doppler ultrasound!

Consider arteriography
8. Initial Management of Potential Vascular Injuries

Peripheral vascular injuries do not compete with those that are immediately life threatening, but take priority over most other injuries.

Once the initial resuscitation is under way, bleeding controlled, and the airway secure, the extent and nature of the vascular injuries are fully assessed.

Vessel wounds causing distal ischemia require urgent operative restoration of flow; the repair is delayed only for hemodynamic stabilization and treatment of other life-threatening problems.

Additional resuscitation or further diagnostic evaluation (such as arteriography) can be accomplished in the operating room, if indicated.

9. Emergency

The first 6 hours !!!!!!!!!!!!!

Threatened viability: indicates a state of reversible ischemia if arterial obstruction is promptly relieved.

Signs:

1. Ischemic pain or

2. Mild and Incomplete neurologic deficit is present.

3. Pulsatile flow in pedal arteries is not audible with Doppler, but venous signals are demonstrable.
10. Irreversible ischemic change:

- Profound sensory loss and
- Muscle paralysis,
- Absent capillary skin flow,
- Muscle rigor, and skin marbling are characteristic.

Neither arterial nor venous flow is audible; major amputation is required, regardless of therapy.

10. Prehospital Care:

- Stabilize the extremity in the anatomic position.
- Control hemorrhage with direct pressure.
- Apply a tourniquet proximal to the injury if direct pressure is not effective in controlling hemorrhage.

Emergency Department Care

- Immediately reduce displaced or angulated fractures if any evidence or suspicion of vascular compromise exists. Promptly reduce dislocations of the elbow and knee to prevent further injury to neurovascular structures.
External hemorrhage usually can be controlled with direct pressure, but a blood pressure cuff or tourniquets should be applied proximally to the injury if compression fails or is not possible.

Once the patient has been stabilized, identify peripheral vascular injuries and restore normal circulation as rapidly as possible.

Do not apply clamps or hemostats to vascular structures, since this may make definitive repair more difficult and damage surrounding tissues.

11. Arteriography and Duplex scan

Arteriography is the single most useful diagnostic procedure for detecting an arterial injury.

Clinical studies have supported its accuracy in the management of such trauma, but the procedure has only recently been accepted as a means of detecting arterial wound.

12. DUPLEX SCAN

Definition:

Real-time B-mode—anatomic image combined with

A pulsed-wave Doppler image (flow determination).

- Duplex scan should be performed by a competent vascular technologist or surgeon
14. FINE POINTS IN PERIPHERAL VASCULAR REPAIR

- Small vascular clamps or vessel loops
- • Pass balloon catheters into artery proximal and distal to repair
- • “Regional” heparin (50 units/mL), 10–15 mL into artery proximal and distal to repair
- • Completion arteriography
- • Fasciotomy for compartment pressure >30–35mmHg (suspect compartment syndrome if prolonged period of shock or arterial occlusion, combined arterio-venous injuries, need for arterial or venous ligation, or massive crush or swelling is present)

15. OPTIONS FOR PERIPHERAL VASCULAR REPAIR

- Lateral arteriorrhaphy or venorrhaphy
  - • Patch angioplasty
- • Resection with end-to-end anastomosis
- • Resection with interposition graft
  - • Bypass graft
- • Extraanatomic bypass
- Ligation
### ETIOLOGY

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<thead>
<tr>
<th>1. Emboli</th>
<th>4. Dissection</th>
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</thead>
<tbody>
<tr>
<td>2. Thrombosis</td>
<td>spontan</td>
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<tr>
<td>atheromatose</td>
<td>iatrogen</td>
</tr>
<tr>
<td>post surgery</td>
<td></td>
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<td>hypovolemie</td>
<td></td>
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<tr>
<td>thombocytemie</td>
<td></td>
</tr>
<tr>
<td>maligne tumor…</td>
<td></td>
</tr>
<tr>
<td>3. Spasm</td>
<td>5. Compression</td>
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<tr>
<td>ERGOTAMINE</td>
<td>DVT</td>
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<td>adrenergical infusion</td>
<td>post surgery</td>
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ARTERIAL EMBOLI

1. Cardiac (80% to 90%)
2. Aneurysma
3. Paradoxal emboli
ACUTE ISCHEMIA - SIMPTOMS

6’s P PRATT
1954

1. Pain
2. Paleness (palor)
3. Paresthesia
4. Pulselessness
5. Paralysis
6. Perishing cold
(Extreme cold)
1. **Viable**: not immediately threatened.
   1. There is no ischemic pain,
   2. No neurologic deficit,
   3. Adequate skin capillary circulation, and
   4. Clearly audible **Doppler pulsatile** flow signal in pedal arteries (ankle pressure > 30 mm Hg).

2. **Threatened viability**: indicates a state of reversible ischemia if arterial obstruction is promptly relieved.

   **Signs:**
   1. Ischemic pain or
   2. Mild and incomplete neurologic deficit is present.
   3. Pulsatile flow in pedal arteries is **not audible** with Doppler, but venous signals are demonstrable.
3. Irreversible ischemic change:

- Profound sensory loss and
- Muscle paralysis,
- Absent capillary skin flow,
- Muscle rigor, and skin marbling are characteristic.

Neither arterial nor venous flow is audible; major amputation is required, regardless of therapy.

Acute ischemia - medical treatment

1. Heparin I.V.
2. Analgetics (even morphine)
3. Quickly transport hospital
Fogarty catheter and clots from superficial femoral artery

Pharmacologic thrombolysis

The most commonly used plasminogen activators are:

- recombinant tissue-plasminogen activator (rt-PA),
  - reteplase,
  - urokinase,
- prourokinase, and
  - streptokinase
As pointed out by Haimovici in 1960, however, reperfusion of an entirely severely ischemic limb often results in a systemic inflammatory response of such magnitude that it may be lethal.
Extremity Vascular Trauma

Introduction

Patients with extremity vascular traumas present daily in emergency departments (EDs) and trauma centers worldwide. While much of the current state-of-the-art information is the result of wartime observations, the incidence of civilian extremity vascular trauma is significant. A basic understanding of both blunt and penetrating injuries to the extremities and the resultant vascular abnormalities that occur with these injuries helps minimize mortality and morbidity in these patients.

Crushed and mangled foot of a person who was involved in a motor vehicle accident.

History of the Procedure

Extremity vascular injuries have been documented during episodes of armed conflict as far back as the Greek and Roman civilizations and undoubtedly occurred before those eras. Extremity amputations were the most common procedure performed by military surgeons in the US Civil War and World War II. DeBakey and Simeone calculated the amputation rate from vascular injuries in World War II as greater than 40%.1 Amputation was primarily a means of saving the life of the soldier in an era with no antibiotics, limited surgical technology, and no critical care.

With the advance of general medical and surgical science and a concomitant improvement in military technology, the amputation rate from vascular injury in the Korean War and the Vietnam War dropped to approximately 15%. Rich and colleagues collected the vascular database information that has provided modern surgeons with an invaluable source of data that sets the standard for management of extremity vascular injury.2,3
**Problem**

Civilian extremity vascular injury, as with the wartime experience, is most prevalent in cases of penetrating trauma; however, unlike the military experience, this penetrating trauma is usually due to knife wounds or low-velocity handgun injuries.4 Fortunately, high-velocity assault weapon injuries and explosive injuries are rare in the United States.

In many parts of the world, regional conflicts in which antipersonnel mines are used has given rise to a large population of children and civilian adults with extremity vascular and soft tissue injuries resulting in amputations. Civilian trauma surgeons expecting to render aid and services in these areas can refer to references such as Husum and colleagues' War Surgery Field Manual to augment their knowledge of civilian wartime injuries.5

**Frequency**

The actual frequency of extremity vascular injuries worldwide is difficult to quantify.

In the United States, it is possible to separate iatrogenic vascular injury from traumatic injury and to reference hospital discharge data for the frequency of diagnosis codes. However, this method may significantly underestimate the actual frequency based on the method used to code the diagnosis and the importance and ranking attached to the diagnosis. In many cases, government report forms only record the top 3 discharge diagnostic codes, enough to potentially miss codes due to iatrogenic injury.

With the increased interest in the United States, more precise incidence numbers may be observed in the next few years. Mattox et al6 and Feliciano et al7 have shown an increasing number of iatrogenic vascular injuries occurring in Houston over the last few decades, an observation that is probably mirrored nationwide.

Data on blunt and penetrating injury are somewhat easier to derive. In wartime circumstances, the number of injuries may be extreme. Sherif reported 224 extremity vascular injuries in 18 months during the Afghanistan War, roughly 150
per year.8 Fasol et al reported 94 patients in 3 months (ie, approximately 376/y) on the Thailand-Cambodia border.9 In both studies, antipersonnel mines caused the majority of civilian extremity vascular injuries.

At a university teaching hospital in Australia, Tobin;10 reported 10 cases per year of extremity vascular injuries in Tbilisi, Georgia, Razmadze11 reported 10.5 cases per year; in Sweden, Kjellstrom and Risburg12 reported 8.2 cases per year; and in Oxford, United Kingdom, Magee et al13 reported 4.7 cases per year. Penetrating injuries, both violent and nonviolent, predominated as the causes of vascular injuries in these reviews.

In the United States, the situation is similar, although numbers are generally higher. Humphrey et al14 reported 12.4 extremity vascular injuries per year at a rural trauma center in Missouri; Feliciano et al7 reported approximately 55 lower extremity vascular injuries per year at Ben Taub General Hospital (a high-volume urban trauma center) in Houston, TX. In both extremes, the predominant cause of injury, especially in isolated vascular injury, was due to penetrating causes. Mattox et al6 and Feliciano et al7 have also pointed out that the number of iatrogenic vascular injuries has significantly increased since 1958 as more and varied physician specialties access the vascular tree. (See Caps' excellent article on the epidemiology of vascular injuries in Seminars in Vascular Surgery15 and Mattox and colleagues' paper on epidemiological evolution of these injuries in Annals of Surgery [1989]).6

**Etiology**

Extremity vascular injury may result from penetrating injury (eg, gunshot wounds,16 knife injuries), but not all penetrating injuries are violent in nature. Many penetrating extremity injuries reported in the literature are from industrial accidents (eg, nail guns) or are iatrogenic complications of vascular access procedures for other medical problems.

Blunt injuries causing vascular injury typically result from motor vehicle accidents but may include falls, assaults, and crush injuries. Fractured long bones or dislocated joints frequently increase the overall risk of vascular injury, but certain
injuries (eg, posterior knee dislocation) are more likely to cause vascular injury than other injuries (eg, a Colles fracture of the wrist, which rarely results in radial or ulnar artery injury). The worldwide increase in explosive-type injuries constitutes an emerging third modality that combines the pathology of both blunt and penetrating injury to the extremities. Terrorist bombings, civilian land mine injuries, and combat-related injuries are becoming more common, and all physicians will undoubtedly encounter these patients sometime in their career.

Pathophysiology

As noted by the preponderance of penetrating injury in the published medical literature, the vascular tree, both arterial and venous, appears to have some limited natural protection from stretching and bending, which results in fewer blunt injuries to the extremity vasculature following trauma. The smooth muscle of the arterial media protects the patient from both stretch-type injuries and minor puncture wounds, which heal spontaneously in most cases. The smooth muscle layer also offers mild protection from death due to ongoing hemorrhage.

When the arterial vessel is transected, vascular spasm coupled with low systemic blood pressure appears to promote clotting at the site of injury and to preserve vital organ perfusion better than that which occurs with ongoing uncontrolled hemorrhage. This partially explains the prehospital finding that, in the subset of penetrating trauma, limited or no fluid resuscitation until arrival at the hospital may improve patient survival and outcome.

Presentation

Worldwide, patients with extremity vascular injuries most frequently present after a penetrating injury to an extremity. In the United States, high-speed motor vehicle accidents, often with fractures or dislocations, result in the next largest group of patients. In patients with large lacerations or open wounds, persisting or increasing hemorrhage with resuscitation is an early indication of vascular injury requiring operative exploration.

Vascular injuries can be classified clinically into hard signs and soft signs of injury based on examination.
Classic so-called **hard signs** of vascular injury include the following:

- Observed pulsatile bleeding
- Arterial thrill (ie, vibration) by manual palpation
- Bruit over or near the artery by auscultation
- Signs of distal ischemia
- Visible expanding hematoma

These signs are used to identify patients requiring surgical intervention. A finding of cool, cold, and pulseless extremities may be attributable to a low systemic blood pressure, but isolated pulse abnormalities and significant variation in pulse quality from side to side are strong indicators of underlying proximal vascular injury. Neurologic deficit, delayed capillary refill, and bony abnormalities should increase the suspicion of extremity vascular injury and the need for emergent arteriography or surgical exploration and repair.

**Soft signs** of vascular injury include the following:

- Significant hemorrhage found on history
- Decreased pulse compared to the contralateral extremity
- Bony injury or proximity penetrating wound
- Neurologic abnormality

Clinical examination and reexamination remain the mainstays for identifying and treating these wounds. Clinical examination and findings should determine the need for adjunctive studies such as noninvasive Doppler ultrasound and arteriography. The physical examination may be augmented by measurement of the ankle-brachial index (ABI), also referred to as the arterial pressure index in the literature. Measurement of the ABI is a standard component in the evaluation of atherosclerotic peripheral vascular disease, and its value extends to the identification of penetrating injuries to extremity vessels. Both hard and soft signs
help direct the physician to the best diagnostic and treatment options for an individual patient.

**Indications**

In general, hard signs (eg, change in pulse quality compared to the opposite extremity, loss of pulse in the extremity) are absolute indications for further diagnostic studies (eg, arteriogram, exploration and direct visualization in the operating room). Softer signs (eg, temperature change, color change, delayed capillary refill, neurologic deficit) should alert the clinician to the need for close observation and monitoring. If the ABI is higher than 0.9, many authors advocate observation, but if the ABI is lower than 0.9, further evaluation is warranted. In these cases, many authors now recommend duplex Doppler vascular studies as a rapid, noninvasive method of assessing vascular injury. However, an arteriogram in stable patients and operative exploration in unstable or bleeding patients remain the criterion standards of care.

**Relevant Anatomy** A thorough knowledge of basic medical school anatomy of the extremities is essential in the evaluation and management of extremity vascular injuries. While it is often possible to directly visualize an arterial injury through an open wound, obtaining proximal and distal control for vascular reconstruction requires intimate knowledge of vascular, muscular, and bony anatomy to allow rapid access to the arterial tree proximally and distally, while minimizing incision length and surgical tissue dissection.

Frequently, especially in cases of blunt trauma and arterial trauma with ongoing hemorrhage, the normal tissue planes are destroyed and the smooth muscle in both the artery and the vein cause retraction of the vessels into the depths of the wound. Operative identification of arterial and venous injury as a prelude to repair often requires proximal and distal control of the artery or vein, which may require extending the wound in both directions or making counterincisions.

Temporary vascular control can be achieved by simply applying pressure to the vessel proximal to the injury (eg, femoral pressure in a lower extremity wound). The use of tourniquets, while helpful in the operating room, should be limited to
patients at risk for exsanguination in the prehospital and field environments who are not responsive to direct pressure for hemorrhage control. The use of tourniquets, especially those left for prolonged periods, markedly increases the incidence of amputation of an injured extremity. Any medical personnel applying a prehospital tourniquet for extremity vascular injury should clearly document its necessity as a lifesaving anti-exsanguination device when direct pressure fails and should understand that, in most cases, a tourniquet saves a life but results in loss of an extremity.

**Contraindications**

Preexisting renal insufficiency and allergies (seafood, iodine, contrast dye) are relative contraindications for arteriography in the assessment of vascular injury of an extremity. Pre-angiography volume resuscitation and sodium bicarbonate may help minimize the complications noted above.

Persistent massive hemorrhage and hemodynamic instability are principal contraindications for any diagnostic studies, and patients with these conditions require urgent operative exploration for diagnostic and therapeutic measures. Duplex Doppler studies may provide important information regarding vascular injury in most stable patients who have contraindications to arteriography.

**Laboratory Studies**

Baseline blood work should consist of a CBC count with platelet count, electrolytes, BUN, and creatinine evaluations.

Typing and crossmatching of packed red blood cells for 4-8 U, depending on the severity of injury and hemorrhage, is also recommended.

Prothrombin time and activated partial thromboplastin time may be helpful in patients who are comatose and unable to provide an adequate medical history, although statistically, findings are rarely abnormal when the medical history documents no medications (eg, warfarin) or a history of bleeding problems.

In acute hemorrhage without equilibration, remember that the hematocrit or hemoglobin level may appear to be within the laboratory reference range even though there may be a significant cellular volume loss.
Imaging Studies

Plain x-ray films of the injured extremity are a rapid means of determining the presence of fractured bones and foreign bodies. Certain fractures (eg, supracondylar femur fractures) have a higher incidence of vascular injuries, and recognition of these types of injuries alerts the clinician to the risk of vascular injury.

CT scanning has been used in extremity trauma to visualize bony anatomy and soft tissues but still is not proven as a diagnostic modality in peripheral vascular injury. As such, CT scanning should not be used except in unusual circumstances.

Arteriography in the angiography suite is reserved for patients who are hemodynamically stable and preferably without renal failure or insufficiency. Most interventional radiologists require preprocedural BUN and creatinine measurements before proceeding with these studies. As soon as practicable, blood for these assays should be drawn in the resuscitation area to avoid delays in angiography, which may lead to delays in operative intervention.

In many cases, the surgeon can perform on-table angiography in the operating room with minimal risk to the patient. Surgeons should be familiar with arterial access points and the contrast materials available in their institution. Knowledge of total dye load and baseline renal status minimizes complications in this situation.

Duplex Doppler ultrasound studies of injured extremities have been shown to be a viable alternative to angiography in many centers. This study can be performed by the surgeon in the ED or in the resuscitation bay and can provide immediate and valuable information regarding patient vascular status or injury. Duplex Doppler ultrasound may be of limited use in patients with splints, extensive orthopedic hardware, areas of large tissue and skin loss, and when used by inexperienced personnel. Johansen et al offer a more detailed discussion of noninvasive tests in a screening situation.17
Other Tests

Ankle-brachial index

Measurement of the ABI is useful with atherosclerotic peripheral vascular disease and may be helpful in determining vascular insufficiency, but ABI cannot localize the site of injury.

Measurement of the ABI is a helpful component of the evaluation of penetrating arterial injury; however, the ABI cannot localize the site of injury.

A prospective study by Lynch and Johansen18 suggests that measurement of the ABI approaches the accuracy of arteriogram in identifying arterial injuries, and, more importantly, accurately identifies injuries needing intervention. Nassoura and colleagues supported this finding in a subsequent prospective trial.19

No diagnostic test is perfect; nevertheless, measurement of the ABI offers a noninvasive, simple, and reproducible method to accurately screen for penetrating arterial injury.

Assessing for a Doppler signal in peripheral vessels is more sensitive than manual palpation and is helpful in assessing for total occlusion or transection of the arterial tree.

Staging

Organ injury scaling may be helpful in the acute setting but should not override clinical experience and individual patient needs. Vascular injury scaling is also helpful for epidemiological study, peer review, and coding and billing. For information regarding organ injury scaling of peripheral vascular injuries currently sanctioned by the American Association for the Surgery of Trauma, see the study by Moore et al.20

The Mangled Extremity Severity Score (MESS) is an objective criterion for amputation prediction after lower or upper extremity injury. A MESS of >7 has
been used as a cutoff point for amputation prediction. Prichayudh et al examined the result of upper extremity vascular injury management and amputation rate as related to MESS in 52 patients. Seven of 52 patients underwent amputation (overall amputation rate, 13.46%). Multivariate analysis revealed that the only factor significantly associated with amputation was the MESS. No amputations were performed in 33 patients who had a MESS of <7. Secondary amputations (amputation after primary operation) were done in 4 of 49 patients (8.16%). All amputation patients suffered blunt injuries and had a MESS of >7 (range, 7-11). Amputation was avoided in 12 of 19 patients who had a MESS >7.21

A MESS of >7 does not always indicate that amputation is required; however, MESS is a better predictor for patients who do not require amputation when the score is <7. The decision regarding whether or not to amputate should be made individually based on clinical signs and intraoperative findings of irreversible limb ischemia.

**Medical Therapy**

Medical therapy alone is rarely an option in penetrating or blunt trauma to the extremity vasculature with hard signs. Asymptomatic patients or patients with only soft signs can often be observed, but this is best performed by a surgeon who is prepared to operate if the clinical examination changes. The observation must be performed with the understanding that if the examination findings change or if hard signs develop, surgical intervention is necessary.

While pharmacologic anticoagulation is a viable therapy for arterial thrombosis in some situations, acute injury of the arteriovenous tree usually requires surgical intervention and mechanical repair. Limited anticoagulation or antiplatelet drugs may be helpful after vascular repair, especially with prosthetic material, but carefully weigh the benefit of these drugs against the potential for hemorrhage in other injured tissue, especially with concurrent brain or spinal injury.
Surgical Therapy

Surgical intervention when suspecting peripheral vascular injuries can be as minor as operative visualization of normal vascular anatomy for diagnostic purposes or as extensive as reconstruction and replacement of entire segments of injured vessels.22 Timing of surgical intervention can be critical to outcome in extremity vascular injury. Vascular reconstruction that occurs within 3 hours of injury is generally accepted to have the best outcome. While this can frequently be accomplished in urban Level 1 Trauma Centers, it becomes more difficult in rural areas where availability of rapid EMS transport, geographic location of the hospital, and availability of interventional radiology and surgical subspecialists may be limited.

In most cases in which the injured segment is 1 cm or less, dissecting and freeing edges and performing a primary anastomosis is frequently possible. Take care to avoid traction on perforating branches or excessive dissection, which may devascularize surrounding tissue. Attention to vascular surgical technique should minimize tension on the vessel and stricture at the anastomotic site.

In more severe cases with multiple associated injuries, hemorrhage control by ligation of actively bleeding arterial or venous vessels may be all that is possible. Tissue viability distal to an arterial ligation depends on regional arterial anatomy, collateral blood flow, preexisting atherosclerotic disease, competent venous outflow, and volume status.

Although venous ligation is counter-intuitive, it may carry a higher risk than arterial ligation. Certain vessels, such as the popliteal vein, carry a high postligation amputation rate, while the rate for femoral or external iliac vein ligation is statistically lower. The risk of subsequent amputation after any ligation is much higher than after vascular repair, but patients with severe brain injury or hemodynamic instability may not tolerate a 2- to 3-hour operation to repair a vascular injury, and damage-control techniques with arterial or venous ligation may save lives. Use of intravenous chemical vasoconstrictors (phenylephrine [Neo-Synephrine, norepinephrine) should be minimized in the postoperative period.
If the patient's condition and hemodynamic status allow prolonged operative intervention, general replacement of an injured peripheral arterial segment is accomplished with an autologous vein. The saphenous or cephalic veins harvested from the same or contralateral extremity are the most commonly used vein segments. Polytetrafluoroethylene (PTFE) can be used in some situations but is usually reserved for above-knee or above-elbow applications. PTFE has been successfully used in contaminated fields with a low infection rate23 for both venous and arterial reconstruction. In some trauma centers, PTFE is the preferred conduit and has replaced the use of an autologous vein in above-knee, below-knee, and elbow reconstruction. Stevens et al24 summarize the causes of failure of arterial reconstruction.

Typically, in most acute situations, venous injuries are primarily ligated, but, in a select number of injuries in hemodynamically stable patients, venous reconstruction may be an option. Very little prospective data is available in the trauma literature, but readers are directed to an older but more pertinent retrospective review in the Journal of Vascular Surgery for more information.25

After reconstruction in the stable patient or vascular ligation in damage-control situations has been completed, the surgeon should consider the risk of reperfusion injury and the potential for compartment syndrome.26 While this is more common in distal lower extremities, it is also possible in proximal compartments and the upper extremities (see Image). **Fasciotomies** increase the risk of infection, increase fluid and blood loss, and eventually require reoperation for either skin closure or skin grafting.27 These complications should be weighed against the risk of compartment syndrome with risk of limb loss, renal failure from myoglobin release, and tissue gangrene. Monitoring compartment pressures in the postoperative period in conjunction with the clinical examination is possible, but prophylactic fasciotomies, even with the attendant risks noted above, are to be recommended in the more severe cases.
Crushed and mangled foot of a person who was involved in a motor vehicle accident.

The most challenging injuries are those of the mangled extremity, with concurrent bony, soft tissue, nerve, and vascular injury. The treatment of these complex injuries precludes detailed description in a short review, but many authors have evaluated the factors that determine the risk of amputation.

Scoring systems have been developed as a means to predict amputation and functional outcome. Scoring systems such as Mangled Extremity Syndrome Index (MESI), Mangled Extremity Severity Score (MESS), Predictive Salvage Index (PSI), and Limb Salvage Index (LSI) have been reviewed by Durham et al. Prediction of amputation was sensitive and specific, but prediction of functional outcome was universally poor.

The MESS score appears to be the most commonly used method and is based on criteria that include (1) degree of skeletal/soft tissue injury, (2) limb ischemia, (3) shock, and (4) patient age.
Note that some authors have been unable to validate individual scoring systems, and no one system is universally accepted.31

Interventional radiologic techniques should also be noted as an option in acute injury, but the indications and timing are still being developed. Coil embolization of complications of vascular trauma, such as arteriovenous malformations and pseudoaneurysms, are more commonplace and are described in more detail in Follow-up care. Endovascular stenting has been reported for acute traumatic injuries since 1994, but it is not yet available in most facilities.32,33 Long-term outcome and complication rates have not been calculated, and although the technique is promising, more long-term follow-up study is necessary.

Preoperative Details

If planning reconstruction, the best results have been reported in patients who are hemodynamically stable with normal laboratory findings and preoperative arteriography to localize the injury.

In some cases, operative intervention is primarily performed for life-saving hemorrhage control rather than for operative repair with limb salvage.

Intraoperative Details

Initial ligation of life-threatening vascular hemorrhage may allow stabilization of patients and subsequent exploration and repair of the injured vessels. In the patient who remains hemodynamically unstable, the surgeon should balance the desire to save the limb with that of preserving the patient's life.

Postoperative Details

Frequent monitoring and vascular checks (eg, pulse presence, quality, capillary refill) should continue for the first 24-48 hours. Consideration of anticoagulation and antiplatelet agents should be balanced with the risk of fatal hemorrhage from other injuries (eg, head and chest injuries).
Maintain adequate hydration, especially after administration of contrast dye, after episodes of hypotension, and in the presence of concomitant renal injury. A urine output of 20 mL/h or more is ideal in adult patients.

**Follow-up.**

Vascular repair with palpable pulses in the postoperative period rarely requires repeat angiography. If a completion angiogram was not performed in the operating room, duplex Doppler ultrasound may provide a less invasive method of monitoring graft status.

Advise patients of the risks and symptoms of thrombosis or vascular occlusion so that they may quickly contact the surgeon or obtain evaluation in a local ED if problems occur. The surgeon should consider the need for anticoagulation or antiplatelet medications (eg, coumadin, aspirin), balancing the overall risk to patients with the needs of the graft and vascular repair.

**Complications**

Thrombosis of the graft remains the most common complication of vascular injury and blood vessel repair. Narrowing of the vessel with primary repair or kinking of the graft, especially after repetitive orthopedic intervention may compromise volume of flow and may require revision of the repair. Ligation of vessels for emergent hemorrhage control may result in ischemia, leading to amputation more frequently than vascular repair.

One of the more difficult situations for patients and surgeons occurs when permanent nerve injury ensues but is diagnosed late because of concurrent head or other injury. Functional vasculature with significant irreparable denervation of motor and sensory components of the extremity usually results in a useless appendage, which causes more problems or complications than amputation. Splinting or bracing the extremity occasionally provides an acceptable functional result and should be considered, but many patients opt for amputation and a functional prosthesis rather than a nonfunctional insensate extremity that requires constant care and monitoring.
Outcome and Prognosis

In 1986, Floyd and Kerstein documented 10 patients with successful vascular reconstructions; however, in every case, the patients' outcome included a permanent disability that was moderately severe to severe. In most cases, the disability was due to concurrent partial or complete nerve injury. In addition, while no early amputations were necessary, there was a 40% amputation rate.

In 1994, Humphrey et al noted a reduction in the amputation rate from 18% to 7%, with a stable 4.8% patient mortality rate with institution of a helicopter transport system in rural Missouri.

In 1996, Magee et al reported a 6% amputation rate and a 19% complication rate at 6-month follow-up in the United Kingdom. However, no information was noted regarding disability.

In 1999, Razmadze reported a 16% early and late amputation rate, with a 7.6% patient mortality rate in the former Soviet republic of Georgia.

These data clearly show that extremity vascular injury, especially those with concomitant nerve, bone, and significant soft tissue injury, can be disastrous to patients. Early and aggressive vascular repair improves patient outcome but cannot reverse the effects of some injuries. Amputation and disability rates remain high, even with optimal transport, trauma care, and successful operative intervention.

Future and Controversies

Improved Emergency Medical Services (EMS) systems, faster transport times, availability of interventional radiological techniques, improved surgical technique, and new vascular conduits may further reduce the morbidity and mortality of extremity vascular injury. The future of limiting the morbidity and mortality of these injuries probably lies with advancements in other areas, eg, motor vehicle safety, worldwide control and cleanup of antipersonnel mines, and injury prevention programs.
The atheromatous core usually occupies the deeper central regions of the plaque and contains amorphous as well as crystalline and droplet forms of lipid. Cells with morphologic and functional characteristics of smooth muscle cells or macrophages are noted about the necrotic core and at the edges or shoulders of the plaques.

- Both cell types may contain lipid vacuoles.
- In addition, calcium salts and myxoid deposits as well as matrix fibers, including collagen, elastin, fine fibrillar material, structures resembling basal lamina, and amorphous ground substance are evident.
Arterial occlusive disease

Caracterisation
1. First stenosis second occlusion
2. “Multilevel disease”
3. Colateral circulation development

Collateral circulation
Fontaine Classification

- **Stage I**: well compensated – asymptomatic
- **Stage II**: Insufficiency during exercise (intermittent claudication)
  - II-A – more than 250 m
  - II B – less than 250 m
- **Stage III**: rest pain
- **Stage IV**: Anoxic tissue damage (necrosis, ulcer).
Critical limb ischemia (CLI):
- Patient experience pain for 2 weeks (with medical treatment)
- Superficial lesions of gangrene

Systolic Index < 50 mm Hg
- in nondiabetic
Systolic Index < 30 mm Hg
- in diabetic people
Claudication is a relative benign syndrome

At 5 years

- At least another 300 people with asymptomatic PAOD
- 100 patients presenting to doctor with claudication
- Another 100 patients with claudication will not present to a doctor

Local outcome in leg
- 75 stabilise or improve claudication
- 25 will deteriorate

Systemic outcome
- 5 to 10 non-fatal CV events in 5 years
- 30 will die within 5 years
- 55 to 60 will be alive without new CV event in 5 years

Medical and invasive/surgical treatment

Treatment

- **Stage I si II -A**
  - medical treatment
- **Stage II -B, III si IV**
  - Medical and invasive/surgical treatment
Aortoiliac Occlusive Disease

Introduction

In patients with peripheral arterial disease, obstructing plaques caused by atherosclerotic occlusive disease commonly occur in the infrarenal aorta and iliac arteries. Atherosclerotic plaques may induce symptoms either by obstructing blood flow or by breaking apart and embolizing atherosclerotic and/or thrombotic debris to more distal blood vessels. If the plaques are large enough to impinge on the arterial lumen, reduction of blood flow to the extremities occurs. Several risk factors exist for development of the arterial lesions, and recognition of these factors enables physicians to prescribe nonoperative treatment that may alleviate symptoms as well as prolong life.

Surgical treatment of aortoiliac occlusive disease (AIOD) has been well standardized for many years, and the outcomes are quite good. However, the additional techniques of percutaneous transluminal angioplasty (PTA) and stenting have offered more alternatives to open surgery and offer successful techniques to patients who may have been considered at an unacceptably high risk for conventional open surgical repairs. Catheter-based endovascular treatments for aortoiliac occlusive disease (AIOD) offer the advantages of less morbidity, faster recovery, and shorter hospital stays. In fact, most endovascular interventions today are simply performed as outpatient procedures. This chapter reviews the risk factors for development of atherosclerotic occlusive disease of the aorta and iliac arteries and describes the natural history, diagnosis, and treatment of the disease.

History of the Procedure

Before prosthetic grafts for aortic bypasses became available, the first direct surgical reconstructions on the aorta were performed using the technique of
thromboendarterectomy (TEA), first described by Dos Santos of Lisbon in 1947.1 The initial procedure was performed on a patient with superficial femoral artery (SFA) obstruction, and Dos Santos termed the procedure disobliteration. Edwin J. Wylie, MD, adapted this technique to the aortoiliac region and, in 1951, performed the first aortoiliac endarterectomy in the United States.2 With the discovery of suitable prosthetic graft materials for aortic replacement in the 1960s, surgical treatment of aortoiliac occlusive disease (AIOD) became available to even more patients.

In 1964, Dotter first performed percutaneous iliac angioplasty using a coaxial system of metal dilators.3 This procedure proved to have limited application due to the cumbersome nature of the device. However, Dotter's early work paved the way for Grüntzig, who, in 1974, developed a catheter with an inflatable polyvinyl chloride balloon that could be passed over a guidewire.4 This device became the cornerstone for the percutaneous treatment of arterial occlusive lesions today. In 1985, Julio Palmaz introduced the first stent that helped to improve the results of angioplasty for arterial occlusive disease.5 Since the advent of angioplasty and stenting, the technology has evolved at an astronomical rate. The design and quality of endovascular devices, as well as the ease and accuracy of performing the procedures, have improved. These improvements have led to improved patient outcomes following endovascular interventions for aortoiliac occlusive disease (AIOD).

**Problem** Aortoiliac occlusive disease (AIOD) occurs commonly in patients with peripheral arterial disease (PAD). Significant lesions in the aortoiliac arterial segment are exposed easily by palpation of the femoral pulses. Any diminution of the palpable femoral pulse indicates that a more proximal obstruction exists. Obstructive lesions may be present in the infrarenal aorta, common iliac, internal iliac (hypogastric), external iliac, or combinations of any or all of these vessels. Occasionally, degenerated nonstenotic atheromatous disease exists in these vessels and may manifest by atheroembolism to the foot, the "blue toe" or "trash foot" syndrome.
Frequency

At least half of patients with peripheral arterial disease (PAD) have no symptoms, and, therefore, the exact incidence and prevalence of the condition is unknown. However, the incidence of PAD is known to increase with advancing age so that, by age 70 years, as many as 25% of the US population is affected. Occlusive disease involving the aortoiliac arterial segment occurs commonly in patients with peripheral arterial disease (PAD) and is second only to occlusive disease of the SFA in frequency.

Etiology

Atherosclerosis is the most common etiology of occlusive plaques in the aorta and iliac arteries. Several risk factors exist for the development of atherosclerotic plaques in the aortoiliac arterial segment. **Cigarette smoking** and **hypercholesterolemia** are observed more commonly in patients with aortoiliac occlusive disease (AIOD) as compared with infrainguinal occlusive disease. In addition, patients with aortoiliac occlusive disease (AIOD) tend to be younger and less likely to have diabetes.

An uncommon cause of aortic obstruction is Takayasu disease, a nonspecific arteritis that may cause obstruction of the abdominal aorta and its branches. The etiology of Takayasu disease is not known. For the purpose of this chapter, only occlusive lesions caused by atherosclerosis are considered.

Pathophysiology

Atherosclerosis is an extraordinarily complex degenerative disease with no known single cause. However, many variables are known to contribute to the development of atherosclerotic lesions. One popular theory emphasizes that atherosclerosis occurs as a response to arterial injury. Factors that are known to be injurious to the arterial wall include mechanical factors such as hypertension and low wall shear stress, as well as chemical factors such as nicotine, **hyperlipidemia**, **hyperglycemia**, and **homocysteine**.
Lipid accumulation begins in the smooth muscle cells and macrophages that occur as an inflammatory response to endothelial injury, and the "fatty streak" begins to form in the arterial wall. The atheroma consists of differing compositions of cholesterol, cholesterol esters, and triglycerides. Some plaques are unstable, and fissures occur on the surface of the plaque that expose the circulating platelets to the inner elements of the atheroma. Platelet aggregation then is stimulated. Platelets bind to fibrin through activation of the glycoprotein (Gp) IIb/IIIa receptor on the platelets, and a fresh blood clot forms in the area of plaque breakdown. These unstable plaques are prone to atheromatous embolization and/or propagation of clot that eventually can occlude the arterial lumen.

If the atheroma enlarges enough to occupy at least 50% of the arterial lumen, the flow velocity of blood through that stenosis can significantly increase. The oxygen requirements of the lower extremity at rest are low enough that even with a moderate proximal stenosis, no increase in blood flow velocity occurs. During exercise, however, the oxygen debt that occurs in ischemic muscle cannot be relieved because of the proximal obstruction of blood flow; this results in claudication symptoms. In more advanced cases, critical tissue ischemia occurs, and neuropathic rest pain or tissue loss ensues. However, critical limb ischemia is seldom, if ever, caused by aortoiliac occlusive disease (AIOD) alone. Commonly, in patients with critical limb ischemia, multiple arterial segments are involved in the occlusive atherosclerotic process.

**Presentation**

The most common symptom of patients with hemodynamically significant aortoiliac disease is claudication. The word claudication stems from the Latin word claudicatio, to limp. The symptom complex of claudication is defined as muscle cramps in the leg(s) that occur following exercise and are relieved by resting. In any individual patient, the exercise distance at which claudication occurs is quite constant. Claudication usually occurs first in the calf muscles, although thigh, hip, and buttocks muscles also can be affected when more extensive proximal lesions are present. Location of the muscle pain (ie, calf vs thigh) does not necessarily correlate with the level of arterial obstruction. However, more proximal symptoms (ie, buttocks or thigh claudication) are generally associated with severe aortoiliac occlusive disease.
Symptoms of buttock claudication can occur in association with erectile dysfunction in patients with absent femoral pulses. This constellation of symptoms is termed Leriche syndrome, named for the surgeon who described the condition in 1923. Leriche syndrome occurs when either preocclusive stenosis or complete occlusion of the infrarenal aorta is present due to severe aortic atherosclerosis. Due to the chronic nature of the occlusive process leading to development of rich collateral vessels that supply the lower extremity, limb-threatening ischemia seldom occurs.

Indications

Treatment of patients with peripheral arterial disease (PAD) has 2 goals. The first and foremost goal is to reduce the risk of vascular events (myocardial infarction, stroke, vascular death) that occur at an alarmingly high rate in patients with PAD. About 30% of patients with peripheral arterial disease (PAD) die within 5 years, and death is usually due to an ischemic coronary event. The second goal of treatment is to improve symptoms in those patients with claudication and prevent amputation in patients with critical limb ischemia. Critical limb ischemia is present when patients have symptoms of ischemic rest pain, nonhealing foot ulcers, or gangrene, and its presence mandates urgent evaluation with aortography and endovascular and/or surgical revascularization to prevent limb loss.

At least half of patients with peripheral arterial disease (PAD) are asymptomatic and are diagnosed only by physical examination and/or measurement of the ankle/brachial index (ABI). An ABI less than 0.9 clearly is abnormal and confirms the diagnosis of peripheral arterial disease (PAD). An abnormal ABI should alert the clinician to the fact that this group of patients is at risk for early mortality from cardiovascular causes, ie, myocardial infarction, stroke, other vascular death. The goal for treatment of asymptomatic patients is to reduce the risk of subsequent vascular events.

Relevant Anatomy

Three distinct arterial segments distal to the visceral bearing portion of the abdominal aorta may become diseased by atherosclerosis. Type I atherosclerosis
involves the infrarenal aorta and common iliac arteries only. This pattern of atherosclerosis is present in about 5-10\% of patients with peripheral arterial disease (PAD) and occurs more commonly in women. The vessels distal to the common iliac arteries usually are generally normal or only minimally diseased. Type II atherosclerosis involves the infrarenal aorta, common and external iliac arteries, and may extend into the common femoral arteries. This pattern is observed in 35\% of patients with peripheral arterial disease (PAD). Type III atherosclerosis is the most severe form and, unfortunately, also the most common. This pattern of atherosclerosis involves the infrarenal aorta, iliac, femoral, popliteal, and tibial arteries.

Diabetes mellitus is a risk factor that results in a characteristic pattern of atherosclerotic lesions in patients with peripheral arterial disease (PAD). The proximal inflow (aorta, iliac) arteries tend be normal. However, the femoropopliteal segment (including the profunda femoris artery), and especially the proximal tibial arteries, are usually severely diseased. Fortunately, the distal tibial and plantar vessels may be normal, enabling successful arterial reconstruction for limb-threatening ischemia.

**Contraindications** At least 50\% of patients with peripheral arterial disease (PAD) may be asymptomatic. Because natural history data are poor for iliac stenosis, surgical and/or endovascular intervention should not be considered if patients truly are asymptomatic. Surgical intervention for limb-threatening ischemia is accepted universally, unless the limb is deemed nonviable. Determining whether or not to intervene in a patient with mild claudication may not be as straightforward.

An important role exists for conservative therapy in patients with aortoiliac occlusive disease (AIOD). Although surgical therapy usually alleviates symptoms, the patient must be apprised of the operative risk of mortality (2-3\%) as well as anticipated outcomes over time. Since the advent of catheter-based treatments for aortoiliac occlusive disease (AIOD), asymptomatic patients are often treated prophylactically with either angioplasty or stenting of iliac arterial lesions that are discovered during coronary angiography. This practice of drive-by angioplasty should not be recommended.
Laboratory Studies

Examine a serum lipid profile that includes total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides (TG). Furthermore, in younger patients or those with a strong family history of atherosclerosis at any early age, lipoprotein (a) and homocysteine levels should be determined.

If a history of diabetes exists, a glycosylated hemoglobin level (Hgb A1c) should be checked. Excellent control of diabetes reduces long-term complications, and the American Diabetic Association (ADA) currently recommends that the Hgb A1c be less than 7%.

If a patient has a history of thrombosis in any venous or arterial segment or a family history of a clotting disorder, an evaluation for hypercoagulability is necessary. Tests include routine prothrombin time, partial thromboplastin time, platelet count, factor V-Leiden, factor II (prothrombin) C-20210a, anticardiolipin antibody, baseline protein C, protein S, and antithrombin III levels.

Imaging Studies

Contrast aortography is not always required, unless interventional therapy (percutaneous transluminal angioplasty /stent or surgical revascularization) is planned. Serum creatinine is checked to validate a baseline level prior to the use of contrast agents that may be nephrotoxic.

Computerized tomographic arteriography (CTA) is an excellent modality for planning operative or endovascular treatments. CTA has the advantage of producing 3-dimensional images of the arterial system that are as accurate as those of conventional catheter arteriography. However, iodinated contrast agent is still required to obtain the images in CTA, although direct arterial cannulation is not needed.

As an alternative to conventional angiography, the surgeon may consider magnetic resonance angiography (MRA) or arterial duplex mapping as definitive imaging studies for planning surgery. MRA is overly sensitive and may show significant arterial stenoses that are simply not present.
Other Tests

The Doppler-derived ABI is a simple office-based examination that confirms the diagnosis of peripheral arterial disease (PAD) if the value is less than 0.9. The ABI also can grade the severity of peripheral arterial disease (PAD). Note that Doppler-derived segmental arterial pressures do not reflect the severity of AIOD accurately.

In addition, the ABI is not very sensitive in identifying patients with mild occlusive lesions in the aortoiliac segment. A treadmill exercise stress test should be recommended for those patients with mild iliac occlusive disease who have symptoms suggestive of claudication even though the ABI is normal at rest. Following exercise, the blood flow through stenotic vessels increases and the pressure decline across these lesions is augmented.

Moreover, if the blood pressure cuff is unable to compress the vessels adequately, the Doppler-derived pressures may be falsely elevated. This may occur in patients with diabetes or end-stage renal disease. In the event that supranormal (falsely elevated) Doppler-derived pressures are encountered, pulse volume recordings (PVR) may be useful in evaluating leg perfusion. The PVR waveform reflects the volume of blood in the leg during an individual cardiac cycle. A normal waveform demonstrates a brisk upstroke, a sharp systolic peak, and a downstroke with a dicrotic notch. With significant peripheral arterial disease (PAD), the dicrotic notch is lost, the slope of the upstroke and downstroke decline, the amplitude of the waveform is reduced, and the contour of the systolic peak is more rounded.

Because an association of coronary disease in patients with peripheral arterial disease (PAD) exists, obtain an electrocardiogram even in patients without cardiac history.

For those patients being considered for an intra-abdominal aortic procedure, pulmonary function tests are important if a history of obstructive pulmonary disease or dyspnea is present. Many times the surgical approach needs to be altered based on the results of this preoperative evaluation.

If a patient has a history of thrombosis in any venous or arterial segment or a family history of a clotting disorder, an evaluation for hypercoagulability is necessary. Tests include routine prothrombin time, activated partial thromboplastin
time, platelet count, factor V-Leiden, factor II (prothrombin) C-20210a, anticardiolipin antibody, protein C and protein S levels, and antithrombin III.

An intensive preoperative cardiac evaluation is reserved for patients with newly onset angina pectoris, unstable angina pectoris, or evidence of ventricular dysfunction based on dobutamine stress echocardiogram. Adenosine thallium perfusion tests are not routinely performed because of the high sensitivity and low specificity.

**Treatment**

**Medical Therapy**

The 2 goals for the clinician treating aortoiliac occlusive disease (AIOD) are (1) improving symptoms and (2) reducing the associated risk of myocardial infarction, stroke, and vascular death. Three fundamental principles are involved in the treatment of symptomatic peripheral arterial disease (PAD) due to aortoiliac occlusive disease (AIOD).

First, the risk factors must be identified and aggressively treated. The 2 most important risk factors for peripheral arterial disease (PAD) are cigarette smoking and diabetes. Complete cessation of smoking is mandatory. Carefully regulate serum glucose. The goal for adequate glucose control is an Hgb A1c level lower than 7%. The goal of hypertension control should be blood pressures lower than 140/90 mm Hg. Finally, the LDL cholesterol level should be reduced to less than 100 mg%. This usually can be accomplished with the use of hepatic 3-methylglutaryl coenzyme A (HMG CoA) reductase inhibitors (statins). In addition to modification of risk factors, patients with aortoiliac occlusive disease (AIOD) should receive lifelong antiplatelet therapy to reduce the risk of myocardial infarction, stroke, and vascular death.

Secondly, initiate a walking exercise program. No fewer than 28 prospective randomized clinical trials attest to the efficacy of walking exercise to treat
Every trial has demonstrated improvements in walking distance from 180-340%. Supervised walking programs usually produce better results than unsupervised exercise. Walking exercise even has been compared to angioplasty and found to produce superior results. Walking exercise improves symptoms of claudication because the muscle enzymes involved in oxygen extraction and utilization become more efficient over time.

Finally, 2 Food and Drug Administration (FDA)–approved pharmacologic agents may improve the symptoms of claudication caused by lower extremity arterial occlusive disease. Pentoxifylline is a methyl xanthine derivative that acts as a hemorheologic agent, lowering blood viscosity. Unfortunately, pentoxifylline only is effective in 30-40% of patients and must be taken 3 times daily. If it is effective, walking distances only improve modestly. Cilostazol, on the other hand, is a newer agent that belongs to the class of phosphodiesterase III inhibitors and has been shown to be more effective statistically than either pentoxifylline or placebo. The mechanism of action is not well understood. Adverse effects may include headache and loose stools, but the medication generally is well tolerated. It should not be used in patients with significant congestive heart failure.

**Surgical Therapy**

Direct arterial reconstruction on the diseased aortoiliac arterial segment is well established. Aortoiliac endarterectomy (TEA) and aortobifemoral bypass (AFB) are the 2 traditional means of surgically treating aortoiliac occlusive disease (AIOD). Both procedures have similar risk and results, and the outcomes have stood the test of time. In 1966, Blaisdell introduced axillofemoral bypass as an extra-anatomic technique for improving inflow to the lower extremities without the need for an abdominal procedure. More recently, with the advent of arterial stents, endovascular repair of aortoiliac lesions has become a reasonable alternative to consider if the pathologic anatomy is suitable.

TEA of the aorta and iliac arteries was the first reconstructive procedure performed for aortoiliac occlusive disease (AIOD). The concept is simple. A dissection plane exists between the arterial media and the obstructing plaque. When the appropriate plane is entered, the arterial intima, plaque, and internal elastic lamina of the media are removed as a single specimen. Early in the experience, surgeons were
concerned that the remaining portion of arterial wall was not sturdy enough to hold blood under arterial pressure. However, with more experience, it became clear that the remaining portion of the vessel (external elastic lamina of the media and adventitia) following TEA provided a secure and durable conduit with excellent long-term patency. When aortoiliac TEA is used to treat patients with type I atherosclerotic occlusive disease, the patency rates are excellent. However, the results are not as good when TEA is applied to patients with more extensive distal occlusive lesions in the external iliac and femoral arteries.

Today, aortoiliac TEA is not used as commonly as AFB, primarily because the procedure is best suited for patients with type I atherosclerosis, occlusive disease limited to the infrarenal aorta and common iliac arteries, as depicted in the image below. Patients with type I atherosclerosis comprise the minority of patients with PAD. Furthermore, younger surgeons may not have had proper exposure to the technique of aortoiliac TEA during their training and therefore do not have appropriate experience using the procedure to treat aortoiliac occlusive disease (AIOD).
Type I atherosclerosis with occlusive disease limited to the infrarenal aorta and common iliac arteries.

Aortoiliac TEA has significant advantages over conventional AFB for the treatment of aortoiliac occlusive disease (AIOD). First and most obvious is that prosthetic material is not needed to perform the arterial reconstruction. Even for the most experienced surgeons, aortic prosthetic graft infections occur in 0.5-3% of patients following AFB. The mortality rate associated with the treatment of aortic graft infection ranges from 11-27%. In addition, a similar rate is observed for early amputation following aortic graft infection.

Many patients undergo aortoiliac TEA not for the indication of removing the plaques that obstruct blood flow, but rather to remove the source of
atheroembolism causing blue toe syndrome. Aortoiliac TEA is ideally suited for this indication because the offending degenerated atheroma is removed, leaving a clean, glistening surface that soon is covered by new functional endothelium.

The only significant disadvantage of TEA compared with AFB is that a larger, more meticulous dissection is required to expose and control the branches of the infrarenal aorta. For this reason, some surgeons may opt for AFB even if the occlusive process is limited to the infrarenal aorta and common iliac arteries.

Finally, aortoiliac TEA should not be performed if occlusive plaques involve the more distal external iliac and femoral arteries. The "tail" of the atheroma in the common iliac artery may extend into the orifice of the external iliac artery, and this tongue usually is removed easily during the course of the procedure. However, if diffuse disease exists in the more distal external iliac and femoral arteries, AFB is a more suitable alternative.

AFB is the most common open surgical alternative used to treat aortoiliac occlusive disease (AIOD). In the early experience of aortic surgery, unilateral AFB or even aortoiliac bypass (AIB) often was performed to limit the extent of the procedure. However, as more experience was gained with these operations, using the common femoral arteries as the outflow target clearly produced better long-term patency results. Unilateral AFB is performed infrequently today because the extremity that was neglected initially seldom truly is healthy and invariably demonstrates symptoms from progressive atherosclerosis. Therefore, performing AFB bilaterally to avoid the need for subsequent inflow operative procedures on the limb that demonstrates less extensive occlusive disease is appropriate.

The original approach to AFB was transabdominal. As an alternative approach, retroperitoneal exposure of the aorta can be used to avoid entering the peritoneum. Some authors have advocated this approach based upon a theoretical advantage of fewer pulmonary problems, more rapid resolution of postoperative ileus, and fewer days in the hospital. Other studies have not supported this proposed benefit.
In some circumstances, a bypass serving both legs can be constructed using a single common iliac artery as the donor site. This procedure can be performed through either a transperitoneal or retroperitoneal approach.

For higher-risk patients who are less likely to tolerate an abdominal operation, extra-anatomic bypass was developed in the mid 1960s. Axillobifemoral bypass provided an extracavitary means of improving blood flow to the lower extremities. This procedure proved especially useful in the treatment of aortic graft infections. However, the long-term patency of extra-anatomic bypass is distinctly inferior to conventional aortobifemoral bypass.

With the advent of percutaneous transluminal angioplasty (PTA) and stents, excellent minimally invasive alternatives to conventional open reconstructive surgery now are available. If applied to the appropriate anatomical problem, the results of iliac angioplasty/stent placement rival open surgical results. For isolated segmental common iliac artery stenoses, angioplasty/stenting rivals open surgical results. However, for occlusive disease that diffusely involves the aortoiliac segment, direct open surgical repair still offers the best long-term outcome. However, through catheter-based treatments (angioplasty/stenting), patients with significant operative risk due to comorbid diseases can be offered therapy.

**Preoperative Details**

Because most patients with aortoiliac occlusive disease (AIOD) are older than 50 years, finding associated ischemic heart disease is not uncommon, even if classic anginal symptoms are not present. Hertzer and colleagues found that most patients undergoing aortic operations for arterial occlusive disease had diseased coronary arteries when coronary arteriography was performed. Moreover, Porter and associates found a significant incidence of occlusive plaques in the carotid arteries in a similar group of patients. Clearly, aortoiliac occlusive disease (AIOD) does not exist in a vacuum. However, despite the association of coronary and extracranial arterial occlusive disease with peripheral arterial disease (PAD), every patient clearly does not need an extensive preoperative evaluation prior to undergoing aortic surgery.
Preoperative cardiac evaluations are reserved for patients with either an abnormal finding on ECG and/or a history of new-onset or unstable angina or with symptoms of ventricular dysfunction (orthopnea, dyspnea on exertion). Patients who have had coronary angioplasty or bypass or who have a history of stable angina on appropriate medication probably do not need a preoperative cardiac evaluation, unless a change has occurred in either exercise tolerance or anginal pattern.

Immediately before the induction of anesthesia, the anesthesiologist places an epidural catheter. Although the catheter is not used during the procedure, the analgesic relief provided by instillation of narcotic and local anesthetic agents in the postoperative period is invaluable.

In addition, a systemic dose of perioperative cephalosporin antibiotic is administered intravenously before the skin incision is made. The antibiotic is continued postoperatively for 24-28 hours to lower the risk of graft infection.

**Intraoperative Details**

Both of the conventional surgical procedures used to treat aortoiliac occlusive disease (AIOD), TEA and AFB, are performed through either a longitudinal midline or transverse intra-abdominal incision and even may be performed via a retroperitoneal exposure to the aorta. More dissection is needed with TEA. Total circumferential mobilization of the infrarenal aorta and common iliac arteries is required in order to perform aortoiliac TEA. The proximal extent of the dissection is the level of the renal arteries as long as the occlusive plaques do not extend proximally and impinge on the orifices of these vessels. If occlusive disease extends cephalad to the renal arteries, the dissection must be carried proximally to the origin of the superior mesenteric artery (SMA) to allow placement of the aortic occluding clamp at the base of this vessel.

An alternative approach for suprarenal control of the aorta is placement of the aortic cross clamp above the level of the celiac axis, a maneuver that is not difficult and is quite familiar to vascular surgeons. The lumbar, middle sacral, and inferior mesenteric (IMA) arteries must be managed using vessel loops to control back bleeding when the aorta is opened. Take care to identify and preserve any accessory renal arterial branches that may be present in as many as 20% of patients. In addition, the proximal portion of the external iliac and hypogastric
arteries should be dissected adequately to allow placement of occluding clamps on these vessels distal enough from the origin to view the proximal portion of the external iliac artery. The difficult portion of the dissection occurs around the distal aorta and proximal common iliac arteries. The inferior vena cava and common iliac veins may be quite adherent to the arteries at this point, and care must be taken to avoid injury to these veins. After the dissection is completed, 5000 units of intravenous heparin are administered prior to arterial occlusion.

The distal clamps are placed first to reduce the incidence of atheroembolism that may occur following application of the aortic clamp. The aorta is incised longitudinally extending from 2 cm distal to the aortic occluding clamp proximally to 2 cm proximal to the aortic bifurcation distally. Place the line of incision on the right side of the anterior surface of the aorta to avoid the origin of the IMA. The endarterectomy plane easily is established where the atheromatous disease is most severe. Grasp the plaque and gently push away the remaining arterial wall. The dissection is continued distally until the bifurcation is reached, and the appropriate plane is continued into the orifice of each common iliac artery.

The iliac artery may be incised longitudinally (if the length of the common iliac is long), or even transversely, directly over the common iliac bifurcation. The author prefers a longitudinal incision extending from the proximal common iliac artery 2 cm from the origin to the iliac bifurcation because it affords the surgeon a better view of the endarterectomized surface and the endarterectomy endpoint. A bridge of arterial wall is preserved between the abdominal aortic incision and the common iliac incisions. Once the entire plaque is mobilized in the common iliac, the entire specimen may be pulled in a cephalad direction and removed entirely as a single specimen resembling a pair of pants. Take care to examine the distal endpoint in the iliac artery. A tongue of atheroma may continue into the origin of the external iliac and hypogastric arteries. This tail of atheroma must be excised in a more superficial plane to avoid extending the endarterectomy into the deeper plane used to perform TEA. The plane of the atheroma actually is easy to discern because the atheroma usually is a darker yellow color and has a different consistency than the more normal adherent intima.

After any remaining plaque and/or strands of media are removed, the arteriotomies are closed with continuous polypropylene sutures. If the aorta is small (<2 cm), a
A prosthetic, zero-porosity Dacron patch is used to avoid narrowing that may occur during primary closure of a longitudinal arteriotomy. Once blood flow has been restored, femoral pulses should be palpated to confirm the presence of adequate inflow. A similar aortic exposure is used to perform AFB. In addition, the common femoral, proximal superficial femoral, and proximal profunda femoris arteries are mobilized through longitudinal groin incisions that are made just lateral to the femoral pulse. If the pulse is not present, the proper line of incision is found by measuring 3-4 finger breadths lateral to the pubic tubercle. Cover the skin in a povidone-iodine–impregnated plastic drape to help avoid skin contact with the prosthetic graft. The infrarenal aorta immediately adjacent to the renal arteries is mobilized. Circumferential mobilization of the aorta is not necessary. The common femoral artery and its branches are mobilized from the inguinal ligament to the bifurcation, exposing enough superficial femoral and profunda femoris arteries to allow placement of an arterial occluding clamp. The aortic anastomosis may be performed in either an end-to-end or an end-to-side configuration using continuous polypropylene suture. Although partial occluding aortic clamps have been used when performing end-to-side anastomoses, a better view of the aortic lumen is obtained with the use of proximal and distal clamps that totally occlude the aorta.

If the aorta is filled with atherosclerotic debris that appears loose and may embolize when flow is restored, perform an end-to-end aortic anastomosis and oversew the distal aorta. The configuration of the proximal anastomosis is not as important as its location. The anastomosis to the aorta must be placed near the renal arteries to help avoid recurrent atheromatous and/or aneurysmal disease that may involve infrarenal aorta that remains proximal to the aortic anastomosis.

Once the proximal anastomosis is completed and no bleeding is present, the limbs of the prosthetic graft are passed carefully through retroperitoneal tunnels that were made before the patient received intravenous heparin. The tunnels are made directly anterior to the iliac arteries and posterior to the ureters. The circumflex iliac veins must be avoided during creation of the tunnel and passage of the graft limbs. Partial incision of the inguinal ligament may aid in constructing the tunnel and identifying these large troublesome veins.

The common femoral artery is incised longitudinally, and a conventional end-to-side femoral anastomosis is performed using continuous polypropylene suture.
Take care to examine the origins of the 2 outflow branches (SFA and profunda) of the common femoral artery. Not uncommonly, the SFA has significant occlusive disease. If the SFA is occluded, any stenosis in the proximal portion of the profunda must be repaired to insure adequate long-term patency of the aortic graft limb. If the common femoral artery is severely diseased, limited local TEA may need to be performed to facilitate an adequate femoral anastomosis.

**Postoperative Details** In the past, all patients were monitored in an intensive care environment for the first 24-48 hours following an aortic operation. In the last decade, it has become increasingly common for patients undergoing operations for occlusive disease to avoid the ICU and have their postoperative care on a regular surgical floor. For patients with hemodynamic concerns, systemic arterial as well as pulmonary capillary wedge (PCWP) pressures help to plan intravenous fluid requirements. In addition, hourly urinary output through a bladder catheter is recorded. Although significant blood loss is not common, the hematocrit is monitored every 6-12 hours during the first 24 hours.

If the operation has proceeded smoothly, perform extubation at the end of the procedure. Preoperative pulmonary function tests help to predict which patients are likely to develop postoperative respiratory problems. When the forced expiratory volume in 1 second (FEV1) is less than 1000 cc, one can anticipate difficult respiratory problems associated with conventional aortic surgical approaches through midline incisions. For such cases, a transverse intra-abdominal or retroperitoneal incision may help to reduce postoperative respiratory complications.

Most large fluid shifts occur following aortic surgery and are related to the size of the dissection and the length of the operation as well as the amount of intraoperative blood loss. Patients tend to gain significant "wet weight" during the first 48 hours postoperatively. By the beginning of the third postoperative day, mobilization of the excess water back into the intravascular compartment begins to occur. Urine output increases, PCWP rises, and hematocrit may drift downward.
Also monitor the perfusion to the lower extremities carefully. If pedal pulses cannot be palpated due to SFA occlusive disease, monitor Doppler flow as well as ABI. After successful revascularization, ABI should increase by at least 15%.

**Follow-up**

In general, results of the treatment for aortoiliac occlusive disease (AIOD) are excellent, but patients still need follow-up care at regular intervals. See patients every 3-6 months for the first year and every 6-12 months thereafter. If a prosthetic graft has been implanted, a lifelong risk of graft infection exists that the patient must recognize. Moreover, oral antibiotic prophylaxis is appropriate before dental procedures, urologic instrumentation, sigmoidoscopy, or other gastrointestinal surgical procedures.

**Complications** Several complications are related both to aortoiliac TEA and AFB, and others are associated only with one or the other. Perioperative thrombosis may be a complication of either procedure and generally is related to technical problems. For example, a plaque that dissects, causing restriction in blood flow and subsequent thrombosis, may occur as a complication of either procedure. Visualization of endarterectomy endpoints and suturing of plaques that may elevate when blood flow is restored may help to reduce the risk of dissection and subsequent thrombosis.

Intraoperative atheroembolism is another complication that may occur during surgical dissection and mobilization of the vessels or following release of the occluding clamps during reperfusion. Meticulous dissection during mobilization of the arteries is imperative. Furthermore, placement of the distal occluding clamps before application of the proximal clamps may help to reduce the risk of atheroembolism that is inherent during any aortic operation.

Injury to adjacent structures (ie, duodenum, inferior vena cava, iliac veins, ureters) usually is easy to avoid with careful technique. However, care must be used with mechanical retractors to avoid inadvertent injury to adjacent structures. Care is necessary both in the retroperitoneum and in the groin to avoid injury to nerves adjacent to major vessels.
Careful closure of groin wounds is necessary in order to avoid a lymphocele, which can lead to graft infection.

A specific complication related to the use of prosthetic material for AFB is development of aortic graft infection, which occurs in 0.5-3% of cases. Usually, presentation of the infection follows the aortic procedure by a significant length of time (20-24 mo). Most commonly, a complication of healing in the groin wound is the first sign that a serious, life-threatening and limb-threatening problem must be dealt with.

Graft infections can be classified into 2 groups, depending on the etiologic microbial involved. The more virulent organisms (ie, Staphylococcus aureus, gram-negative bacilli) usually are responsible for causing a more severe type of clinical infection. When systemic signs of sepsis occur with graft infection, a virulent organism is present.

On the other hand, a significant number of graft infections are caused by Staphylococcus epidermidis. These infections are much more indolent, and the extent of graft involvement may be harder to determine. Even with the most skilled physician, the mortality rate following treatment of aortic graft infection is 11-27%. Moreover, the risk of amputation following graft infection is almost as high.

Major complications rates associated with catheter-based treatments (percutaneous transluminal angioplasty/stents) for aortoiliac occlusive disease range from 2.3-17%. The problem can occur in the target vessel, the access site, or even other arteries that are far removed anatomically (ie, atheroembolism). These complications include dissection, acute thrombosis, atheroembolism, and even arterial perforation. Complications related to the contrast agent (ie, anaphylaxis [rarely] or contrast-induced renal dysfunction) are uncommon.

**Outcome and Prognosis**

Outcomes following aortic operations for aortoiliac occlusive disease (AIOD) are measured in terms of operative mortality rates and patency of the arterial reconstruction over time. These outcomes are similar for both aortoiliac TEA or AFB. The operative mortality rate (30-d) is 2-3%. Long-term patency is excellent
too. The patency rate at 5 years following AFB or TEA is 85-90%. If patients continue to smoke, however, these excellent patency rates are reduced by half.

Outcomes for extra-anatomic (axillofemoral/femoral-femoral) bypasses are clearly not as good as either AFB or aortoiliac TEA. Operative mortality rates for extra-anatomic bypass might be expected to be better than AFB due to the extracavitary nature of these procedures and the fact that aortic occlusion is not required during the course of the operation. However, an operative mortality rate of 0-4% for femorofemoral bypass and 2-11% for axillobifemoral bypass is a reflection of the selected patients in whom these procedures are performed. Five-year primary patency of extra-anatomic bypasses performed for aortoiliac occlusive disease (AIOD) ranges from 19-50% for axillobifemoral bypass and 44-85% for femorofemoral bypass.

Endovascular techniques (ie, percutaneous transluminal angioplasty, stent placement) offer alternatives to conventional surgical repair. Therefore, understanding the outcomes offered with such interventions is important. Although isolated stenosis of the infrarenal aorta or common iliac artery is uncommon, this lesion is suited ideally to percutaneous transluminal angioplasty (PTA) and/or stent placement. With localized, segmental occlusive disease in the aorta, initial technical success can be achieved in 95% of cases, with 5-year patency rates of 80-87% using percutaneous transluminal angioplasty (PTA). Initial success rates using percutaneous transluminal angioplasty (PTA) for iliac lesions are 93-97%, with 5-year patency rates of 54-85%. These results seem to be improved when arterial stents are used either primarily or as an adjunct to percutaneous transluminal angioplasty (PTA) for the treatment of iliac artery stenosis.

**Future and Controversies**

No controversy exists regarding the appropriate surgical procedure to treat aortoiliac occlusive disease (AIOD). Use TEA only in cases of type I atherosclerosis. TEA also is an excellent option for those patients with blue toe syndrome from severe ulcerogenic aortoiliac atherosclerosis that involves only the infrarenal aorta and common iliac arteries.
Some authors have advocated performing the aortic procedure through a retroperitoneal rather than an intra-abdominal approach. Unfortunately, despite some excellent work in this area, outcomes are similar whether the procedure is performed in a retroperitoneal or transabdominal fashion.

A more controversial area is whether proximal occlusive disease should be treated nonoperatively, using angioplasty and stent placement rather than the more invasive aortic operation. It seems clear that angioplasty and/or stent placement is a suitable alternative for patients with very focal occlusive disease in the common iliac artery but offers a poor alternative for more diffuse disease that involves the external iliac artery. Furthermore, the patency results for patients who have had total occlusions in the iliac arteries treated by endovascular therapy are definitely inferior to conventional surgical results.

The current controversy involves the appropriate place for minimally invasive treatment of aortoiliac occlusive disease (AIOD). Laparoscopically assisted AFBs have been performed both in animals and humans with satisfactory results. However, a significant learning curve seems to be involved, and no long-term follow-up data are available for review.
Infrainguinal Occlusive Disease

Introduction

This material is a review of chronic infrainguinal atherosclerotic arterial occlusive disease caused by atherosclerosis involving the femoral, popliteal, and/or infrapopliteal arteries. Because chronic atherosclerotic disease may result in acute circulatory compromise, acute arterial occlusion is also covered. Less common etiologies of lower extremity arterial insufficiency, such as atheroembolism, Buerger disease, popliteal artery entrapment syndrome, and cystic adventitial disease, are briefly discussed.

Decision-making in the management of vascular disease changes frequently as new information becomes available and as new technologies emerge.1 Furthermore, therapeutic recommendations for a given population may not be applicable to individual patients with even slightly differing risk factors, co-morbidities, or vascular anatomy.2

Problem

Although most patients with infrainguinal disease are treated nonoperatively, over 100,000 vascular reconstructive procedures are performed yearly in the United States alone. Unfortunately, intervention fails in up to 50% of cases within 5 years.3

Of the symptomatic patients under medical care, within 5 years, approximately 25% develop progressive symptoms, 5-10% require surgical intervention, and 1-2% undergo major amputation.4 The vast majority of patients with intermittent claudication remain stable or improve with noninvasive management. According to Baumgartner, Schainfeld, and Graziani, 25% of patients with claudication will eventually require revascularization and only 5% will develop critical limb ischemia. Within the first year after the initial diagnosis, 6-9% of patients require
intervention. Subsequently 2-3% of patients per year require intervention. Because lower extremity atherosclerosis is a marker for systemic atherosclerotic disease, these patients have significant systemic morbidities. Thirty percent of patients with peripheral artery disease die within 5 years and 40% die within 10 years.4,6 Feringa et al observed a cohort of 2,642 patients having ankle-brachial indices less than or equal to 0.9.6 They discovered that the major factors associated with mortality in this group of patients included renal dysfunction, heart failure, ST-segment changes, age greater than 65 years, hypercholesterolemia, ankle-brachial index lower than 0.60, Q-waves, diabetes, cerebrovascular disease, and pulmonary disease. They also found that the use of statins, aspirin, and beta-blockers correlated with reduced 10-year mortality.

**Frequency**

Chronic atherosclerotic lower extremity disease is present in 20% of the population older than 55 years.7 Most affected persons are asymptomatic. In fact, estimates indicate that only approximately 20% of people with atherosclerotic lower extremity disease present to a physician because of symptoms. Another 20% are symptomatic but do not seek medical attention.

**Etiology** Commonly accepted risk factors for both the occurrence and progression of atherosclerotic vascular disease include abnormal glucose tolerance, cigarette smoking, advanced age, hyperlipidemia, and hypertension.

Certain biochemical factors have also been shown to be independent risk factors for atherosclerotic peripheral vascular disease. Such factors include increased plasma fibrinogen levels, hyperhomocysteinemia, and high-sensitivity C-reactive protein. These factors may also increase the risk of bypass graft stenosis and reocclusion. When more than one risk factor is present, the cumulative risk is often greater than individual risk factors combined. This is especially true of cigarette smoking, which, when accompanied by another risk factor (such as hypertension or hyperlipidemia) increases the disease risk to more than twice the sum of the individual risks.
**Pathophysiology**

**Atherosclerotic occlusive disease**

With atherosclerotic occlusion of a major lower extremity artery, the limb is perfused via collateral pathways. Although this alternate pathway may be adequate at rest, it becomes inadequate as the oxygen demands of the leg musculature increase with activity. This results in calf muscle pain or fatigue, a symptom known as intermittent claudication. As the degree of atherosclerotic occlusion worsens, blood flow, even at rest, may become impaired. This may cause ischemic pain at rest, ischemic ulceration, and gangrene.

**Acute arterial occlusion**

**Acute occlusion of peripheral arteries** commonly involves the infrainguinal segment. Underlying atherosclerotic disease may result in intraluminal strictures that impair blood flow and cause acute thrombosis. Emboli typically lodge at bifurcations and, hence, tend to occlude the distal common femoral artery (the most common site, comprising 34% of all arterial emboli) or distal popliteal artery (14%). Popliteal artery aneurysms may thrombose as a result of turbulent blood flow. The clinical indications of acute occlusion of lower extremity arteries are the following classic 6 "P"s:

- Paresthesias
- Pain
- Poikilothermia (coldness) or perishing cold
- Pallor
- Pulselessness
- Paralysis

The anatomic level at which pulse loss occurs helps identify the location of the occlusion.
Presentation

Most people harboring atherosclerotic disease of the lower extremities are asymptomatic; others develop ischemic symptoms. Some patients attribute ambulatory difficulties to "old age," unaware of the existence of a potentially correctible problem.

Symptomatic patients may present with intermittent claudication, ischemic pain at rest, nonhealing ulceration of the foot, as depicted in the image below, or frank ischemia of the foot.

Pressure ulcer of the heel exacerbated by infrainguinal arterial occlusive disease.

Cramping or fatigue of major muscle groups in one or both lower extremities that is reproducible upon walking a specific distance suggests intermittent claudication. This symptom increases during ambulation until walking is no longer possible, and it is relieved by several minutes of rest. The onset of claudication may occur sooner with more rapid walking or when walking uphill or up stairs. The claudication of infrainguinal occlusive disease typically involves the calf muscles, while symptoms that occur in the buttocks or thighs suggest aortoiliac occlusive disease.

Physical; Physical examination discloses absent or diminished peripheral pulses below a certain level. Although diminished common femoral artery pulsation is characteristic of aortoiliac disease, infrainguinal disease alone is characterized by normal femoral pulses at the level of the inguinal ligament and diminished or absent pulses distally.

Specifically, loss of the femoral pulse just below the inguinal ligament occurs with a proximal superficial femoral artery occlusion. Loss of the popliteal artery pulse suggests superficial femoral artery occlusion, typically in the adductor canal. Loss of pedal pulses is characteristic of disease involving the distal popliteal artery or its trifurcation.
Importantly however, be aware that absence of the dorsalis pedis pulse may be a normal anatomic variant, noted in approximately 10% of the population. On the other hand, the posterior tibial pulse is present in 99.8% of persons aged 0-19 years. Hence, absence of both pedal pulses is a more specific indicator of peripheral arterial disease.

Other findings suggestive of atherosclerotic disease include a bruit heard overlying the iliac or femoral arteries, skin atrophy, loss of pedal hair growth, cyanosis of the toes, ulceration or ischemic necrosis, and, after 1-2 minutes of elevation above heart level, pallor of the involved foot followed by dependent rubor, as depicted in the image below.

Cyanosis of the first toe and dependent rubor of the foot characteristic of arterial insufficiency.

**Differential diagnoses**

**Pseudoclaudication**

Although ischemic findings in the face of absent pulses clearly pinpoint arterial insufficiency as the culprit, intermittent claudication, even when associated with absent pulses, is not always due to arterial insufficiency.

If symptoms are not always reproducible (ie, the patient sometimes has "good days" when ambulation is not limited by claudication) or if the symptoms are associated with low back pain or radiculopathy, the clinician should consider the possibility of pseudoclaudication, which occurs as a result of spinal stenosis or cauda equina syndrome.

In that case, the pulse deficit may be an incidental finding of asymptomatic atherosclerosis. Noninvasive vascular laboratory testing (see Lab Studies), lumbosacral imaging, and neurologic evaluation all may contribute to distinguishing between these possibilities.
Two rather unusual conditions, venous claudication due to extensive iliofemoral venous thrombosis and chronic compartment syndrome due to calf muscle hypertrophy in athletes, result in a bursting type of pain in the calf with ambulation, which subsides slowly with elevation. In each case, the etiology is impaired venous outflow.

**Atheroembolism;**

Patchy areas of ischemia involving the feet, especially in the presence of palpable pedal pulses, suggest the possibility of atheroembolism of plaque fragments from ulcerated, although nonocclusive, proximal atherosclerotic plaques or from thrombus lining the wall of an infrarenal aortic aneurysm (see Abdominal Aortic Aneurysm).

**Buerger disease**

Severe ischemia of the toes with absent pedal pulses but normal proximal pulses in a man aged 35-50 years who smokes cigarettes may be the result of Buerger disease (thromboangiitis obliterans).15 Ischemia of the fingers may also be present. The digits are cool, moist, mottled, and sometimes have tender shallow ulcers. Migratory superficial phlebitis may occur.

**Collagen-vascular disease** must be excluded.16 See the eMedicine article Buerger Disease (Thromboangiitis Obliterans). Angiography reveals pathognomonic findings of "corkscrew" arteries. Treatment is discontinuation of smoking and good local wound care. Vascular surgery is rarely possible because of the poor quality of the distal arteries.

**Complex regional pain syndromes** (eg, posttraumatic pain syndromes, causalgia, mimocausalgia, Sudeck atrophy, reflex sympathetic dystrophy); Complex regional pain syndromes (CRPSs) have been classified by the World Health Organization as CRPS II (ie, causalgia), which is associated with a demonstrable nerve injury, and as CRPS I (ie, mimocausalgia, reflex sympathetic dystrophy, Sudeck atrophy), which includes the remainder. Causalgia (ie, causos, heat; algos, pain) was first described in patients with arterial and nerve injuries sustained during the American Civil War. Both variants remain poorly understood and often misdiagnosed.
Although the exact pathophysiology is elusive, the sympathetic nervous system clearly plays a focal role. Therefore, surgical sympathectomy—perfected decades ago by vascular surgeons to manage nonreconstructible arterial disease (a common situation at the time)—was once the mainstay for treatment of the CRPSs.

Although surgical sympathectomy is now mostly notable only for historic purposes, sympathetic blockade is quite effective and is commonly performed for the CRPSs. Hence, currently the treatment of CRPSs is performed mainly by pain management specialists. Nonetheless, because the vascular surgeon has always been primarily responsible for the diagnosis of extremity symptoms, it is not uncommon for patients with CRPS to report to a vascular surgeon because of extremity pain. Such pain may occur after extremity trauma but may seem disproportionate to the degree of injury. Pain may also manifest after delayed revascularization of an acutely ischemic extremity. The diagnosis is often one of exclusion and thus requires a high index of clinical suspicion. The diagnosis should be considered more strongly if severe superficial burning pain and agonizing hypersensitivity are present and are associated with vasomotor abnormalities such as edema, erythema, and hyperhidrosis. Radiographic studies may demonstrate relative and patchy osteopenia in the involved extremity. In addition to symptomatic relief, management of the CRPSs requires sympathetic blockade. This is best performed during the early, acute stage when the clinical course may be reversible. As the disease progresses, the erythema gives way to cyanotic mottling, the acute edema transforms to brawny edema, and the pain becomes unrelenting and disabling. These findings occur at approximately the third month and represent the second, or dystrophic, stage. At this point, both plain radiographs and bone scans tend to demonstrate indicative findings.

Over time, disuse leads to atrophy, soft tissue fibrosis, and joint contractures. Radiographs confirm ankylosis and severe osteoporosis. This signals the third, or atrophic, stage. Note that atrophy can also occur because of intentional disuse for anticipated secondary gain. Such patients reportedly do not respond to treatment until litigation has concluded.
Typically, the clinician does not even consider the diagnosis of a CRPS until the second stage. At that point, a dramatic clinical response to sympathetic blockade may confirm the diagnosis. Unfortunately, too much damage may have already occurred for sympathetic ablation to be effective and to break the vicious cycle of pain, sympathetic overactivity, and pain; the progression of the CRPS may be inexorable and irreversible.;One other caveat is that in the face of coexisting arterial disease, the vascular surgeon who may attribute the symptoms to ischemia and thereby may consider bypass should be aware that a surgical incision tends to exacerbate the pain in an extremity afflicted with a CRPS.

Indications

Indications for lower extremity revascularization include gangrene, pain at rest, nonhealing arterial ulcer, and disabling claudication.

Relevant Anatomy

The inguinal (Poupart) ligament is a tough, fibrous band stretching from the anterior superior iliac spine to the pubic tubercle. The common femoral artery is a continuation of the external iliac artery, beginning just under the middle of the inguinal ligament. It is palpable as the femoral pulse and is well suited to both percutaneous and surgical access because of its relatively superficial position. Approximately 1-2 inches distal to the inguinal ligament, the common femoral artery divides into the deep femoral (profunda femoris) artery, usually arising in the posterolateral position, and the superficial femoral artery.

The deep femoral artery gives rise to several very proximal branches that tend to maintain patency even in persons with extensive atherosclerotic disease, thus providing the major source of collateral circulation around an occluded superficial femoral artery.

The term superficial femoral artery is somewhat of a misnomer in that it is superficial for only a few inches until it courses under the sartorius muscle and into the aponeurotic covering of the adductor (Hunter) canal. When it emerges anterior
to the adductor magnus, the superficial femoral artery becomes the popliteal artery. Because the popliteal artery is bounded posteriorly by the popliteal vein, nerve, and fascia and the semimembranosus, gastrocnemius, plantaris, and soleus muscles, it is the most difficult of the lower extremity pulses to accurately assess.

The popliteal artery passes posterior to the knee joint and into the upper leg where, just distal to the popliteus muscle, it divides into the anterior tibial artery and the tibioperoneal trunk. The anterior tibial artery passes laterally through the interosseous membrane and lies on the interosseous membrane throughout much of the leg. As it reaches the lower leg, it lies on the tibia and then becomes superficial at the ankle joint, at which point it is called the dorsalis pedis artery and, hence, is palpable as the dorsalis pedis pulse.

The tibioperoneal trunk divides within approximately 1 inch of its origin into the peroneal artery and the posterior tibial artery. The peroneal artery lies on the medial surface of the fibula and ends in terminal branches near the os calcis. The peroneal artery, which is too deep to be palpable as a pulse, often remains patent despite atherosclerotic occlusion of the anterior and posterior tibial arteries and, thus, may be a usable site for the distal anastomosis of bypass grafts in patients with advanced infrapopliteal occlusive disease. The posterior tibial artery runs along the medial side of the leg and posterior to the medial malleolus, where it is superficial and palpable as the posterior tibial pulse.

**The greater (long) saphenous vein** originates on the medial side of the dorsum of the foot and runs anterior to the medial malleolus. It then runs posteromedially to the tibia, posteriorly to the medial condyle of the femur, and along the medial thigh, coursing anteriorly until it enters the femoral vein through the foramen ovale, just below the inguinal ligament. The length and relatively superficial course of the greater saphenous vein make it ideally suited for use in infrainguinal bypass surgery.

**Contraindications**; In nonambulatory patients with ischemic pain at rest, gangrene, or extensive nonhealing wounds, primary lower extremity amputation may be a better choice than vascular bypass surgery.
Atherosclerotic Disease of the Carotid Artery

Introduction

Atherosclerosis is a degenerative disease of the arteries resulting in plaques consisting of necrotic cells, lipids, and cholesterol crystals. These plaques can result in symptoms by causing a stenosis, embolizing, and thrombosing. Atherosclerosis is a diffuse process with a predilection for certain arteries. This article describes the history and impact of this process as it occurs in the extracranial carotid artery.

History of the Procedure

The ancient Greeks recognized the importance of the extracranial carotid artery and named it from the Greek word karoo, which means to stupefy. In 1875, Growers described a patient with right hemiplegia that he attributed to an occluded left carotid artery. In 1914, Hunt emphasized the relationship between extracranial carotid disease and stroke using the phrase cerebral intermittent claudication. The surgical management of stroke was suggested in 1951 by Fisher who stated the following: "It is even conceivable that some day vascular surgery will find a way to bypass the occluded portion of the artery during the period of ominous fleeting symptoms."

The initial report of a successful surgical resection of a carotid plaque and primary anastomosis came from Eastcott, Pickering, and Rob in 1954. In 1975, DeBakey reported the 19-year follow-up of a carotid endarterectomy, the current procedure used to surgically manage atherosclerotic disease of the carotid bulb.
**Problem**

Stroke from any cause represents the third leading cause of death in the United States. Half a million new strokes occur each year in the United States, resulting in approximately 150,000 deaths. Stroke is the leading cause of serious long-term disability in the United States. Direct and indirect cost of stroke in the United States in 1997 was estimated at $40 billion.

**Frequency**

Incidence of new stroke is approximately 160 cases per 100,000 population per year. The incidence and mortality rate of stroke have reached a plateau over the past 10 years. The risk of stroke increases with age, hypertension, the presence of a carotid bruit, diabetes, smoking, atrial fibrillation, obesity, hyperlipidemia, and elevated homocysteine level.

**Etiology**

Ninety percent of all extracranial carotid lesions are due to atherosclerosis.

The exact cause of atherosclerosis is unknown, and it may be the result of multiple etiologies. This concept has been referred to as the response to injury hypothesis. Infectious agents, hypertension, hyperlipidemia, and cigarette smoking have been cited as potential causes of atherosclerosis.

Other etiologies for carotid lesions include the following:

* Aneurysms
* Arteritis
* Carotid dissection
* Coils and kinks
* Fibromuscular dysplasia
* Radiation
* Vasospasm
Pathophysiology

Currently, embolization is considered the most common mechanism causing ischemic strokes from atherosclerotic lesions in the carotid bulb. Thrombosis and low flow are other possible mechanisms. Stroke is one of the most devastating complications of carotid stenosis. However, carotid stenosis is not the only cause of stroke. In fact, consider that 45% of strokes in patients with asymptomatic stenosis of 60-99% may be caused by lacunar infarcts or cardiac emboli.

Presentation

Amaurosis fugax (transient visual loss)

Transient ischemic attacks (TIAs)

Crescendo TIAs

Stroke-in-evolution

Cerebral infarction

Indications

The following are indications for carotid endarterectomy based on prospective randomized trials: Symptomatic patients with greater than 70% stenosis: Clear benefit was found in the North American Symptomatic Carotid Endarterectomy Trial (NASCET); ipsilateral stroke in 2 years was 9% with surgery and 26% with medical management.1

Symptomatic patients with greater than 50-69% stenosis: Benefit is marginal and appears to be greater for male patients.

Asymptomatic patients with greater than 60% stenosis: Benefit is significantly less than symptomatic patients with greater than 70% stenosis.
Note

Available literature includes considerable overlap in the percent of stenosis used as the threshold for carotid endarterectomy. In general, symptomatic patients with greater than 50% stenosis and healthy, asymptomatic patients with greater than 60% stenosis warrant consideration for carotid endarterectomy.

Symptomatic trials include patients with TIAs or minor strokes within 3 months of entry.

**Relevant Anatomy**

The carotid artery on the right originates from the innominate artery and on the left directly from the aortic arch. The carotid artery enlarges in the mid neck, forming the carotid bulb. It then bifurcates into the external and internal carotid arteries. The carotid sinus and carotid body are located at the bifurcation (see Image 1).

*Arteriogram of the aortic arch and its branches.*
Contraindications

Contraindications to carotid endarterectomy include the following:

- Patients with a severe neurologic deficit following a cerebral infarction
- Patients with an occluded carotid artery
- Concurrent medical illness that would significantly limit the patient's life expectancy

Laboratory Studies

CBC count

Electrolytes, BUN, creatinine

Lipid profile

Prothrombin time (PT)/activated partial thromboplastin time (aPTT): Heparin is administered during carotid endarterectomy, and knowing the PT/aPTT preoperatively is important.

Imaging Studies
CT scan or MRI of the head: All symptomatic patients should have a scan of the head to rule out other intracranial lesions and identify the presence of new and old cerebral infarcts.

**Carotid duplex**

Carotid duplex, with or without color, is the screening test of choice to evaluate for carotid stenosis.

*Many surgeons operate after seeing the results of a carotid duplex alone if the laboratory has credentials and is validated.*

Carotid magnetic resonance angiography

Carotid magnetic resonance angiography (MRA) has a tendency to overstate the significance of the stenosis. Its exact role is not well defined; it may be useful in collaborating the finding of an occluded carotid with duplex.

**Other Tests**

**Electrocardiogram**. Evidence of prior myocardial infarction (MI) and ischemic changes are important to identify.

The most common cause of mortality following carotid endarterectomy is MI.

**Diagnostic Procedures**
Arch and carotid arteriography

This procedure was used in the NASCET to evaluate the percent of stenosis.

The diameter of the narrowest portion of the lesion is divided by the normal internal carotid artery diameter distal to the lesion.

This procedure may be associated with a 1-2% risk of stroke.

Treatment

Medical Therapy

Antiplatelet agents

Aspirin (30–1350 mg qd) irreversibly acetylates the cyclooxygenase of platelets, thus inhibiting platelet synthesis of thromboxane A2. Prostacyclin production in the endothelium is reduced, but this effect is reversible and short-lived. A reduction in TIs, stroke, and death in men was shown in the Canadian Cooperative Study Group.2

Ticlopidine (250 mg bid) is a thienopyridine that irreversibly alters the platelet membrane and inhibits platelet aggregation. It is approximately 10% more effective than aspirin. Toxicity includes neutropenia and diarrhea.

Clopidogrel (75 mg qd) is similar to ticlopidine; risk of neutropenia is low.

Anticoagulation: Warfarin (titrated international normalized ratio [INR] 2–3) use in patients with noncardiac emboli is controversial.

Of the indications listed above (see Indications section), medical management was found to be inferior to carotid endarterectomy.

Surgical Therapy

Endovascular therapy

Carotid angioplasty and stenting
Carotid angioplasty and stenting (CAS) has emerged as a viable option in the
treatment of carotid artery stenosis. Rapid growth and technologic advancements
have allowed this procedure to become a treatment strategy, particularly in high-
risk patients.

Numerous studies, including the Stenting and Angioplasty with Protection in
Patients at High Risk for Endarterectomy (SAPPHIRE) trial, have found that CAS
is not inferior to carotid endarterectomy (CEA) at one year. Published long-term
results show no significant difference between groups in the prespecified
secondary endpoint trial, a composite at 3 years of death, stroke, or myocardial
infarction (MI) within 30 days of the procedure (or death or ipsilateral stroke
between 31 and 1080 days).3

Another study, endarterectomy versus stenting in patients with symptomatic severe
carotid stenosis (EVA-3S trial), revealed a higher stroke and death rate in CAS;
however, cerebral protection was not uniformly used and dual antiplatelet therapy
was not initiated on all patients.4

Most of the trials (see the Reference section) have shown varying results with
CAS. Many are industry sponsored and some have different patient populations (ie,
symptomatic and asymptomatic patients). Further randomized prospective studies
are needed before any conclusion can be made. Despite advances in carotid artery
stenting, carotid endarterectomy remains the current standard of care.

Currently, the Centers for Medicare and Medicaid Services (CMS) have approved
reimbursement for CAS only in symptomatic patients with a high-grade stenosis
(>70%) who are deemed high risk for CEA, and those patients who are at high risk
for CEA and have asymptomatic carotid stenosis greater than 80%.5

Procedural details

The procedure is performed in either an operating room with C-arm capabilities or
an angiographic suite.

Local anesthesia with limited sedation is used so that the patient's neurologic status
can constantly be monitored.
Femoral artery access is achieved and an arch arteriogram performed. The affected side is cannulated and selective carotid arteriograms are then performed. Next, a long sheath is placed over a wire into the common carotid artery and a 0.014 inch filter wire is placed into the internal carotid distal to the lesion to provide for embolic protection. Next, after appropriate sizing, the lesion is predilated quickly with a small balloon. The stent is then placed and postdilated with a larger balloon. A completion arteriogram is then performed to ensure that the lesion has been treated and that no other abnormalities exist within the internal carotid or cerebral views. The procedure is completed, and the access site in the femoral artery is typically closed with a closure device.

The patient is usually monitored overnight and discharged the next day.

**Carotid endarterectomy**

**Preoperative Details**

**Cardiac evaluation**

Patients with carotid artery stenosis have a high incidence of concomitant coronary artery disease.

Adherence to the American Heart Association's recommendations regarding cardiac evaluation for noncardiac surgery should be followed. In summary, they recommend a functional assessment on all patients with a history of new-onset angina and new symptoms following coronary angioplasty or bypass.

Nondiabetic patients younger than 70 years with no cardiac symptoms and normal findings on ECG may undergo carotid endarterectomy without further cardiac workup.

**Preoperative imaging studies**

Imaging studies should be used to determine the extent of stenosis and to evaluate for kinks and coils that may affect the conduct of the operation (see Imaging Studies). Many surgeons who work with certified laboratories proceed with surgery based on the carotid duplex alone. If any doubt exists regarding the degree
of stenosis or the distal extent of the disease, an arch and carotid arteriogram is performed.

The extent of the disease should also be noted, with particular attention to the superior extent of the stenosis. This may impact the type of anesthesia chosen and reveal the need for additional measures to expose an unusually high lesion.

**Intraoperative Details**

**Anesthesia**

Local anesthesia has the advantage of allowing direct evaluation of the patient's neurologic status without sophisticated monitoring. This enables the surgeon to operate on most patients without the need for a shunt, which is a technical nuisance and may pose an increased risk of stroke to the patient.

General anesthesia has the advantage of improved airway control and patient comfort during prolonged operations. However, it does require the use of routine or selected shunting, and selective shunting requires the use of electroencephalography, stump pressures, and transcranial Doppler or some other form of cerebral monitoring to assess the need for a shunt.

**Incision** A vertical incision should be made along the anterior border of the sternocleidomastoid muscle.

An oblique incision should be made in the skin fold over the carotid bifurcation.

**Endarterectomy**

The endarterectomy is carried out in a smooth plane in the media of the artery.

The most important aspect of this portion of the procedure is to obtain a smooth, tapering endpoint on the internal carotid. Occasionally, tacking sutures are required to accomplish this.

Shunt (Yavid or Pruitt-Ynahara). Closing

The endarterectomy is closed either primarily or with a patch.
The technical result should be verified by completion angiography or duplex.

Postoperative Details Perform frequent neurologic assessment.

Institute hemodynamic monitoring, with focus on maintaining the patient's blood pressure at its preoperative range.

Observe the patient for a hematoma that may compromise the airway.

Antiplatelet therapy is necessary.

**Follow-up** Patients are evaluated 2 weeks postoperatively for wound or neurologic complications. Carotid duplex is performed after 6 months and then annually.

For excellent patient education resources, visit eMedicine's Stroke Center and Cholesterol Center. Also, see eMedicine's patient education articles Stroke, High Cholesterol, Understanding Your Cholesterol Level, and Lifestyle Cholesterol Management.

**Complications** Cardiac ischemia Cranial nerve injury Hematoma with or without airway compromise Hypertension and hypotension Perioperative stroke Recurrent stenosis

**Outcome and Prognosis**

Cranial nerve injuries occur in 2-7% of patients. Recurrent laryngeal and hypoglossal nerve dysfunctions are the most common. Postoperative stroke occurs in 1-5% of patients. The perioperative mortality rate is 0.5-1.8%. Recurrent stenosis occurs in 1-20% of cases, and reoperation is necessary in 1-3% of cases. Following a successful carotid endarterectomy, the 2-year stroke risk in the NASCET was 1.6%, compared with 12.2% for the medically managed patients.1 In the NASCET, the cumulative risk of an ipsilateral stroke was 9% for the surgical patients and 26% for the medically managed patients.1 In the Asymptomatic Carotid Atherosclerosis Study (ACAS), the 5-year risk for ipsilateral stroke was 5.1% for the surgical group compared with 11% for the medical group. The stroke risk of arteriography was attributed to the surgical group and was .2%.6 In the Stenting and Angioplasty with Protection in Patients at High Risk for
Endarterectomy (SAPPHIRE) trial, carotid stenting was found to not be inferior to carotid endarterectomy in patients with severe stenosis and coexisting conditions.

Recent meta-analysis revealed that protected (use of embolic protection wire) carotid angioplasty and stenting was associated with a 30-day stroke and death rate of 2.4%.

**Future and Controversies** Carotid angioplasty and stenting research will continue to evolve, and studies are underway to evaluate its role in asymptomatic patients with high grade stenosis. As industry and interest from numerous specialties continues in carotid angioplasty and stenting, the devices available will continue to evolve.
Buerger Disease (Thromboangiitis Obliterans)

Introduction

Background

Buerger disease, a nonatherosclerotic vascular disease also known as thromboangiitis obliterans (TAO), is characterized by the absence or minimal presence of atheromas, segmental vascular inflammation, vasoocclusive phenomenon, and involvement of small- and medium-sized arteries and veins of the upper and lower extremities. The condition is strongly associated with heavy tobacco use, and progression of the disease is closely linked to continued use. The typical presentations are rest pain, unremitting ischemic ulcerations, and gangrene of the digits of hands and feet, and as the disease evolves, the patients may require several surgical amputations.1,2

The first reported case of thromboangiitis obliterans was described in Germany by von Winiwarter in an 1879 article titled "A strange form of endarteritis and endophlebitis with gangrene of the feet."3 A little more than a quarter of a century later, in Brookline, NY, Leo Buerger published a detailed description of the disease in which he referred to the clinical presentation of thromboangiitis obliterans as "presenile spontaneous gangrene."4 The paper discussed the pathological findings in 11 limbs amputated from Jewish patients with the disease.
The feet of a patient with Buerger disease. Note the ischemic ulcers on the distal portion of the left great, second, and fifth toes. Though the patient's right foot is normal in gross appearance, angiography demonstrated compromised arterial flow to both feet.

Pathophysiology

While the etiology of Buerger disease is unknown, exposure to tobacco is essential for both initiation and progression of the disease. The notion that the condition is linked to tobacco exposure is supported by the fact that the disease is more common in countries with heavy use of tobacco and is perhaps most common among natives of Bangladesh who smoke a specific type of cigarettes, homemade from raw tobacco, called "bidi." While the overwhelming majority of patients with Buerger disease smoke, a few cases have been reported in nonsmokers that have been attributed to the use of chewing tobacco.

The disease mechanism underlying Buerger disease remains unclear, but a few observations have led investigators to implicate an immunologic phenomenon that
leads to vasodysfunction and inflammatory thrombi. Patients with the disease show hypersensitivity to intradermally injected tobacco extracts, have increased cellular sensitivity to types I and III collagen, have elevated serum anti–endothelial cell antibody titers, and have impaired peripheral vasculature endothelium-dependent vasorelaxation. Increased prevalence of HLA-A9, HLA-A54, and HLA-B5 is observed in these patients, which suggests a genetic component to the disease.

**Frequency**

The prevalence of Buerger disease has decreased over the past half decade, partly because the prevalence of smoking has decreased, but also because the diagnostic criteria have become more stringent. In 1947, the prevalence of the disease in the United States was 104 cases per 100,000 population. More recently, prevalence has been estimated at 12.6-20 cases per 100,000 population.

**Mortality/Morbidity**

Death from Buerger disease is rare, but in patients with the disease who continue to smoke, 43% require 1 or more amputations in 7.6 years. Most recently, in a December 2004 CDC publication, the 2002 deaths report in the United States divided by cause of death, month, race, and sex (based on the International Classification of Diseases, Tenth Revision, 1992), reported a total of 9 deaths related to TAO, depicting male to female gender ratio of 2:1 and white to black ethnicity ratio of 8:1.

**Race**; Buerger disease is relatively less common in people of northern European descent. Natives of India, Korea, and Japan, and Israeli Jews of Ashkenazi descent, have the highest incidence of the disease.5

**Sex**; Though Buerger disease is more common in males (male-to-female ratio, 3:1), incidence is believed to be increasing among women, and this trend is postulated to be due to the increased prevalence of smoking among women.

**Age** Most patients with Buerger disease are aged 20-45 years.

**Clinical; History**
Because a firm diagnosis of Buerger disease is difficult to establish, a number of different diagnostic criteria have been proposed. Olin asserts that the following criteria must be met for the diagnosis to be made with reasonable certainty:

**Age younger than 45 years**

**Current (or recent) history of tobacco use**

**Presence of distal extremity ischemia (indicated by claudication, pain at rest, ischemic ulcers, or gangrene) documented by noninvasive vascular testing**

**Exclusion of autoimmune diseases, hypercoagulable states, and diabetes mellitus by laboratory tests**

**Exclusion of a proximal source of emboli by echocardiography and arteriography**

**Consistent arteriographic** findings in the clinically involved and noninvolved limbs

Most patients (70-80%) with Buerger disease present with distal ischemic rest pain and/or ischemic ulcerations on the toes, feet, or fingers, as depicted in the images below. The feet of a patient with Buerger disease. Note the ischemic ulcers on the distal portion of the left great, second, and fifth toes. Though the patient's right foot is normal in gross appearance, angiography demonstrated compromised arterial flow to both feet.
Superficial thrombophlebitis of the great toe in a patient with Buerger disease.

Progression of the disease may lead to involvement of more proximal arteries, but involvement of large arteries is unusual.

Patients may also present with claudication of the feet, legs, hands, or arms and often describe the Raynaud phenomenon of sensitivity of the hands and fingers to cold.

Foot or arch claudication may be erroneously attributed to an orthopedic problem.

Patients who seek medical attention late in the course of their disease may present with foot infections and, occasionally, with florid sepsis.

Although classic Buerger disease affects the vessels of the extremities, a few cases of aortic, cerebral, coronary, iliac, mesenteric, pulmonary, and renal thromboangiitis obliterans have been reported.

**Physical**

Patients with Buerger disease develop painful ulcerations and/or frank gangrene of the digits.
The hands and feet of patients with the disease are usually cool and mildly edematous.

Superficial thrombophlebitis (often migratory) occurs in almost half of patients with Buerger disease.

Paresthesias (numbness, tingling, burning, hypoesthesia) of the feet and hands and impaired distal pulses in the presence of normal proximal pulses are usually found in patients with the disease.

More than 80% percent of patients present with involvement of 3-4 limbs.

More recently a point-scoring system has been proposed by Papa to support or contest the diagnosis of TAO using the following criteria.

- Distal extremity (feet, toes, hands, fingers) involvement
- Onset before age 45
- Tobacco use
- Exclusion of atherosclerosis or proximal source of emboli
- Lack of hypercoagulable state
- Lack of definable arteritis (ie, progressive systemic sclerosis, giant cell arteritis)

**Classic arteriographic findings**

- Involvement of digital arteries of finger or toes
- Segmental involvement (ie, "skip areas")
- Corkscrew collaterals
- No atherosclerotic changes

**Classic histopathologic findings**

- Inflammatory cellular infiltrate within thrombus
- Intact internal elastic lamina
- Involvement of surrounding venous tissues

**Table 1. Scoring system for the diagnosis of thromboangiitis obliterans**

**Positive points**

<table>
<thead>
<tr>
<th>Positive point</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at onset</td>
<td>Less than 30 (+2)/30-40 years (+1)</td>
</tr>
<tr>
<td>Foot intermittent claudication</td>
<td>Present (+2)/ by history (+1)</td>
</tr>
<tr>
<td>Upper extremity</td>
<td>Symptomatic (+2)/ asymptomatic (+1)</td>
</tr>
<tr>
<td>Migrating superficial vein thrombosis</td>
<td>Present (+2)/ by history only (+1)</td>
</tr>
<tr>
<td>Raynaud</td>
<td>Present (+2)/ by history only (+1)</td>
</tr>
<tr>
<td>Angiography; biopsy</td>
<td>If typical both (+2)/ either(+1)</td>
</tr>
</tbody>
</table>

**Negative points**

<table>
<thead>
<tr>
<th>Negative point</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at onset</td>
<td>45-50 (-1)/more than 50 years (-2)</td>
</tr>
<tr>
<td>Sex, smoking</td>
<td>Female (-1)/ nonsmoker (-2)</td>
</tr>
<tr>
<td>Location</td>
<td>Single limb (-1)/no LE involved (-2)</td>
</tr>
</tbody>
</table>
Absent pulses Brachial (-1)/femoral (-2)

Arteriosclerosis, diabetes, hypertension, hyperlipidemia Discovered after diagnosis

5.1-10 years (-1)/2.1-5 years later (-2)

Table 2. Sum of points defines the probability of the diagnosis of thromboangiitis obliterans

<table>
<thead>
<tr>
<th>Points</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1</td>
<td>Diagnostic excluded</td>
</tr>
<tr>
<td>2-3</td>
<td>Suspected, low probability</td>
</tr>
<tr>
<td>4-5</td>
<td>Probable, medium probability</td>
</tr>
<tr>
<td>6 or more</td>
<td>Definite, high probability</td>
</tr>
</tbody>
</table>

Causes; Propagating agents include cigarettes, as depicted in the image below, chewing tobacco, nicotine patches, and secondhand tobacco smoke (the latter two have been implicated as propagating agents of the disease only in former smokers).
The tobacco smoke–stained fingers of this patient suggested the man's diagnosis (Buerger disease). The patient presented with small, painful ulcers on the tips of his thumb and ring finger.

**Differential Diagnoses**

- Antiphospholipid Antibody Syndrome and Pregnancy
- Peripheral Arterial Occlusive Disease
- Atherosclerosis
- Polyarteritis Nodosa
- Raynaud Phenomenon
- Reflex Sympathetic Dystrophy
- Diabetes Mellitus, Type 1
- Diabetes Mellitus, Type 2
- Frostbite
- Scleroderma
- Giant Cell Arteritis
- Systemic Lupus Erythematosus
- Takayasu Arteritis
- Infrainguinal Occlusive Disease
- Thoracic Outlet Obstruction

**Other Problems to Be Considered**

Acrocyanosis
Carpal tunnel syndrome
Cervical rib
Ergotism
Juvenile temporal arteritis with eosinophilia
Livedo reticularis
Metatarsalgia
Neuropathy, peripheral
Neurotrophic ulcers
Orthopedic problem of the foot or arch
Trauma
Vasculitis, other causes
Calcinosis cutis, Raynaud phenomenon, esophageal motility disorder, sclerodactyly, and telangiectasia (CREST) syndrome
Systemic lupus erythematosus
Rheumatoid vasculitis
Mixed connective-tissue disease
Antiphospholipid-antibody syndrome
Diabetes mellitus
Embolic occlusion of small or medium arteries
Hyperhomocysteinemia with atherosclerosis
Popliteal artery entrapment syndrome
Repetitive vibratory equipment use
Hypothenar hammer syndrome

**Workup Laboratory Studies**

No specific laboratory tests confirm or exclude the diagnosis of Buerger disease. The primary goal of a laboratory workup in patients thought to have the disease is to exclude other disease processes in the differential diagnosis. Tests often used as markers for the diagnosis of systemic vasculitis, such as the acute-phase reactants, are negative in TAO. A complete serologic profile must be obtained.

CBC count with differential ;Liver function tests ;Renal function tests Urinalysis Glucose (fasting) ; Erythrocyte sedimentation rate; C-reactive protein Antinuclear antibody; Rheumatoid factor Complement; Antiphospholipid antibodies ;

**Imaging Studies Angiography/arteriography**

Arteriographic abnormalities consistent with Buerger disease are sometimes seen in limbs that are not yet clinically involved; therefore, arteriography of all 4 limbs may be required.
The hallmark angiographic findings in patients with Buerger disease are nonatherosclerotic, segmental occlusive lesions of the small- and medium-sized vessels (eg, digital, palmar, plantar, tibial, peroneal, radial, and ulnar arteries) with formation of distinctive small-vessel collaterals around areas of occlusion known as "corkscrew collaterals", as depicted in the image below. Such arteriographic findings suggest Buerger disease but are not pathognomonic because similar lesions can be observed in patients with scleroderma, CREST syndrome, systemic lupus erythematosus, rheumatoid vasculitis, mixed connective-tissue disease, antiphospholipid-antibody syndrome, and even diabetes mellitus.

*This lower extremity arteriogram of the peroneal and tibial arteries of a patient with Buerger disease demonstrates the classic findings of multiple small- and medium-sized arterial occlusions with formation of compensatory "corkscrew collaterals."*

Echocardiography: Always perform echocardiography in patients thought to have Buerger disease in order to exclude a proximal source of emboli as the cause of distal vessel occlusion.

**Other Tests**
An abnormal Allen test result indicating distal arterial disease and establishing involvement of the upper extremities in addition to the lower extremities helps differentiate thromboangiitis obliterans from atherosclerotic disease.

To perform the Allen test, the patient is instructed to make a fist, which exsanguinates the hand and fingers. The examiner's thumbs are then used to occlude the radial and ulnar arteries. The patient then opens the hand, after which the examiner releases the pressure on the ulnar artery while the radial artery remains compressed.

Prompt return of color to the hand indicates patency of the ulnar artery (ie, a normal or negative test result). The patency of the radial artery can then be tested by repeating the maneuver but with the pressure on the radial artery released while the ulnar artery remains compressed.

Failure of the hand to promptly refill with blood indicates occlusion of the respective artery distal to the wrist (ie, an abnormal or positive test result). While an abnormal result can be present in other types of small-vessel occlusive disease of the hands, a positive Allen test finding in a young smoker with leg ulcerations is highly suggestive of Buerger disease.

**Histologic Findings**

Olin contends that a biopsy is rarely needed unless the patient presents with unusual characteristics, such as large-artery involvement, or age older than 45 years.

In its acute phase, Buerger disease is characterized by highly cellular, segmental, occlusive, inflammatory thrombi, with minimal inflammation in the walls of affected blood vessels. Secondary spread from the affected small- and medium-sized arteries to contiguous veins and nerves is often observed. Microscopically, the polymorphonuclear leukocyte–predominant inflammatory cellular aggregate may form microabscesses and multinucleated giant cells.

In the subacute phase, intraluminal thrombosis progressively organizes, but it may defer to vascular recanalization.
The end-stage phase of the disease is characterized by mature thrombus and vascular fibrosis.

In all 3 stages, the integrity of the normal structure of the vessel wall, including the internal elastic lamina, is maintained. This distinguishes thromboangiitis obliterans from arteriosclerosis and from other types of systemic vasculitis, in which disruption of the internal elastic lamina and the media can be extensive

**Treatment**

**Medical Care**

Absolute discontinuation of tobacco use is the only strategy proven to prevent the progression of Buerger disease. Smoking as few as 1 or 2 cigarettes daily, using chewing tobacco, or even using nicotine replacements may keep the disease active.

Except for absolute tobacco avoidance, no forms of therapy are definitive. Treatment with intravenous iloprost (a prostaglandin analogue), an expensive therapy unavailable in the United States, has been shown to be somewhat effective in improving symptoms, accelerating resolution of distal extremity trophic changes, and reducing the amputation rate among patients with Buerger disease. Intravenous iloprost use is probably of greatest value in slowing progressive tissue loss and reducing the need for amputation in patients with critical limb ischemia during the period when they first discontinue cigarette smoking.

The use of thrombolytic therapy in the treatment of Buerger disease has been proposed, but the data for this treatment remain inconclusive and the treatment is thus considered experimental. Recently, Isner and colleagues reported improved healing of ischemic ulcers and relief of rest pain in a small series of patients with Buerger disease using intramuscular gene transfer of vascular endothelial growth factor.10

The following strategies are important in prevention of complications from Buerger disease: Use of well-fitting protective footwear to prevent foot trauma and thermal or chemical injury Early and aggressive treatment of extremity injuries to protect against infections Avoidance of cold environments Avoidance of drugs that lead to vasoconstriction
**Surgical Care**

Given the diffuse segmental nature of thromboangiitis obliterans and the fact that the disease primarily affects small- and medium-sized arteries, surgical revascularization for Buerger disease is usually not feasible and is extremely rare in the United States. However, make every effort to improve distal arterial flow in patients with Buerger disease, and consider autologous vein bypass of coexistent large-vessel atherosclerotic stenoses in patients with severe ischemia who have an acceptable distal target vessel.

Other proposed surgical treatments for Buerger disease are as follows:

- Omental transfer
- Sympathectomy
- Spinal cord stimulator implantation

Ultimate surgical therapy for refractory Buerger disease (in patients who continue smoking) is **distal limb amputation for nonhealing ulcers**, gangrene, or intractable pain. Avoid amputation when possible, but, if it is necessary, perform the operation in a way that preserves as much of the limb as possible.

**Consultations**

- Rheumatologists
- Vascular surgeons

**Smoking cessation counselors**

**Diet:** No dietary restrictions are needed. Diet has not been shown to affect the course of the disease.

Activity Encourage cardiovascular exercise. Activity should be restricted by symptoms only.

Medication Other than the experimental use of iloprost and thrombolytics (as discussed previously), the use of antibiotics to treat infected ulcers, and palliative treatment of ischemic pain with nonsteroidal and narcotic analgesics, all other forms of pharmacologic treatment have been generally ineffective in the treatment of Buerger disease, including steroids, calcium channel blockers, reserpine, pentoxifylline, vasodilators, antiplatelet drugs, and anticoagulants.
Follow-up

**Patients must be counseled to never smoke.**

**Complications** Ulcerations Gangrene Infection Need for amputation

Rare occlusion of coronary, renal, splenic, or mesenteric arteries

**Prognosis**

A striking dichotomy is observed with regard to the prognosis of patients with Buerger disease, which is dependent upon whether absolute avoidance of tobacco is achieved. Among patients with who quit smoking, 94% avoid amputation; among patients who quit smoking before gangrene develops, the amputation rate is near 0%. This is in stark contrast to patients who continue smoking, for whom there is a 43% chance that an amputation will be required sometime during a 7- to 8-year period. It is not uncommon for patients with Buerger disease who continue to smoke to require multiple amputations, and reports have even been made of patients who have required bilateral above-knee and above-elbow amputations. While smoking cessation generally removes the need for limb amputation, patients may continue to have claudication or Raynaud phenomenon even after complete discontinuation of tobacco use.

**Patient Education**

Patients with Buerger disease must be repeatedly implored to quit smoking and can be reassured that if they are able to discontinue tobacco use, the disease will remit and amputation will be avoided.

Physicians should counsel patients that the level of tobacco avoidance required to achieve resolution of their disease often necessitates that they even rigorously limit their exposure to secondhand smoke. This can be extremely difficult for patients who live with another smoker, and it is therefore not unreasonable to consider referring such patients (and their loved ones) to multidisciplinary smoking cessation programs.

Patients with Buerger disease who are bedridden should be educated about the importance of protective heel pads or foam boots.
For excellent patient education resources, visit eMedicine's Public Health Center and Lung and Airway Center. Also, see eMedicine's patient education article Cigarette Smoking.

**Medicolegal Pitfalls**

Failure to recognize Buerger disease and to counsel patients about the necessity for smoking cessation to prevent limb loss can lead to medical/legal vulnerability.

**Special Concerns**

Buerger disease does not occur in pediatric or elderly patients. In the rare event that a pregnant woman presents with Buerger disease, the treatment would remain recommendation of absolute smoking cessation.

The opinions or assertions contained herein are those of the authors and should not be construed as official or reflecting the views of the Department of Defense, the United States Air Force, or any other government agency.
Abdominal Aortic Aneurysm

Introduction

Abdominal aortic aneurysms (AAAs) represent a degenerative process of the abdominal aorta that is often attributed to atherosclerosis; however, the exact cause is not known. A familiar clustering of AAAs has been noted in 15-25% of patients undergoing repair of the problem. Degenerative aneurysms account for more than 90% of all infrarenal AAAs. Other causes include infection, cystic medial necrosis, arteritis, trauma, inherited connective-tissue disorders, and anastomotic disruption.

The disease generally affects elderly white men. Smoking appears to be the risk factor most strongly associated with AAA.

History of the Procedure

Vesalius described the first abdominal aortic aneurysm (AAA) in the 16th century. Before the development of a surgical intervention for the process, attempts at medical management failed. The initial attempts at control used ligation of the aorta, with the expected consequences.

In 1923, Matas performed the first successful aortic ligation on a patient. Attempts were made to induce thrombosis by inserting intraluminal wires. In 1948, Rea wrapped reactive cellophane around the aneurysm in order to induce fibrosis and limit expansion. This technique was used on Albert Einstein in 1949, and he survived 6 years before succumbing to rupture. In 1951, Charles Dubost performed the first AAA repair using a homograft.

Prior to this, aortic aneurysms were treated using a variety of methods, including ligation, intraluminal wiring, and cellophane wrapping. Unfortunately, early homografts became aneurysmal because of preservation techniques. In 1953, Blakemore and Voorhees repaired a ruptured AAA using a Vinyon-N graft (ie, nylon). Later, these grafts were replaced by Dacron and Gore-Tex (ie,
polytetrafluoroethylene [PTFE]) fabrics. The final advance was abandonment of silk sutures, which degenerated, in favor of braided Dacron, polyethylene, and PTFE (ie, Gore-Tex) sutures, all of which retain tensile strength.

Postoperative surgical mortality rates initially remained high (>25%) because the aneurysm sac generally was excised. Nearly simultaneously in 1962, Javid and Creech reported the technique of endoaneurysmorrhaphy (see Image 1). This advancement dramatically reduced mortality. Today, operative mortality rates range from 1.8-5%.

Endoaneurysmorrhaphy.

In the late 1980s, Parodi et al described endovascular repair using a large Palmaz stent and unilateral aortofemoral and femorofemoral crossover Dacron grafts.1 Currently, many devices are used for the endovascular treatment of AAA (see Image 2).
Endovascular grafts.

Aneurysms are defined as a focal dilatation with at least a 50% increase over normal arterial diameter. Thus, an enlargement of at least 3 cm of the abdominal aorta fits the definition. In large ultrasound screening studies, increasing age; male sex; African American race; and increased height, weight, body mass index, and body surface area all were associated with increased infrarenal aortic diameter.

Most cases of abdominal aortic aneurysm (AAA) begin below the renal arteries and end above the iliac arteries. They generally are spindle shaped; however, size, shape, and extent vary considerably. Of AAA cases, 10-20% have focal outpouchings or blebs that are thought to contribute to the potential for rupture. The wall of the aneurysm becomes laminated with thrombus as the blebs enlarge. This can give the appearance of a relatively normal intraluminal diameter in spite of a large extraluminal size.

**Frequency**

Abdominal aortic aneurysms (AAAs) are uncommon in African Americans, Asians, and persons of Hispanic heritage.
In autopsy studies, the frequency rate of AAA ranges from 0.5-3.2%. In a large US Veterans Administration screening study, the prevalence rate was 1.4%. The frequency of rupture is 4.4 cases per 100,000 persons.

AAA is 5 times more common in men than in women and is 3.5 times more common in white males than in African American males. The likelihood of development varies from 3-117 cases per 100,000 person-years. In men, the process appears to begin at approximately age 50 years and reaches peak incidence at approximately age 80 years. In women, the onset is delayed and appears to begin at approximately age 60 years. The reported incidence of rupture varies from 1-21 cases per 100,000 person-years.

**International**

The frequency rate of asymptomatic AAA is 8.2% in the United Kingdom, 8.8% in Italy, 4.2% in Denmark, and 8.5% in Sweden (in males only). The frequency rate of AAA in females is much lower, 0.6-1.4%. The frequency of rupture is 6.9 cases per 100,000 persons in Sweden, 4.8 cases per 100,000 persons in Finland, and 13 cases per 100,000 persons in the United Kingdom.

**Risk factors**;

The peak incidence of AAA occurs in people aged 70 years (see Image 3). The male-to-female incidence ratio in people younger than 80 years is 2:1. When older than 80 years, the ratio changes to 1:1.
Age is a risk factor for development of an aneurysm.

A family history of AAA is a risk factor for AAA. Approximately 25% of cases are in persons with first-degree relatives with AAA. Other risk factors include previous aneurysm repair or peripheral aneurysm (popliteal or femoral), smoking, coronary artery disease, and hypertension (1-15%).

Etiology

AAA is thought to be a degenerative process of the aorta, the cause of which remains unclear. It is often attributed to atherosclerosis because these changes are observed in the aneurysm at the time of surgery. Atherosclerosis fails to explain the development of occlusion, which is observed in the disease process.

AAA appears to have a familial prevalence rate of 15-25%. Studies by Majumder and associates suggest the genetic predisposition is isolated to a single dominant gene with low penetrance that increases with age.3 Tilson et al described the potential for an autoimmune basis for the development of AAA involving the DRB1 major histocompatibility locus.4 This locus has been identified as a basis for inflammatory AAA.

The other causes of AAA include infection, cystic medial necrosis, arteritis, trauma, inherited connective-tissue (structural collagen) disorders, and anastomotic disruption producing pseudoaneurysms.
Pathophysiology

A multidisciplinary research program supported by the US National Heart, Lung, and Blood Institute identified proteolytic degradation of aortic wall connective tissue, inflammation and immune responses, biomechanical wall stress, and molecular genetics as mechanisms important in the development of abdominal aortic aneurysm (AAA). The aortic wall contains smooth muscle, elastin, and collagen arranged in concentric layers in order to withstand arterial pressure. The number of medial elastin layers from the proximal thoracic aorta to the infrarenal aorta is markedly reduced, with medial thinning and intimal thickening. A reduction in collagen and elastin content is noted from the proximal to the distal aorta. Elastin is the principal load-bearing element in the aorta. Elastin fragmentation and degeneration are observed in aneurysm walls. The decrease in content coupled with the histological changes of this matrix protein in aneurysms may explain the propensity for aneurysm formation in the infrarenal aorta.

Immunoreactive proteins are found more conspicuously in the abdominal aorta, and this may contribute to the increased frequency of aneurysms in this location.

The aortic media appear to degrade in AAA by means of a proteolytic process. This implies an increase in the concentration of proteolytic enzymes relative to their inhibitors in the abdominal aorta as the individual ages. Reports have documented increased expression and activity of matrix metalloproteinases (MMPs) in persons with AAAs. MMPs and other proteases have been shown to be secreted into the extracellular matrix of AAAs by macrophages and aortic smooth muscle cells. MMPs and their inhibitors are present in normal aortic tissue and are responsible for vessel wall remodeling. In aneurysmal tissue, a tendency exists for increased MMP activity favoring the degradation of elastin and collagen. The mechanism that tips the balance in favor of degradation of elastin and collagen in the aortic wall of AAAs by MMPs and other proteases is presently unknown.

AAAs demonstrate a chronic adventitial and medial inflammatory infiltrate upon histological examination. Infiltration of AAAs with lymphocytes and macrophages may trigger protease activation via various cytokines (interleukin [IL]−1, IL-6, IL-8, and tumor necrosis factor-alpha). Further study has defined a matrix protein that is immunoreactive with immunoglobulin G in the aneurysm wall. This autoantigen
appears to be a collagen-associated microfibril. Certain infectious agents have been associated with the development of this protein, including Chlamydia pneumoniae and Treponema pallidum; however, a direct cause-and-effect relationship has not been demonstrated.

AAAs arise as a result of a failure of the major structural proteins of the aorta (elastin and collagen). The inciting factors are not known, but a genetic predisposition clearly exists. Surgical specimens of AAA reveal inflammation, with infiltration by lymphocytes and macrophages; thinning of the media; and marked loss of elastin (see Image 4). Recent research has focused on the role of the metalloproteinases, a group of zinc-dependent enzymes responsible for tissue remodeling.

In general, AAAs gradually enlarge (0.2-0.8 mm/y) and eventually rupture. Hemodynamics play an important role. Areas of high stress have been found in AAAs and appear to correlate with the site of rupture. Computer-generated geometric factors have demonstrated that aneurysm volume is a better predictor of areas of peak wall stress than aneurysm diameter. This may have implications in determining which AAAs require surgical repair.

Additionally, molecular genetics has provided some insight into the development of AAAs. Through gene microarray analysis, various genes involved in extracellular matrix degradation, inflammation, and other processes observed in AAA formation have been shown to be up-regulated, while others that may serve to prevent this occurrence are down-regulated. The combination of proteolytic degradation of aortic wall connective tissue, inflammation and immune responses, biomechanical wall stress, and molecular genetics represents a dynamic process that leads to aneurysmal deterioration of aortic tissue.
Presentation

Asymptomatic: Most patients present without an asymptomatic pulsatile abdominal mass (see Image 5). The aortic bifurcation is located just above the umbilicus. Occasionally, an overlying mass (pancreas or stomach) may be mistaken for an AAA. An abdominal bruit is nonspecific for a nonruptured aneurysm. Patients with popliteal artery aneurysms frequently have AAAs (25-50%).

Pulsatile abdominal mass.

Rupture: Persons with AAAs that have ruptured may present in many ways. The most typical manifestation of rupture is abdominal or back pain with a pulsatile abdominal mass. However, the symptoms may be vague, and the abdominal mass may be missed. Symptoms may include groin pain, syncope, paralysis, or flank mass. The diagnosis may be confused with renal calculus, diverticulitis, incarcerated hernia, or lumbar spine disease.

Peripheral emboli: Atheroemboli from small AAAs produce livedo reticularis of the feet or blue toe syndrome (see Image 6).

Atheroemboli from small abdominal aortic aneurysms produce livedo reticularis of the feet (ie, blue toe syndrome).
Acute aortic occlusion: Occasionally, small AAAs thrombose, producing acute claudication.

Aortocaval fistulae: AAAs may rupture into the vena cava, producing large arteriovenous fistulae. In this case, symptoms include tachycardia, congestive heart failure (CHF), leg swelling, abdominal thrill, machinery-type abdominal bruit, renal failure, and peripheral ischemia.

Aortoduodenal fistulae: Finally, an AAA may rupture into the fourth portion of the duodenum. These patients may present with a herald upper gastrointestinal bleed followed by an exsanguinating hemorrhage.

Physical examination

Bilateral upper extremity blood pressures are discernible in patients with AAAs. Hypertension may trigger a workup for renal artery stenosis. Unequal blood pressures (>30 mm Hg) indicate subclavian artery stenosis, and perioperative monitoring is important.

Cervical bruits may indicate carotid artery stenosis. Abdominal examination includes palpation of the aorta and an estimation of the size of the aneurysm. Bruits may indicate the presence of renal or visceral artery stenosis; a thrill is possible with aortocaval fistulae.

Regarding the peripheral pulses, palpate femoral popliteal and pedal pulses (dorsalis pedis or posterior tibial) to determine if an associated aneurysm (femoral/popliteal) or occlusive disease exists. Flank ecchymosis (Grey Turner sign) represents retroperitoneal hemorrhage.

With respect to rectal aspects of the physical examination, guaiac-positive stool is present with associated colon cancer.

Most persons with AAAs are asymptomatic. Patients may describe a pulse in the abdomen and may actually feel a pulsatile mass. At times, AAAs may cause symptoms from local compression, including early satiety, nausea, vomiting, urinary symptoms, or venous thrombosis from venous compression. Back pain can be caused by erosion of the AAA into adjacent vertebrae. Other symptoms include abdominal pain, groin pain, embolic phenomenon to the toes, and fever. Transient
hypotension should prompt consideration of rupture because this finding can progress to frank shock over a period of hours. Temporary loss of consciousness is also a potential symptom of rupture.

Most clinically significant aneurysms are palpable upon routine physical examination; however, the sensitivity of the technique is based on the experience of the examiner, the size of the aneurysm, and the size of the patient. In a recent study, 38% of AAA cases were detected based on physical examination findings, while 62% were detected incidentally based on radiologic studies obtained for other reasons.

**Indications**

Even patients who do not have symptoms from their abdominal aortic aneurysms (AAAs) require surgical intervention because the result of medical management in this population is a mortality rate of 100% over time due to rupture. In addition, these patients have a high likelihood of limb loss from peripheral embolization.

Monitor patients with AAAs smaller than 4 cm in diameter with ultrasound every 6 months, and offer surgical intervention if the aneurysm expands or causes symptoms. In patients with AAAs of 4-5 cm in diameter, elective repair may be of benefit if they are young, have a low operative risk, and have a good life expectancy. Additionally, AAAs in women have been shown to rupture at smaller diameters in comparison with men; therefore, a threshold of 4.5 cm for elective repair has been advocated in this patient population. Patients with AAAs of 5-6 cm in diameter may benefit from repair, especially if they have other contributing factors for rupture, including hypertension, continued smoking, or chronic obstructive pulmonary disease (COPD). For patients at higher risk, the threshold for repair may be a diameter of 6-7 cm, depending on their condition. At this size, the risk of rupture increases with age. These sizes apply to males of average height (170 cm).

**Relevant Anatomy**

The abdominal aorta maintains 3 distinct tissue layers, an intima, media, and adventitia. The intima is composed of the classic endothelial layer. The media contains vascular smooth muscle and matrix proteins, elastin, and collagen. The
diameter of the aorta decreases in size from its thoracic portion to the abdominal and infrarenal portions. A normal aorta shows a reduction in medial elastin layers from the thoracic area to the abdominal portion. Elastin and collagen content are also reduced.

Aneurysms represent a dilatation in all layers of the vessel wall. The shape of the aneurysm can be described as saccular or fusiform, although this description represents a continuum. Aneurysm diameter is an important risk factor for rupture.

The important surgical and endovascular anatomic considerations include associated renal and visceral artery involvement (either occlusive disease or involved in the aneurysm process) and the iliac artery (either occlusive disease or aneurysms). The length of the infrarenal aortic neck is important in helping determine the surgical approach (retroperitoneal vs transabdominal) and the location of the aortic cross clamp. Hypogastric artery (internal iliac) outflow is important in planning surgical repair. Loss of blood flow from the hypogastric artery may result in impotence in males and sigmoid colon ischemia with necrosis.

**Inflammatory aneurysms** represent a subsegment of AAA and are characterized by a thick inflammatory peal. These aneurysms are associated with retroperitoneal fibrosis and adhesion of the duodenum and fibrosis (see Image 7).
Contraindications

Contraindications for operative intervention of abdominal aortic aneurysms (AAAs) include severe COPD, severe cardiac disease, active infection, and medical problems that preclude operative intervention. These patients may benefit best from endovascular stenting of the aneurysm.

Severe life-threatening comorbidities include advanced cancer, end-stage lung disease, or cardiac disease. In many patients, the decision to operate is a balance between risks and benefits. In an elderly patient (>80 y) with significant comorbidities, surgical repair may not be indicated. The decision to intervene should not be based on age alone, even with rupture. The decision is best based on the patient's overall physical status, including a positive attitude toward the surgery.

Patients with known cancer with an indolent course (ie, prostate cancer) may merit aneurysm repair if their estimated survival is 2 years or longer.

Laboratory Studies

CBC count with differential: This study is used to assess transfusion requirements and the possibility of infection.

Blood chemistries (including a renal and liver panel): Ascertain the integrity of renal and hepatic function to best manage the patient postoperatively and to assess operative risk.

Type and crossmatch blood: Prepare for the possibility of transfusion, including clotting factors and platelets.

Urinalysis: Because synthetic material is used in the intervention, assess and eliminate potential foci of infection preoperatively.

Arterial blood gases: Assess pulmonary function preoperatively in order to determine operative risk and postoperative care. Patients who can climb a flight of
stairs without excessive shortness of breath generally do well. If in doubt about the patient's pulmonary status, blood gas tests and pulmonary function tests are helpful.

**Imaging Studies**

**Chest radiography**: This study is used to gain a preliminary assessment of the status of the heart and lungs. Concurrent pulmonary or cardiac disease may need to be addressed prior to treating the aneurysm.

**Abdominal ultrasonography**: This study is used as a preliminary determination of aneurysm presence, size, and extent. It is a cost-effective modality for monitoring patients whose aneurysms are too small for surgical intervention.

**CT scanning**: This study helps more clearly define the anatomy of the aneurysm and other intra-abdominal pathologies.

Although sizing the aneurysm is important, the anatomic relationships important to surgery are also determined, ie, location of the renal arteries, length of the aortic neck, condition of the iliac arteries, and anatomic variants such as a retroaortic left renal vein or horseshoe kidney.

**Enhanced spiral CT** scanning of the abdomen and pelvis with multiplanar reconstruction and CT angiography is the test of choice for preoperative evaluation for open and endovascular repair (see Image 8). Nonenhanced CT scanning is used to size aneurysms.
Enhanced spiral CT scans with multiplanar reconstruction and a CT angiogram.

**Magnetic resonance angiography:** This imaging modality is quickly replacing the traditional angiographic assessment of aneurysms. The study provides excellent anatomical definition and 3-dimensional assessment of the problem. Gadolinium-enhanced magnetic resonance angiography can provide excellent images, even though regional variations in quality are reported.

**Angiography:** This imaging modality remains the criterion standard for the diagnosis of AAA, and it is indicated in the presence of associated renal or visceral involvement, peripheral occlusive disease, or aneurysmal disease. Angiography is also essential with any renal abnormality (eg, horseshoe kidney, pelvic kidney). See Image 9.
Angiography is used to diagnose the renal area. In this instance, an endoleak represented continued pressurization of the sac.

**Echocardiography:** Because of the fluid shift involved during the operative repair of AAA, cardiac function should be assessed using echocardiography. By ascertaining the ejection fraction of the patient, the operative intervention can be planned and cardiac protective measures can be instituted as needed. This study is particularly indicated in patients with a history of CHF or known cardiac enlargement.
Other Tests

Pulmonary function tests: Assessment of pulmonary function is of paramount importance in these patients. Because surgical intervention requires an abdominal incision, preoperative assessment of the patient's pulmonary status allows for tailored postoperative care.

Electrocardiography: Assess cardiac status in all patients with vascular disease. If one vascular bed is involved with an atherosclerotic process, then consider that others also may be involved. Electrocardiography findings provide a baseline assessment of cardiac rhythm and old disease processes.

Stress test: A stress test can be performed to uncover unsuspected cardiac ischemia. Significant coronary disease may need to be addressed before the AAA can be repaired.

Histologic Findings

Abdominal aortic aneurysms (AAAs) contain a chronic inflammatory infiltrate and neovascularity of varying degrees. Inflammatory AAAs may contain germinal centers.

Treatment

Medical Therapy

In patients with small abdominal aortic aneurysms (AAAs), attempt to reduce the expansion rate and rupture risk. Smoking cessation is of paramount importance. Aggressively control hypertension. Institute beta-blocker therapy to reduce blood pressure and stress on the artery wall. These can be administered safely unless the patient has contraindications to their use, such as COPD, allergy to the drug, bradycardia, or severe CHF.
Surgical Therapy

The decision to treat an abdominal aortic aneurysm (AAA) is based on operative risk, the risk of rupture, and the patient’s estimated life expectancy. In 2003, the Society for Vascular Surgery (SVS) published a series of guidelines for the treatment of AAAs based on these principles. The operative risk is based on patients’ comorbidities and hospital factors. Patient characteristics, including age, gender, renal function, and cardiopulmonary disease are perhaps the most important (see Table 1).

Table 1. Operative Mortality Risk of Open AAA Repair

<table>
<thead>
<tr>
<th>AAA diameter (cm)</th>
<th>Rupture risk (%/y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;4</td>
<td>0</td>
</tr>
<tr>
<td>4-5</td>
<td>0.5-5</td>
</tr>
<tr>
<td>5-6</td>
<td>3-15</td>
</tr>
<tr>
<td>6-7</td>
<td>10-20</td>
</tr>
<tr>
<td>7-8</td>
<td>20-40</td>
</tr>
<tr>
<td>&gt;8</td>
<td>30-50</td>
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Furthermore, lower volume hospitals and surgeons are associated with higher mortality. The risk of rupture is generally related to size (see Table 2), but other patient variables are important.
Research also suggests that the aneurysm’s morphology may increase the risk of rupture. Prospective studies have concluded that following aneurysms larger than 5.5 cm with serial ultrasounds or CT scans is safe. A slightly higher rupture rate in women exists, and this threshold may be lower. Thus, the decision to repair an AAA is a complex one in which the patient must play an important role. In some very elderly patients or patients with limited life expectancy, aneurysm repair may not be appropriate. In these patients, the consequences of rupture should be frankly discussed. If rupture occurs, no intervention should be performed.

Abdominal aortic aneurysms are typically repaired by an operative intervention. The procedure can be approached through the traditional open laparotomy approach or, now, by newer minimally invasive methodologies or by the placement of endovascular stents.

Preoperative Details

Preoperatively, obtain a careful history and perform a physical examination and laboratory assessment. From the information derived from these basic assessments, perioperative risk and life expectancy after the proposed procedure can be estimated.

Carefully consider whether the patient's current quality of life is sufficient to justify the operative intervention. Because the disease process affects elderly persons who may be debilitated or may have mental deterioration, this decision is made in conjunction with the patient and family.

Once the decision is made, identify comorbidities and risk factors that increase the operative risk or decrease survival. Ascertain the patient's activity level, stamina, and stability of health. Perform a thorough cardiac assessment tailored in accordance with the patient's history, symptomatology, and results from preliminary screening tests such as the electrocardiogram and stress test.

Because COPD is an independent predictor of operative mortality, assess lung function by performing a room-air arterial blood gas measurement and pulmonary function tests. In patients with abnormal test results, preoperative intervention in
the form of bronchodilators and pulmonary toilet often can reduce operative risks and postoperative complications.

**Preoperative intravenous antibiotics** (usually a cephalosporin) are administered to reduce the risk of infection. Arranging for appropriate intravenous accesses to accommodate blood loss, arterial pressure monitoring through an arterial line, and Foley catheter placement to monitor urine output are routine preparations for surgery. For patients at high risk because of cardiac compromise, a Swan-Ganz catheter is placed to assist with cardiac monitoring and volume assessment. Transesophageal echocardiography can be useful to monitor ventricular volume and cardiac wall motion and to provide a guide with respect to fluid replacement and pressor use.

**Prepare for blood replacement.** The patient should have blood available for transfusion. Intraoperative Cell Saver use and preoperative autologous blood donation have become popular.

Maintain a normal body temperature during the operative intervention to prevent coagulopathy and maintain normal metabolic function. To prevent hypothermia, place a recirculating, warm forced-air blanket on the patient and warm any intravenous fluids and blood before administration.

**Approach**

The aorta may be approached either transabdominally or through the retroperitoneal space. Approach juxtarenal and suprarenal aortic aneurysms from the left retroperitoneal space.

Self-retaining retractors are used. Keep the bowel warm and, if possible, not exteriorized. The abdomen is explored for abnormalities (eg, gallstones, associated intestinal or pancreatic malignancy).

Depending on the patient's anatomy, the aorta can be reconstructed with a tube graft, an aortic iliac bifurcation graft, or an aortofemoral bypass.
For proximal infrarenal control, first identify the left renal vein. Occasionally, patients may have a retroaortic vein (<5%). In this situation, take care when placing the proximal clamp. Division of the left renal vein is usually required to clamp above the renal arteries. Regarding pelvic outflow, in most instances, the inferior mesenteric artery is sacrificed. Therefore, to prevent colon ischemia, make every attempt to restore at least one hypogastric (internal iliac) artery perfusion. If the hypogastric arteries are sacrificed (associated aneurysms), reimplant the inferior mesenteric artery. For supraceliac aortic control, first divide the ligaments to the left lateral segment of the liver and then retract the segment. The crura of the diaphragm are separated, and the aorta is bluntly dissected. Supraceliac control is recommended for inflammatory aneurysms. The aorta is reconstructed from within using PTFE or Dacron. The aneurysm sac is closed, and the graft is put into the duodenum to prevent erosion.

**Special considerations**

Inflammatory aneurysms require supraceliac control, minimal dissection of the duodenum, and balloon occlusion of the iliac arteries.

In patients with inflammatory aneurysms or large iliac artery aneurysms, identify the ureters; occasionally, ureteral stents are recommended in patients with inflammatory aneurysms.

**Prevention of distal embolization**

The patient is heparinized (5000 U intravenously) prior to aortic cross-clamping. If significant intraluminal debris, juxtarenal thrombus, or prior peripheral embolization is present, the distal arteries are clamped first, followed by aortic clamping. Before restoring lower extremity blood flow, both forward flow (aortic) and back flow (iliac) are allowed to remove debris. The graft is also irrigated to flush out debris. The colon is inspected prior to closure, and the femoral arteries are palpated. Before the patient leaves the operating room, determine lower extremity circulation. If a clot was dislodged at the time of aortic clamping, it can be removed with a Fogarty embolectomy catheter. Heparin reversal is not usually required.
Aortic Stent Grafts

Renal Arteries

Type I

Type II

Type II

Type IV

Type III
Varicose Veins

Introduction

History of the Procedure

The description of varicose veins as a clinical entity can be traced back as early as the fifth century BC. Forefathers of medicine including Hippocrates and Galen described the disease and treatment modalities, which are still used today.1 Throughout the centuries, surgical treatments have evolved from large, open surgeries to minimally invasive approaches.

Problem

Varicose veins represent a significant clinical problem and are not just a “cosmetic” issue because of their unsightly nature. The problem arises from the fact that varicose veins actually represent underlying chronic venous insufficiency with ensuing venous hypertension. This venous hypertension leads to a broad spectrum of clinical manifestations, ranging from symptoms to cutaneous findings like varicose veins, reticular veins, telangiectasias, swelling, skin discoloration, and ulcerations, as depicted in the image below.
like varicose veins, reticular veins, telangiectasias, swelling, skin discoloration, and ulcerations, as depicted in the image below.

Pathway leading to varicose veins and other clinical manifestations of venous hypertension.
Varicose veins and even chronic venous insufficiency can be managed conservatively with stockings and compression. More aggressive management can be pursued for cosmesis, worsening cutaneous findings or symptoms despite conservative management, or if the patients prefer surgical management. Most procedures to treat varicose veins can be elective, and emergent treatment and workup is usually reserved for bleeding varicosities or if deep venous thrombosis is suspected.

Frequency

The incidence and prevalence of varicose veins has been studied in a number of cross-sectional studies. In 1973, the United States Tecumseh community health study estimated that about 40 million persons (26 million females) in the US were affected. In 1994, a review by Callam found half of the adult population have minor stigmata of venous disease (women 50-55%; men 40-50%) and fewer than half have visible varicose veins (women 20-25%; men 10-15%). In 2004, these finding were also seen in a French cross-sectional study that found the odds ratio per year for varicose veins were 1.04 for women and 1.05 for men. Age and gender have been the only consistently identified risk factors for varicose veins.

Etiology

The cause of primary varicose veins is incompetent venous valves that result in venous hypertension. Secondary varicose veins result from deep venous thrombosis and its sequelae or congenital anatomic abnormalities. The etiology of these varicose veins can be classified into the following three groups:

Primary: Valvular insufficiency of the superficial veins, most commonly at the saphenofemoral junction.

Secondary Mainly caused by deep vein thrombosis (DVT) that leads to chronic deep venous obstruction or valvular insufficiency. Long-term clinical sequelae from this have been called the postthrombotic syndrome. Catheter-associated DVTs are also included. Pregnancy-induced and progesterone-induced venous wall and valve weakness worsened by expanded circulating blood volume and enlarged uterus compresses the inferior vena cava and venous return from the lower extremities.
Trauma

**Congenital**: This includes any venous malformations. A few examples are listed as follows: Klippel-Trenaunay variants; Avalvulia

**Pathophysiology**

Varicose veins are simply dilated, tortuous veins of the subcutaneous/superficial venous system. However, the pathophysiology behind their formation is complicated and involves the concept of ambulatory venous hypertension. To understand this, the anatomy of the lower extremity venous system must be briefly discussed. Two venous systems are found in the lower extremity, the deep and superficial, as depicted in the image below. The deep system ultimately leads back to the inferior vena cava, then to the heart. The superficial system is found above the deep fascia of the lower extremity, within the subcutaneous tissue. Many superficial veins exist, but they all drain into the 2 largest, the greater saphenous vein (GSV) and the short saphenous vein (SSV), formerly called the lesser saphenous vein.

*Schematic diagram of the deep and superficial venous systems of the lower extremity: (1) Normal venous drainage; arrows depict the flow of venous blood. (2) Venous hypertension: bold arrows are pathways of venous reflux.*
The superficial venous system is connected to the deep system at a number of the following locations:

Perforator veins: These veins transverse the deep fascia of the lower extremity. A number of named perforators are found at the thigh, knee, and leg. See the Anatomy section for more details, as depicted in the image below.

**Named perforators along the greater saphenous distribution.**
Saphenofemoral junction (SFJ): This is located proximally at the groin where the GSV meets the femoral vein, as depicted in the images below.

Major tributaries of the greater saphenous system.
Saphenofemoral junction.

Saphenopopliteal junction (SPJ): This is located behind the knee where the SSV joins with the popliteal vein, as depicted in the image below.
Major tributaries of the greater saphenous system.

In healthy veins, the flow of venous blood is through the superficial system into the deep and up the leg and toward the heart. One-way venous valves are found in both systems and the perforating veins. Incompetence in any of these valves can lead to a disruption in the unidirectional flow of blood toward the heart and result in ambulatory venous hypertension. Furthermore, incompetence in one system can often lead to incompetence in another. In a study by Shami et al, the limbs of 59 patients with venous ulceration were assessed by color duplex ultrasound scanning. In 53% of patients only superficial venous reflux was found, in 15% isolated deep venous reflux was found, and in 32% a combination of deep and superficial venous reflux was found.
Schematic diagram of the deep and superficial venous systems of the lower extremity: (1) Normal venous drainage; arrows depict the flow of venous blood. (2) Venous hypertension bold arrows are pathways of venous reflux.

Incompetence in the superficial venous system alone usually results from failure at valves located at the SFJ and SPJ. The gravitational weight of the column of blood along the length of the vein creates hydrostatic pressure, which is worse at the more distal aspect of the length of vein, as depicted in circle A of the image above.6
Incompetence of the perforating veins leads to hydrodynamic pressure. The calf pump mechanism helps to empty the deep venous system, but if perforating vein valves fail, then the pressure generated in the deep venous system by the calf pump mechanism are transmitted into the superficial system via the incompetent perforating veins, as depicted in the image below.

**Schematic diagram of the deep and superficial venous systems of the lower extremity:** (1) Normal venous drainage; arrows depict the flow of venous blood. (2) Venous hypertension bold arrows are pathways of venous reflux.

Once venous hypertension is present, the venous dysfunction continues to worsen through a vicious cycle. Pooled blood and venous hypertension leads to venous dilatation, which then causes greater valvular insufficiency. Over time, with more local dilatation, other adjacent valves sequentially fail, and after a series of valves
has failed, the entire superficial venous system is incompetent. As mentioned before, this can then cause subsequent perforator and deep venous valvular dysfunction, as depicted in the image below. Pathway leading to varicose veins and other clinical manifestations of venous hypertension. The inciting etiology of superficial valvular insufficiency is often difficult to determine because the clinical manifestations of venous hypertension are delayed. The original cause can be classified as primary, secondary, and congenital as previously described.

The clinical finding of varicose veins, reticular veins, and telangiectasias are due to the hypertension in the superficial venous system that spreads to collateral veins and tributary veins, causing dilated tortuous structures. Treatment modalities are geared towards correcting the superficial venous hypertension. At times, the degree or venous hypertension does not correlate to the clinical findings. The presence and size of visible varicosities are not reliable indicators of the volume or pressure of venous reflux. A vein that is confined within fascial planes or is buried beneath subcutaneous tissue can carry massive amounts of high-pressure reflux without being visible at all. Conversely, even a small increase in pressure can eventually produce massive dilatation of an otherwise normal superficial vein that carries very little flow. In contrast to the superficial veins, the deep veins do not become excessively distended. They can withstand the increased pressure because of their construction and the confining fascia.

**Presentation**

**Subjective symptoms**

Patients may have a host of symptoms, but they are usually caused by venous hypertension rather than the varicose veins themselves. Often, symptoms are purely aesthetic, and patients desire treatment of the unsightly nature of the tortuous, dilated varicosities. Complaints of pain, soreness, burning, aching, throbbing, heavy legs, cramping, muscle fatigue, pruritus, night cramps, and "restless legs" are usually secondary to the venous hypertension. Pain and other symptoms may worsen with the menstrual cycle, with pregnancy, and in response to exogenous hormonal therapy (eg, oral contraceptives).
Also, pain associated with venous hypertension is usually a dull ache that worsens after prolonged standing, and improves by walking or by elevating the legs. This is in contrast to the pain of arterial insufficiency, which is worse with ambulation and elevation. Subjective symptoms are usually more severe early in the progression of disease, less severe in the middle phases, and more severe again with advancing age. Patients who have become acclimatized to their chronic disease may not volunteer information about symptoms. After treatment, patients are often surprised to realize how much chronic discomfort they had accepted as "normal."

**Venous history**

The venous history should also include the following elements:

- History of venous insufficiency (e.g., date of onset of visible abnormal vessels, date of onset of any symptoms, any known prior venous diagnoses, any history of pregnancy-related varices)
- Presence or absence of predisposing factors (e.g., heredity, trauma to the legs, occupational prolonged standing, sports participation)
- History of edema (e.g., date of onset, predisposing factors, site, intensity, hardness, modification after a night's rest)
- History of any prior evaluation of or treatment for venous disease (e.g., medications, injections, surgery, compression)
- History of superficial or deep thrombophlebitis (e.g., date of onset, site, predisposing factors, sequelae)
- History of any other vascular disease (e.g., peripheral arterial disease, coronary artery disease, lymphedema, lymphangitis)
- Family history of vascular disease of any type

**Physical examination findings**

The physical examination of the venous system is fraught with difficulty. As mentioned earlier, the severity of symptoms does not necessarily correlate with the size or extent of visible varices or with the volume of reflux. Furthermore, most of
the deep venous system cannot be directly inspected, palpated, auscultated, or percussed. In most areas of the body, examination of the superficial venous system must serve as an indirect guide to the deep system.

**Inspection:** Inspection should be performed in an organized manner, usually progressing from distal to proximal and from front to back. The perineal region, pubic region, and abdominal wall must also be inspected. The following items should be noted:

Surgical scars from prior intervention

Pigmentations and skin changes (ie, brownish darkening of the skin, resulting from extravasated blood that causes lipodermatosclerosis. This usually occurs in medial ankle region but may extend to leg and foot.)

*Varicose veins* – *Visible, palpable veins in the subcutaneous skin greater than 3 mm, as depicted in the image below.*

Varicose veins.
**Reticular veins** (also called blue veins, subdermal varices, and venulectasias) – Visible, dilated bluish subdermal, nonpalpable veins 1-3 mm, as depicted in the image below.

**Telangiectases** (also called spider veins, hyphen webs, and thread veins) – Dilated intradermal venules greater than 1 mm in diameter, as depicted in the image below.

**Eczema** – Erythematous dermatitis, which may progress to blistering, weeping, or scaling eruption of the skin of the leg.

**Atrophie blanche** (white atrophy) – Localized, often circular whitish and atrophic skin areas surrounded by dilated capillaries and sometimes hyperpigmentation. (Scars of healed ulceration are excluded from this definition.)

**Corona phlebectatica** (also called malleolar flare and ankle flare) – Fan-shaped pattern of numerous small intradermal veins on the medial or lateral aspects of the ankle and foot.
Ulcers of the medial ankle – Most likely the result of underlying venous insufficiency. (Skin changes or ulcerations that are localized only to the lateral aspect of the ankle are more likely to be related to prior trauma or to arterial insufficiency than to pure venous insufficiency, as depicted in the images below.

*Lipodermatosclerosis.*
Venous stasis ulcer.

Palpation; The entire surface of the skin is palpated lightly with the fingertips because dilated veins may be palpable even where they are not visible. Distal and proximal arterial pulses are also palpated. An ankle-brachial index is useful if arterial insufficiency is suggested.

The anteromedial surface of the lower limb is the territory of the greater saphenous vein (GSV). The arch of the vein may be palpated in some patients with healthy veins, but this segment of the vein is particularly well appreciated in patients with truncal reflux at the saphenofemoral junction (SFJ). It is best palpated 2 fingerbreadths below the inguinal ligament and just medial to the femoral artery. If reflux is present, a forced coughing maneuver may produce a palpable thrill or sudden expansion at this level.

The posterior surface of the calf is the territory of the short saphenous vein. This may be palpable in the popliteal fossa in some slender patients. Normal superficial veins above the foot are usually not palpable even after prolonged standing.
Palpation of an area of leg pain or tenderness may reveal a firm, thickened, thrombosed vein. These palpable thrombosed vessels are superficial veins, but an associated DVT may exist in as many as 40% of patients with superficial phlebitis.

Varices of recent onset are easily distinguished from chronic varices by palpation. Newly dilated vessels sit on the surface of the muscle or bone; chronic varices erode into underlying muscle or bone, creating deep "boggy" or "spongy" pockets in the calf muscle and deep palpable bony notches, especially over the anterior tibia.

Palpation often reveals fascial defects in the calf along the course of an abnormal vein at sites where superficial tributaries emerge through openings in the superficial fascia. Incompetent perforating veins may connect the superficial and deep venous systems through these fascial defects, but the finding is neither sensitive nor specific for perforator incompetence.

**Percussion;** This technique is useful in determining whether 2 venous segments are directly interconnected. With the patient in a standing position, a vein segment is percussed at one position while an examining hand feels for a "pulse wave" at another position. Percussion can be used to trace out the course of veins already detected by palpation, to discover varicose veins that could not be palpated, and to assess the relationships between the various varicose vein networks. Valsalva or cough with the examiners hand over the medial aspect of the knee can often elicit a palpable pulse wave with florid saphenofemoral junction incompetence.

**Indications;** Surgical removal or obliteration of varicose veins is often for cosmetic reasons alone. Noncosmetic indications include symptomatic varicosities (eg, pain, fatigability, heaviness, recurrent superficial thrombophlebitis, bleeding), or for the treatment of venous hypertension after skin or subcutaneous tissue changes, such as lipodermatosclerosis, atrophie blanche, ulceration, or hyperpigmentation, have developed.

**Conservative treatment** with stockings and external compression is an acceptable alternative to surgery, but worsening cutaneous findings or symptoms despite these measure usually warrant intervention. Nonetheless, a patient’s desire for surgical management over conservative treatment or for cosmetic purposes alone are both reasonable relative indications for surgery.
**Relevant Anatomy:** The greater saphenous vein (GSV) originates on the medial foot as part of the venous arch and receives tributaries from deep veins of the foot as it courses upward along the anterior aspect of the medial malleolus. From the ankle, the GSV continues along the anteromedial aspect of the calf to the knee and into the thigh, where it is found more medially. From the upper calf to the groin, the GSV is usually contained within an envelope of thin fascia. Visualization of this fascial envelope is an important way of identifying the GSV with duplex ultrasound. This fascial envelope often prevents the GSV from becoming significantly dilated, even when large volumes of reflux pass along its entire length. A normal GSV is typically 3-4 mm in diameter in the mid thigh.

Along its course, a variable number of named perforating veins may connect the GSV to the deep system at the femoral, posterior tibial, gastrocnemius, and soleal veins. The Cockett perforators, between the ankle and the knee, are a special group of perforating veins. Rather than directly connecting the superficial to deep venous systems, they connect the subfascial deep system with the posterior arch vein, which then empties into the GSV, as depicted in the image below.

![Named perforators along the greater saphenous distribution.](image-url)
Besides perforating veins, the GSV has numerous superficial tributaries as it passes through the thigh. The most important of these are the posteromedial and anterolateral thigh veins, found at the level of the mid thigh, and the anterior and posterior accessory saphenous veins at the level of the canal of Hunter in the upper thigh, where a perforating vein often connects the GSV to the femoral vein. Just below the SFJ, the GSV receives several additional important tributary veins. These include the lateral and medial femoral cutaneous branches, the external circumflex iliac vein, the superficial epigastric vein, and the internal pudendal vein, as depicted in the image below. These tributaries are frequently involved in the reflux that leads to the appearance of surface varicose veins on the lower thigh or upper calf.

Major tributaries of the greater saphenous system.

The termination point of the GSV into the common femoral vein is called the saphenofemoral junction in the English literature but is known as the crosse (ie, shepherd's crook) in the French medical literature, as depicted in the image below. The terminal valve of the GSV is located within the junction itself. In most cases, at least one additional subterminal valve is present within the first few centimeters of the GSV. Most patients have a single subterminal valve that can be readily identified approximately 1 cm distal to the junctional valve.
Reflux at or near the SFJ does not always come through the terminal valve of the GSV, nor does it always involve the entire trunk of the GSV. Reflux can enter the GSV below the subterminal valve or even immediately below the junction, passing through a failed subterminal valve to mimic true SFJ incompetence. Reflux can also pass directly into any of the other veins that join the GSV at that level, or it may pass a few centimeters along the GSV and then abandon the GSV for another branch vessel, as depicted in the image below.

Schematic diagram of the deep and superficial venous systems of the lower extremity: (1) Normal venous drainage; arrows depict the flow of venous blood. (2) Venous hypertension bold arrows are pathways of venous reflux.

When a perforating vein is the primary site of reflux, dilatation of the vessel proceeds both proximally and distally. When dilatation reaches the most proximal portion of the vein, the saphenofemoral or saphenopopliteal junction is often recruited as a secondary point of reflux. Although most large varices are tributaries off of an incompetent GSV or SSV, failed perforating veins or connecting veins
can also give rise to independent varices in the greater saphenous distribution without involving the saphenous system itself. Identifying the originating point and the primary pathway of reflux in the thigh is often difficult, which is why duplex ultrasound has become so helpful in varicose vein workup.9,10

**Contraindications**

Patients with venous outflow obstruction should not have their varicosities ablated because they are important bypass pathways that allow blood to flow around the obstruction. Those patients who cannot remain active enough to reduce the risk of postoperative deep vein thrombosis (DVT) should not undergo surgery. Surgery during pregnancy is contraindicated because many varicose veins of pregnancy spontaneously regress after delivery.

**Laboratory Studies**

No currently available lab test is useful in the diagnosis or therapy of varicose veins. Patients with varicose veins may have a spuriously positive D-dimer test result because of chronic low-level thrombosis within varices. See the eMedicine topic Deep Venous Thrombosis and Thrombophlebitis for more information.

Diagnostic Procedures; **The duplex ultrasound** (US) has become the most useful tool for workup and has replaced many of the physical examination maneuvers and physiological tests once used for diagnosis.

Tests used to rule out deep vein thrombosis obstruction as a cause of varicose veins

**Duplex US:** This is noninvasive imaging with good sensitivity and selectivity. See the DVT section for more discussion.

**Perthes maneuver/Linton test:** This is a physical examination technique in which a tourniquet is placed over the proximal part of the leg to compress any superficial varicose veins while leaving deep veins unaffected. The patient walks or performs toe-stands to activate the calf-muscle pump which normally causes varicose veins to be emptied. However, if obstruction of the deep system exists, then activation of the calf-muscle pump causes a paradoxical congestion of the superficial venous system and engorgement of varicose veins resulting in a positive test. To verify,
the patient is then placed supine, and the leg is then elevated (Linton test). If varices distal to the tourniquet fail to drain after a few seconds, again deep venous obstruction must be considered. This test is rarely performed in practice today with the advent of duplex imaging and assessment of the superficial and deep venous systems.

**Maximum venous outflow (MVO):** The MVO is a functional test to help detect obstruction to venous outflow. It can help detect more proximal occlusion of the iliac veins and IVC, as well as extrinsic causes of obstruction in addition to DVTs. MVO uses **plethysmography** (technique to measure volume changes of the leg) to measure the speed at which blood can flow out of a maximally congested lower leg when an occluding thigh tourniquet is suddenly removed.11

**Magnetic resonance venography (MRV):** The most sensitive and most specific test to find causes of anatomic obstruction. MRV is particularly useful because unsuspected nonvascular causes for leg pain and edema may often be seen on the scan image when the clinical presentation erroneously suggests venous insufficiency or venous obstruction. However, this is an expensive test used only as an adjuvant when doubt still exists.

**Tests used to demonstrate reflux**

**Duplex US with color-flow imaging** (sometimes called triplex ultrasound): This is a special type of 2-dimensional ultrasound that uses Doppler-flow information to add color for blood flow in the image. Vessels in the blood are colored red for flow in one direction and blue for flow in the other, with a graduated color scale to reflect the speed of the flow. Venous valvular reflux is defined as regurgitant flow with Valsalva that lasts greater than 2 seconds.

**Trendelenburg test:** This physical examination technique distinguish patients with reflux at the SFJ from those with incompetent deep venous valves. The leg is elevated until the congested superficial veins have all collapsed. Direct pressure is used to occlude the GSV just below the SFJ. The patient stands with the occlusion still in place. If the distal superficial varicosities remains empty or fills very slowly, the principal entry point of high pressure into the superficial system is at
the SFJ. Rapid filling despite manual occlusion means that some other reflux pathway is involved.

**Doppler auscultation**: A Doppler transducer is positioned along the axis of a vein with the probe at an angle of 45° to the skin. When the distal vein is compressed, audible forward flow exists. If the valves are competent, no audible backward flow is heard with the release of compression. If the valves are incompetent, an audible backflow exists. These compression-decompression maneuvers are repeated while gradually ascending the limb to a level at which the reflux can no longer be appreciated.

**Venous refilling time (VRT)**: This is a physiologic test, again using plethysmography. The VRT is the time necessary for the lower leg to become infused with blood after the calf-muscle pump has emptied the lower leg as thoroughly as possible. In healthy subjects, venous refilling is greater than 120 seconds. In patients with mild and asymptomatic venous insufficiency, VRT is between 40 and 120 seconds. In patients with significant venous insufficiency, VRT is abnormally fast at 20-40 seconds. Such patients often complain of nocturnal leg cramps, restless legs, leg soreness, burning leg pain, and premature leg fatigue. A VRT of less than 20 seconds is markedly abnormal, and is nearly always symptomatic. If the VRT is less than 10 seconds, venous ulcerations are likely.

Muscle pump ejection fraction (MPEF): The MPEF test is used to detect failure of the calf muscle pump to expel blood from the lower leg. MPEF results are highly repeatable but require a skilled operator. The patient performs ankle dorsiflexion 10-20 times, and plethysmography is used to record the change in calf blood volume. In healthy patients, the venous systems will drain, but in patients with muscle pump failure, severe proximal obstruction, or severe deep vein insufficiency, the amount of blood remaining within the calf has little or no change.11
Tests used to define anatomy

**Duplex US**

Two-dimensional ultrasound forms an anatomic picture based on the time delay of ultrasonic pulses reflected from deep structures. Structures that absorb, transmit, or scatter ultrasonic waves appear as dark areas; structures that reflect the waves back to the transducer appear as white areas in the image. Vessel walls reflect ultrasound waves; blood flowing in a vessel absorbs and scatters ultrasound waves in all directions. The normal vessel appears as a dark-filled, white-walled structure.

Duplex ultrasound is a combination of anatomic imaging by 2-dimensional ultrasound and flow detection by Doppler shift. With duplex ultrasound, after the 2-dimensional anatomic image is displayed, a particular spot in the image can be selected for Doppler-shift measurement of flow direction and velocity.

Structural details that can be observed include the most delicate venous valves, small perforating veins, reticular veins as small as 1 mm in diameter, and (using special 13-MHz probes) even tiny lymphatic channels.

Direct contrast venogram: An intravenous catheter is placed in a dorsal vein of the foot, and radiographic contrast material is infused into the vein. X-rays are then used to obtain an image of the superficial venous anatomy. If deep vein imaging is desired, a superficial tourniquet is placed around the leg to occlude the superficial veins and contrast is forced into the deep veins. Assessment of reflux can be difficult because it requires passing a catheter from ankle to groin, with selective introduction of contrast material into each vein segment. This is a labor-intensive and invasive venous imaging technique with a 15% chance of developing new venous thrombosis from the procedure itself. It is rarely used, and has been replaced by duplex ultrasound. Its use is reserved for difficult or confusing cases.

**Staging**

This classification is based on the clinical, etiological, anatomic, pathophysiological (CEAP) classification from the American Venous Forum, last revised 2004.12 It is used to standardize recording of venous disease, as follows:
Clinical

C0 - No visible or palpable signs of venous disease
C1 - Telangiectases or reticular veins
C2 - Varicose veins
C3 - Edema
C4a - Pigmentation or eczema
C4b - Lipodermatosclerosis or atrophie blanche
C5 - Healed venous ulcer
C6 - Active venous ulcer

S – Symptomatic, includes: ache, pain, tightness, skin irritation, heaviness, and muscle cramps, and other complaints attributable to venous dysfunction

A - Asymptomatic

Etiologic classification

Ec - Congenital
Ep - Primary
Es - Secondary (post-thrombotic)
En - No venous cause identified

Anatomic classification

As - Superficial veins
Ap - Perforator veins
Ad - Deep veins
An - No venous location identified
Pathophysiologic classification Basic CEAP

Pr - Reflux

Po - Obstruction

Pr,o – Reflux and obstruction

Pn - No venous pathophysiology identifiable

Advanced CEAP: Same as basic CEAP, with addition that any of 18 named venous segments can be used as locators for venous pathology

Superficial veins: (1) telangiectasias or reticular veins, GSV (2) above knee or (3) below knee, (4) small saphenous vein, or (5) nonsaphenous veins

Deep veins: (6) Inferior vena cava, (7) common iliac vein, (8) internal iliac vein, (9) external iliac vein, (10) pelvic veins - gonadal, broad ligament veins, other, (11) common femoral vein, (12) deep femoral vein, (13) femoral vein, (14) popliteal vein, (15) crural veins (anterior tibial, posterior tibial, peroneal veins (all paired)), or (16) muscular veins - gastrocnemial, soleal veins, other.

Perforating veins: Thigh or calf

Example: A patient has painful swelling of the leg, and varicose veins, lipodermatosclerosis, and active ulceration. Duplex scanning on May 17, 2004, showed axial reflux of the great saphenous vein above and below the knee, incompetent calf perforator veins, and axial reflux in the femoral and popliteal veins. No signs of post-thrombotic obstruction are present.

Classification according to basic CEAP: C6,S, Ep,As,p,d, Pr.

Classification according to advanced CEAP: C2,3,4b,6,S, Ep,As,p,d, Pr2,3,18,13,14
Treatment  Surgical Therapy

The surgical treatment of varicose veins have been under development for more than 2000 years, but until the present era, relatively little weight was given to the cosmetic outcome of treatment. Current therapies are becoming less invasive with improved recovery, but long-term outcomes are uncertain. Therapies aim to remove the superficial venous system either through surgery, endovenous ablation, or sclerotherapy ablation. In 90% of cases where venous hypertension is from superficial and perforator vein reflux, removal or obliteration of the GSV alone can resolve the venous hypertension. In the remaining 10%, additional treatment to the incompetent perforator veins may be needed. Additionally, if severe deep venous incompetence exists, treatment of the GSV alone usually does not resolve the venous hypertension. In both these cases, additional interventions with subfascial endoscopic perforating vein surgery (SEPS), perforator vein ablation, and/or venous reconstruction can be attempted, but these details are not further discussed in this article. For now, the authors will discuss the procedures to remove or obliterate the superficial venous system, starting from most to least invasive. Historical perspectives, advantages, and disadvantages to each technique will also be addressed. However, prior to any intervention, duplex US should always be used to map all major reflux pathways, and a skin marker should be used to mark all surface vessels to be removed.

Open techniques

The Rindfleisch-Friedel procedure of the early 1900s involved one incision to the level of the deep fascia that wrapped around the leg 6 times, creating a spiral gutter that brought into view a large number of superficial veins, each one of which was ligated. This wound was left open to heal by granulation. The Linton procedure, introduced in the late 1930s, used a large linear medial leg incision that brought into view all the superficial and perforator veins of the leg. Incompetent superficial veins were removed, and perforating veins were interrupted.
Friedrich Trendelenburg, in the late 1800s, introduced a mid-thigh ligation of the GSV. The outcomes were variable, and this procedure was later modified by Trendelenburg's student Perthes, who advocated a groin incision and a ligation of the GSV at the saphenofemoral junction. Later, even better outcomes were found if saphenectomy (removal of the GSV) with ligation at the SFJ was performed over ligation alone. In a randomized trial, two thirds of patients with ligation without saphenectomy could be expected to need reintervention within 5 years for recurrent reflux, either through recanalization or collateral formation around the ligated GSV.14,15

**GSV saphenectomy:** Surgical removal of the GSV has evolved from large open incisions to less invasive stripping. Original methods of stripping used different devices and variations of techniques. The Mayo stripper was an extraluminal ring that cut the tributaries as it was passes along the vein. The Babcock device was an intraluminal stripper with an acorn-shaped head that pleated up the vein as it pulled the vessel loose from its attachments. The Keller device was an internal wire used to pull the vein through itself, as is done today with perforation-invagination (PIN) strippers. Currently, the technique of PIN stripping begins with a 2- to 3-cm incision made at the groin crease. The femoral vein and SFJ are exposed with dissection and all tributaries of the SFJ must be identified and flush-ligated to minimize the incidence of reflux recurrence.

After ligation and division of the junction, the stripping instrument (usually a stiff but flexible length of wire or plastic) is passed into the GSV at the groin and threaded through the incompetent vein distally to the level of the upper calf. The stripper is brought out through a small incision (5 mm or smaller) approximately 1 cm from the tibial tuberosity at the knee. An inverting head is attached to the stripper at the groin and is secured to the proximal end of the vein. The vessel is then inverted into itself, tearing away from each tributary and perforator as the stripper is pulled downward through the leg and out through the incision in the upper calf, as depicted in the image below. If desired, a long epinephrine-soaked gauze or ligature may be secured to the stripper before invagination, allowing hemostatic packing to be pulled into place after stripping is complete.
An older technique of stripping to the ankle (rather than to just the knee) has fallen into disfavor because of a high incidence of complications, including damage to the saphenous nerve, which is closely associated with the vein below the knee.9

**SSV saphenectomy;** Removal of the short saphenous vein is complicated by variable local anatomy and risk of injury to the popliteal vein and peroneal nerve. The saphenopopliteal junction must be located by duplex examination before beginning the dissection, and adequate direct visualization of the junction is essential. After ligation and division of the junction, the stripping instrument (often a more rigid stripper that facilitates navigation) is passed downward into the distal calf, where it is brought out through a small incision (2-4 mm). The stripper is secured to the proximal end of the vein, which is invaginated into itself as it is pulled downward from knee to ankle and withdrawn from below.
Stab phlebectomy (or ambulatory phlebectomy) Muller Technique

Performed by Galen as early as the second century, this procedure came back into modern favor during the 1960s and has increased in popularity ever since. This procedure is extremely useful for the treatment of residual vein clusters after saphenectomy and for removal of nontruncal tributaries when the saphenous vein is competent. A microincision is made over the vessel using a tiny blade or a large needle, a phlebectomy hook is introduced into the microincision, and the vein is delivered through the incision. With traction, as long a segment as possible is pulled out of the body until the vein breaks or cannot be pulled any further. Another microincision is made and the process is begun again and repeated along the entire length of the vein to be extracted. Short segments of veins can be removed through tiny incisions without ligatures, and skin closure is not necessary.9

Endovascular techniques Endovenous (EV) laser. A laser fiber produces endoluminal heat that destroys the vascular endothelium. A Seldinger technique is used to advance a long catheter along the entire length of the truncal varicosity (usually the GSV) to be ablated. A bare laser fiber is passed through the catheter until the end protrudes from the tip of the catheter by approximately 2 cm, and the laser fiber tip is positioned at the SFJ just distal to the subterminal valve. The position is confirmed by ultrasound and by use of the laser guide light.

Under ultrasound guidance, tumescent solution with a local anesthetic is injected around the entire length of the vessel, separating it from its fascial sheath. This serves to insulate the heat from damaging adjacent structures, including nerves and skin, as well as pain control. Firm pressure is applied to collapse the vein around the laser fiber, and the laser is fired generating heat, leading to intraluminal steam bubbles and irreversible endothelial damage and thrombosis. The fiber and catheter are withdrawn approximately 2 mm, and the laser is fired again. This process is repeated along the entire course of the vessel.16
Radiofrequency (RF) ablation. RF thermal energy is delivered directly to the vessel wall, causing protein denaturation, collagenous contraction, and immediate closure of the vessel. In contrast to laser therapy, the RF catheter actually comes into contact with the lumen walls. An introducer sheath is inserted into the proposed vein of treatment (again usually the GSV). A special RF ablation catheter is passed through the sheath and along the vein until the active tip is at the SFJ just distal to the subterminal valve. Just like the endovenous laser, tumescent local anesthetic is injected. Metal fingers at the tip of the RF catheter are deployed until they make contact with the vessel endothelium. RF energy is delivered, both in and around the vessel to be treated. Thermal sensors record the temperature within the vessel and deliver just enough energy to ensure endothelial ablation. The RF catheter is withdrawn a short distance, and the process is repeated all along the length of the vein to be treated.

Minimally invasive techniques

Sclerotherapy; Chemical sclerosis of varicose veins has waxed and waned in popularity since the late 1800s. Modern sclerosants with an acceptable risk profile became widely available in the 1930s, and, since that time, there use has expanded. Initially, sclerotherapy was used as a surgical adjunct after saphenectomy to treat residual varicosities, reticular veins, or telangiectasias. Now it is being used to treat the GSV and main tributaries. Under US guidance, a sclerosing substance is injected into abnormal vessels to produce endothelial destruction that is followed by formation of a fibrotic cord and eventual reabsorption of all vascular tissue layers, as depicted in the image below.

Ultrasound image of the GSV after foam sclerotherapy treatment. Note the hyperechogenicity within the vein is from the foam. Local treatment of the superficial manifestations of venous insufficiency will always fail if the underlying high points of reflux have not been found and treated. Even when the patient appears to have only primary telangiectasias and the initial treatment seems to be successful, recurrences will be seen very quickly if unrecognized reflux exists in larger subsurface vessels.
Caution must be used when using sclerosing agents, inadvertent injection into an arteriovenous malformation or directly into an unrecognized artery can cause extensive tissue loss or loss of the entire limb. Inadvertent injection of concentrated sclerosants into the deep system can cause DVT, pulmonary embolism, and death. The most commonly used sclerosants today are polidocanol and sodium tetradecyl sulfate. Both are known as detergent sclerosants because they are amphiphilic substances, inactive in dilute solution, but biologically active when they form micelles. These agents are preferred because they have a low incidence of allergic reactions, produce a low incidence of staining and other adverse cutaneous effects, and are relatively forgiving if extravasated. Polidocanol, the most forgiving sclerosing agent, was originally developed as a local anesthetic agent. Other agents that have fallen out of favor include sodium morrhuate, associated with a relatively high incidence of anaphylaxis. Ethanolamine oleate, a weak detergent, is excessively soluble, decreasing its ability to denature cell surface proteins. Hypertonic saline in a 20% or 23.4% solution can be used as a sclerosing agent, but, because of dilutional effects with injection, it is difficult to achieve adequate sclerosis of large vessels without exceeding a tolerable salt load. If extravasated, it almost invariably causes significant necrosis. The addition of foam with the sclerosing agents has allowed for decreased amounts of sclerosing agent injection and improved efficacy. Foam pushes blood out of the vein, allowing for less dilution and more contact of the sclerosant with the endothelium. Homemade foam is usually air agitated in saline. This has theoretical risks of air emboli, so commercially available foam consists of mostly carbon dioxide. Varisolve (BTG, West Conshohocken, PA) is one such product using carbon dioxide foam and polidocanol sclerosant, as depicted in the image below.

In the US, sodium tetradecyl sulfate, sodium morrhuate, and ethanolamine oleate were all developed prior to the establishment of the FDA. These agents have never been submitted to the FDA for approval, but they are available in the United States as grandfathered agents. No FDA–approved foam/sclerosing agents are available; however, the Varisolve product is currently under clinical trials in the United States after being used extensively in Europe.
**Postoperative Details**

After treatment of large varicose veins by any method, a 30- to 40-mm Hg gradient compression stocking is applied and patients are instructed to maintain or increase their normal activity levels. Most practitioners also recommend the use of gradient compression stockings even after treatment of spider veins and smaller tributary veins. Ace wraps and other long-stretch bandages should not be used. These elastic bandages fail to maintain adequate compression for more than a few hours. They often slip or are misapplied by patients, with a resulting tourniquet effect that causes distal swelling and increases the risk of DVT. Activity is particularly important after treatment by any technique because all modalities of treatment for varicose disease have the potential to increase the risk of DVT. Activity is a strong protective factor against venous stasis. Activity is so important that most venous specialists will not treat a patient who is unable to remain active following treatment.

**Follow-up** For patient education resources, visit eMedicine's Circulatory Problems Center. Also, see eMedicine's patient education articles Varicose Veins, Blood Clot in the Legs, and Phlebitis.

**Complications** A correct diagnosis of superficial venous insufficiency is essential. Veins should be treated only if they are incompetent and if a normal collateral pathway exits. Removal of a saphenous vein with a competent termination will not aid in the management of nontruncal tributary varices. In the setting of deep system obstruction, varicosities are hemodynamically helpful because they provide a bypass pathway for venous return. Hemodynamically helpful varices must not be removed or sclerosed. Ablation of these varicosities will cause rapid onset of pain and swelling of the extremity, eventually followed by the development of new varicose bypass pathways. The most annoying minor complications of any venous surgery are dysesthesias from injury to the sural nerve or the saphenous nerve. Subcutaneous hematoma is a common complication, regardless of treatment technique used. It is easily managed with warm compress, NSAIDS, or aspiration if necessary.
At the saphenofemoral junction, accidental treatment of the femoral vein by inappropriate RF or laser catheter placement, or spread of sclerosant (not visualizing progression with US), or inappropriate surgical ligation can all lead to endothelium damage at the deep vein, causing deep vein thrombosis (DVT) formation with the potential of pulmonary embolism (PE) and even death. Other complications, such as postoperative infection and arterial injury, are less common and may be kept to a minimum through strict attention to good technique. Endovenous treatment techniques (with RF and laser therapy) have the potential of excessive tissue heating, which can lead to skin burns. This problem can be avoided if sufficient volumes of tumescent anesthetic are injected to elevate the skin away from the vein.

**Outcome and Prognosis**

With appropriate treatment, the vast majority of patients have a good outcome and the progression of their disease is arrested. Saphenectomy has been the criterion standard to which most therapies are compared. In a randomized trial comparing radiofrequency (RF) ablation to saphenectomy, 2-year reoccurrence rates were nonsignificantly different at 14% versus 21%, respectively. Quality of life scores were better for RF obliteration versus saphenectomy. The long-term results of endovenous (EV) laser compared with saphenectomy have not been as well validated. In a randomized trial, follow-up was only 6 months, and the authors found 0% versus 5.8% recanalization in EV laser versus saphenectomy. Saphenectomy did have higher postoperative pain, but overall quality of life scores remained equal. Other results were similar between the saphenectomy versus EV laser groups; mean time to resume normal physical activity was 7.7 versus 6.9 calendar days, mean time to resume work was 7.6 versus 7.0 calendar days, and total cost of the procedures, including lost wages, were $3948 versus $4347, respectively. One randomized trial has compared RF to EV laser ablation of the GSV. This was a single institution, and they found significantly increased closure rate after RF (80%) versus EV laser (66%) treatment at one year follow-up. The DVT, paresthesias, leg edema, and superficial thrombophlebitis complication rates were equal in the 2 groups.
Foam sclerotherapy has been used in Europe, and phase III randomized clinical trials, with multiple arms, have compared it with saphenectomy and sclerotherapy without foam. At 12 months, GSV closure rates were 87.2% in the saphenectomy versus 68.2% in the Varisolve arm. But in the other arm, sclerotherapy without foam versus Varisolve, closure rates for the Varisolve group were much improved at 93.8%. Although surgery was more efficacious, Varisolve did cause less pain and patients returned to normal more quickly. In the 710 patients enrolled, no pulmonary embolus were found, and DVTs were found in 2.5% of Varisolve, none in surgery, and 0.8% in those who received sclerotherapy without foam. In the US clinical trials are underway to assure there is no increased risk of embolic stroke from the use of Varisolve.

**Future and Controversies** Management of varicose veins has evolved over the centuries and will continue to do so. Less invasive techniques continue to be refined but long-term efficacy must always be questioned and compared with the criterion standard of surgical saphenectomy.
Chronic Venous Insufficiency

Introduction

Chronic venous insufficiency (CVI) is a common condition affecting 2-5% of Americans. Historically, CVI was known as postphlebitic syndrome and postthrombotic syndrome, both of which refer to the etiology of most cases. However, these names have been abandoned because they fail to recognize another common cause of the disease, the congenital absence of venous valves.

Picture of venous valve: Thrombosis can begin as blood flow becomes turbulent, permitting platelets to remain in the valve sinus. This forms the nidus of a thrombus.
History of the Procedure In 1914, Homans postulated that the relative hypoxia of static venous blood decreases the amount of oxygen reaching the skin, causing skin changes and ulcers characteristic of CVI. In 1930, Landis et al demonstrated the direct relationship between venous hypertension in the legs and increased capillary intraluminal pressures. In 1953, Piulacks et al theorized that arteriovenous fistulas in the skin of the lower extremities cause hypoxia, resulting in changes to the skin and tissues. In 1982, Burnand et al presented the fibrin cuff hypothesis, which describes the primary problem as venous hypertension in the lower extremities causing leakage of plasma proteins, particularly fibrinogen. A fibrin cuff encircles affected capillaries, decreasing oxygen diffusion to surrounding tissues. In 1988, Coleridge-Smith et al described the white-cell trapping theory, which hypothesizes that venous hypertension and resultant increased capillary pressures trap white blood cells in the capillaries, where they become activated and damage capillary beds. Increased capillary permeability allows seepage of plasma proteins and fibrinogen into the interstitium, where a fibrin cuff forms, thus decreasing oxygen diffusion to surrounding tissues.

Problem In addition to poor cosmesis, CVI can lead to chronic life-threatening infections of the lower extremities. Pain, especially after ambulating, is a hallmark of the disease. CVI causes characteristic changes, called lipodermatosclerosis, to the skin of the lower extremities, which lead to eventual skin ulceration.

Frequency CVI is a significant public health problem in the United States. Of all Americans, estimates indicate that 2-5% have some changes associated with CVI. Approximately 24 million Americans have varicose veins. Approximately 6 million Americans have skin changes associated with CVI. Venous stasis ulcers affect approximately 500,000 people. The mean incidence for hospital admission for CVI is 92 per 100,000 admissions.

Epidemiology: Peak incidence occurs in women aged 40-49 years and in men aged 70-79 years.
**Etiology** Congenital absence of or damage to venous valves in the superficial and communicating systems can cause CVI. Venous incompetence due to thrombi and formation of thrombi favored by the Virchow triad (venous stasis, hypercoagulability, endothelial trauma) also can cause CVI, as depicted in the image below. Varicose veins rarely are associated with the development of CVI. Picture of venous valve: Thrombosis can begin as blood flow becomes turbulent, permitting platelets to remain in the valve sinus. This forms the nidus of a thrombus.

**Risk factors** associated with chronic venous insufficiency

Age: Incidence of CVI rises substantially with age.

Family history: History of deep vein thrombosis (DVT), which renders venous valves incompetent, causing backflow and increased venous pressure, is a risk factor.

Lifestyle: A sedentary lifestyle minimizes the pump action of calf muscles on venous return, causing higher venous pressure. CVI occurs more frequently in women who are obese. Vocations that involve standing for long periods predispose individuals to increased venous pressure in dependent lower extremities. A higher incidence of CVI is observed in men who smoke.

**Pathophysiology**

Two major mechanisms in the body prevent venous hypertension. First, bicuspid valves in the veins prevent backflow and venous pooling. DVTs commonly occur at these valves, causing irreversible damage to the valve. Second, during normal ambulation, calf muscles decrease venous pressures by approximately 70% in the lower extremities. With rest, pressures return to normal in approximately 30 seconds. In diseased veins, ambulation decreases venous pressures by only 20%. When ambulation is stopped, pressure in the vein lumen increases slowly, returning to normal over a period of minutes, as depicted in the image below.
Hemodynamic charting of (a) healthy patients, (b) patients with only varicose veins, (c) patients with incompetent perforator veins, and (d) patients with deep and perforator incompetence.

Venous hypertension in diseased veins is thought to cause CVI by the following sequence of events. Increased venous pressure transcends the venules to the capillaries, impeding flow. Low-flow states within the capillaries cause leukocyte trapping. Trapped leukocytes release proteolytic enzymes and oxygen free radicals, which damage capillary basement membranes. Plasma proteins, such as fibrinogen, leak into the surrounding tissues, forming a fibrin cuff. Interstitial fibrin and resultant edema decrease oxygen delivery to the tissues, resulting in local hypoxia. Inflammation and tissue loss result.

Presentation

Clinical manifestations include the following:

**Varicose veins:** In addition to poor cosmesis, varicose veins serve as indicators of venous hypertension, the most common reason for patient complaints regarding CVI, as depicted in the image below.8

**Thrombosis** can begin as blood flow becomes turbulent, permitting platelets to remain in the valve sinus. This forms the nidus of a thrombus.
**Leg discomfort:** Venous hypertension in muscles and fascial compartments of the lower leg from exercise and prolonged standing results in the characteristic ache of CVI. The discomfort is described as pain, pressure, burning, itching, dull ache, or heaviness in affected calves or legs.

**Nonhealing ulcers:** Typically, these lesions occur around the medial malleolus, where venous pressure is maximal due to the presence of large perforating veins, as depicted in the image below.

**Leg edema:** Damage done to capillary basement membranes by white blood cells results in leg edema.

**Lipodermatosclerosis:** These characteristic skin changes in the lower extremities include capillary proliferation, fat necrosis, and fibrosis of skin and subcutaneous tissues. Skin becomes reddish or brown because of the deposition of hemosiderin from red blood cells, as depicted in the image below.9

![Perforator vein bulging into subcutaneous tissue.](image)
**Indications**

Surgical treatment is reserved for those with discomfort or ulcers refractory to medical management. Indications for vein ligation: This technique is reserved for cases of CVI that include reflux in the saphenous system causing severe symptoms. For this reason, a diagnosis of reflux must be established preoperatively, usually with photoplethysmography or duplex imaging.

**Relevant Anatomy**

The venous network in the lower extremities commonly affected by CVI is divided into 3 systems. The first is superficial veins, which include the lesser and greater saphenous veins and their tributaries, as depicted in the 1st image below. The second is deep veins, which include the anterior tibial, posterior tibial, peroneal, popliteal, deep femoral, superficial femoral, and iliac veins. The third is perforating or communicating veins, as depicted in the 2nd and 3rd image below.

*Lower leg venous anatomy.*
Perforating veins of the lower leg.

Venogram demonstrating incompetent perforating veins.
Contraindications

In patients with symptomatic greater saphenous varicosities, the presence of an occluded deep system must be ruled out. Deep occlusion is an absolute contraindication to vein ligation. Obtaining venographic studies of the deep venous system prior to superficial vein ligation is imperative.

Imaging Studies

Doppler bidirectional-flow studies and Doppler color-flow studies are used to assess venous flow, its direction, and the presence of thrombus.

Other Tests

Photoplethysmography uses infrared light to assess capillary filling during exercise. Increased capillary filling is indicative of venous reflux and, consequently, incompetent veins.

Outflow plethysmography involves placing and subsequently releasing a tourniquet on the lower extremity; the veins should quickly return to baseline pressures. Failure to do so indicates reflux.

Treatment

Medical Therapy

Nonsurgical treatments for CVI include the following:

Leg elevation; By keeping the legs elevated, venous flow is augmented by gravity, lowering venous pressures and ameliorating edema. While sitting, the legs should be above the thighs. Supine, the legs should be above the level of the heart.
Compression stockings; First described by Jobst in 1940, compression stockings produce graded pressures from the foot to the knee or thigh to decrease edema and minimize venous hypertension. For two clinical studies of compression therapy, see Vanscheidt et al.11

Unna boots; First described by Unna in 1854, the Unna boot now is the mainstay of treatment for people with venous ulcers. Unna boots are rolled bandages that contain a combination of calamine lotion, glycerin, zinc oxide, and gelatin.

Injection sclerotherapy; Injection of sclerosing agent directly into veins usually is reserved for telangiectatic lesions rather than CVI.

Phlebotonics have not been proven to be beneficial for CVI.12

Surgical Therapy

Approximately 8% of patients require surgical intervention for CVI. Surgical treatment is reserved for those with discomfort or ulcers refractory to medical management. Below are several conditions and the surgical options considered appropriate for each.

Chronic venous insufficiency resulting from superficial vein disorders

Vein ligation is the treatment of choice for superficial vein disorders. Historically, the entire greater saphenous vein system was removed; this has been replaced by the stab evulsion technique. Several 2- to 3-mm incisions are made overlying the greater saphenous at various levels. The vein is dissected from the underlying tissues and any perforators are ligated. A small hook or blunt needle is used to extract as much of the vein as possible.

Typically, stab evulsion is limited to areas above the knee in the greater saphenous system to avoid damage to the saphenous nerve or sural nerve. This technique is reserved for CVI in which reflux in the saphenous system occurs and causes severe symptoms. For this reason, a diagnosis (usually accomplished with photoplethysmography or duplex imaging of reflux) must be established.
preoperatively. Hematoma, sural or saphenous nerve damage, and infection are possible complications of vein ligation.

Chronic venous insufficiency resulting from deep vein disorders

The decision to operate on a patient with venous obstruction in the deep veins should be made only after a careful assessment of symptom severity and direct measurement of both arm and foot venous pressures. Venography alone is not sufficient because many patients with occlusive disease have extensive collateral circulation, rendering them less symptomatic. Clot lysis (eg, tissue plasminogen activator [TPA], urokinase) and thrombectomy have been tried but have largely been abandoned owing to extremely high recurrence rates.

For iliofemoral disease, the operation of choice is a saphenous vein crossover graft. In the procedure, the contralateral saphenous vein is mobilized and divided at its distal end. It then is tunneled suprapublically and anastomosed to the femoral vein on the diseased side. The result is the diversion of venous blood through the graft and into the intact contralateral venous system, as depicted in the image below.

Chronic venous stasis ulcer.

Because of a relatively high failure rate of 20%, ringed polytetrafluoroethylene (PTFE) grafts are used. The long-term patency is unknown.

Superficial femoral vein occlusion

Described by Warren in 1954 and Husni in 1983,13 the Husni bypass (as it has come to be called) is used to treat occlusion of the superficial femoral vein. The ipsilateral greater saphenous vein is harvested and used as an in situ popliteal-femoral vein bypass. This surgery is performed infrequently due to the high failure rate (approximately 40%). For a minimally invasive technique using stents, see Raju and Neglén.14
**Deep vein incompetence** Valvuloplasty is reserved for patients with a congenital absence of functional valves. A venotomy is performed, and the valve cusps are plicated. To ensure an adequate result, plicating 20-25% of each cusp is recommended. The addition of a PTFE sleeve around the operative site is used routinely to maintain valve integrity. When combined with the ligation of perforating veins, valvuloplasty has a superior outcome in 80% of cases after 5 years. With vein segment transposition, a vein with normal function in close proximity to the diseased vessel is identified. The incompetent vein then is dissected, mobilized, and transposed on to the normal vein distal to a functional valve. With vein valve transplantation, a valve-containing segment of a competent axillary or brachial vein is mobilized and inserted into either the popliteal or the femoral systems. The incompetent segment of the leg vein is excised and replaced with the transplant segment. Allograft or cadaveric vein transplants are being tested, with long-term results pending.

Commonly, both Doppler bidirectional-flow studies and Doppler color-flow studies are used to assess venous flow, its direction, and the presence of thrombus.

Photoplethysmography uses infrared light to assess capillary filling during exercise. Increased capillary filling is indicative of venous reflux and, consequently, incompetent veins.

Outflow plethysmography involves placing and subsequently releasing a tourniquet on the lower extremity; the veins should quickly return to baseline pressures, and failure to do so indicates reflux.

**Intraoperative Details**

Careful monitoring of a patient's cardiac status and vital signs is extremely important. In addition, periodic monitoring of hemoglobin and hematocrit levels yields essential intraoperative data.

**Postoperative Details:** Anticoagulation with heparin (or low molecular weight heparin) in the immediate postoperative period and long-term prophylaxis with Coumadin are recommended.
Follow-up; Patients should be observed frequently for wound infection after discharge, beginning 1 week postoperatively. Sutures or staples typically stay in 2-4 weeks, depending on the health of the skin at the operative site.

Outcome and Prognosis; Hematoma, sural or saphenous nerve damage, and infection are possible complications of lower-extremity vein ligation. Clot lysis (eg, TPA, urokinase) and thrombectomy have been tried but generally have been abandoned due to extremely high recurrence rates. For iliofemoral disease, the operation of choice is a saphenous vein crossover graft. Due to a relatively high failure rate of 20%, ringed PTFE grafts are being used. The long-term patency is unknown. The Husni bypass for superficial femoral vein occlusion is performed infrequently due to the high failure rate (approximately 40%).

Surgery for CVI resulting from deep vein incompetence includes valvuloplasty and allograft or cadaveric vein transplant. Valvuloplasty for patients with congenital absence of functional valves, when combined with the ligation of perforating veins, has a superior outcome in 80% of cases after 5 years. Allograft or cadaveric vein transplants are being tested, with long-term results pending.

Tsai et al examined the National Inpatient Sample from 1988-2000 and found that mean hospital charges were $13,900 and did not change over the time period examined. They also found that deep venous thrombosis affected 1.3% of patients and amputation was necessary in 1.2%, with an overall mortality of 1.6%.

Future and Controversies

Subfascial endoscopic perforator surgery (SEPS) is gaining in popularity as a means of treating CVI. Endoscopic techniques are used to find and ligate perforating veins. Preliminary reports are encouraging. The 1997 North American Subfascial Endoscopic Perforator Surgery Registry showed that after SEPS, the average healing time for ulcers was 42 days, with a recurrence rate of 3%. Ulcers treated with SEPS heal 4 times faster than ulcers treated conventionally. In addition, morbidity of SEPS is significantly lower than traditional operations. Long-term results are pending.